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DERMATOLOGY
The demographics of skin disease

DEFINITIONS

Dermatology is the study, investigation and management of diseases of the skin, hair and nails.

There are over 2000 diagnoses ranging from rare genodermatoses to the ubiquitous acne. Most presentations are for the 10 commonest skin diseases.

For the purpose of presenting data, the numbers below relate to a NHS primary care cluster or clinical commissioning group (CCG) covering a population of 100,000.

PREVALENCE AND INCIDENCE

In any year, about 50% of the population will develop a problem with their skin and 25% will develop a skin condition that necessitates a consultation with their GP. 95% of this group have always been managed in primary care with approximately 5% being referred to secondary care services (1-2% of GP list referred per year). The commonest dermatology conditions presenting in the general population are shown in table 1.

Table 1 Prevalence of skin conditions in the general population expressed as rates per 1000 (to nearest unit).

<table>
<thead>
<tr>
<th>Diagnostic category</th>
<th>Rate per 1000 Population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All Grades</td>
</tr>
<tr>
<td>Eczema</td>
<td>90</td>
</tr>
<tr>
<td>Prurigo and allied conditions</td>
<td>82</td>
</tr>
<tr>
<td>Acne</td>
<td>86</td>
</tr>
<tr>
<td>Scaly dermatoses</td>
<td>85</td>
</tr>
<tr>
<td>Erythematous and other dermatoses</td>
<td>75</td>
</tr>
<tr>
<td>Nail disorders</td>
<td>33</td>
</tr>
<tr>
<td>Tumours and vascular lesion</td>
<td>205</td>
</tr>
<tr>
<td>Scalp and hair disorders</td>
<td>82</td>
</tr>
<tr>
<td>Infective and parasitic conditions</td>
<td>46</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>16</td>
</tr>
<tr>
<td>Warts</td>
<td>34</td>
</tr>
<tr>
<td>Mouth and tongue disorders</td>
<td>9</td>
</tr>
<tr>
<td>Chronic ulcer</td>
<td>2</td>
</tr>
<tr>
<td>Any (one or more) skin condition</td>
<td>555</td>
</tr>
</tbody>
</table>

Demand

1. In any NHS CCG serving a population of 100,000 people it has been estimated that 25,000 people will have some form of skin disease.
2. Nearly a third of these sufferers (7,500) will treat themselves, and nearly two-thirds (14,550) will consult a GP.
3. Over the last 20 years there has been a sustained, up to 20% increase in the incidence of childhood atopic dermatitis
4. There is a consistent year on year increase in melanoma and non melanoma skin cancer
5. The increase in the number of elderly in the population poses its own challenges in managing age related skin problems, asteatosis, pruritus, leg ulcers, skin cancer immunobullous disease etc etc.
6. Of all GP consultations, 15% are for skin problems (table 2).
7. Table 2 Proportion of people with skin conditions among people consulting their GP

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage of people consulting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin and subcutaneous infections</td>
<td>4%</td>
</tr>
<tr>
<td>Atopic dermatitis (including eczema and nappy rash)</td>
<td>3%</td>
</tr>
<tr>
<td>Contact dermatitis</td>
<td>2%</td>
</tr>
<tr>
<td>Acne</td>
<td>2%</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>1%</td>
</tr>
<tr>
<td>Other skin conditions</td>
<td>3%</td>
</tr>
<tr>
<td>Total skin conditions</td>
<td>15%</td>
</tr>
</tbody>
</table>

GPs classify a third of these conditions as 'minor' - equating to 4,550 minor skin conditions receiving GP advice in our CCG population of 100,000.

Despite the immense workload generated by skin disease the total NHS expenditure is only £2bn.
SECTION 1

CONDITIONS
ACNE VULGARIS

DEFINITION

This common skin disease affects virtually all adolescents to some extent and is a chronic inflammatory disease of the pilo-sebaceous apparatus. It can also be a significant problem in some adults particularly women in their late 20s early 30s. Only a small minority of sufferers will seek medical advice.

CAUSE

The condition is probably hormone driven causing an increase in sebum production, an abnormal proliferation and differentiation of ductal keratinocytes leading to occlusion and colonisation with the commensal bacteria Propionobacterium acnes leading to an inflammatory reaction.

Exacerbating factors may include:-

- Hormonal factors – some females will experience a flare around their periods.
- Diet - there is no evidence that diet has any role in the aetiology of acne.
- Stress may be an aggravating factor.
- Drugs – topical and oral steroids, anabolic steroids and lithium may exacerbate.

UV exposure may help acne (especially comedonal), but do NOT advise the use of sunbeds

CLINICAL FEATURES

Formerly considered a disease of teenagers, more and more adults, particularly females, in their 20s and 30s are presenting with and seeking treatment for their acne. Though predominantly occurring on the face, acne will also affect the back and chest to a certain degree. The treatment of acne depends upon the lesions the patient presents with and may require combination of topical and systemic agents.

Lesions include:-

- comedones – blackheads (open) and whiteheads (closed) (Fig 1)
- papules/pustules (Fig 2)
- nodules (Fig 3)
- cysts (Fig 3)
- scars- keloidal or atrophic (ice pick)

DIFFERENTIAL DIAGNOSIS

Although usually a straightforward diagnosis in the adolescent, gram negative folliculitis may occur after long term antibiotic treatment and may present as treatment resistant acne. In the adult the differential diagnosis may include rosacea. If an adult woman suddenly gets devastating acne and hirsutism one should consider a hormonal tumour.

INVESTIGATIONS
• None for most patients. Hormone profile for older female with hirsutism, menstrual irregularity etc.
• Liver function tests and fasting lipids. For females of childbearing age being considered for Isotretinoin please arrange a serum pregnancy test less than 7 days before the appointment. Availability of these will minimize appointments.

**MANAGEMENT**

The majority of people with acne will self medicate with over the counter preparations from the pharmacy.

*Comedonal acne*
Managed with topical agents, usually a topical Retinoid, Benzoyl peroxide, Adapalene, Azelaic acid or Nicotinamide. Many of these products can be quite irritant to the skin so advise patient to build up length of and frequency of application. They must be used for at least 6 weeks before seeing any benefit.

Agents recommended in the Wirral formulary include:-

**First choice**
**Benzoyl Peroxide 5% gel** - Apply once or twice daily

**Second choice**
**Tretinoin (Retin-A®) 0.025% cream** - Apply thinly once or twice daily
Or
**Adapalene (Differin®) 0.1% cream/gel** - Apply once daily at night

*Mild inflammatory acne (with papules and a few pustules).*
Initially try topical antibiotics or topical antibiotics in combination with benzyl peroxide or adapalene (1 at either end of the day) or as combination products. Treatment is to be applied twice daily to all potentially affected areas for a minimum of 3 months. These agents may also be used after a course of oral antibiotics or Retinoids if small comedones of pustules persist. Topical treatment can be maintained for prolonged periods.

Agents recommended in the Wirral formulary include:-

**First choice**
**Benzoyl peroxide 5% / clindamycin 1% (Duac Once Daily®)** - Apply once daily in the evening
Or
**Isotretinoin 0.05% / erythromycin 2% (Isotrexin®)** - Apply thinly once or twice daily
**Adapalene 0.1%/benzoyl peroxide 2.5% (Epiduo®)**

*Inflammatory acne (with papules pustules and more inflammatory nodules).*

Usually requires oral antibiotics such as tetracyclines, erythromycin or occasionally trimethoprim (minocyclines are rarely used because of risk of hepatotoxicity and lupus like syndrome). Treatment must be given for a minimum of 3 months before moving to another agent if no response. Efficacy can be improved if oral antibiotics are combined with topical anti comedonal treatments if both comedonal and inflammatory lesions exist. Some
research suggests using Benzoyl peroxide in conjunction with oral antibiotics reduces the development of antibiotic resistant propionobacterium acnes.

Oral antibiotics recommended on the Wirral formulary are:-

First choice - severe inflammatory acne
**Oxytetracycline** 500mg, orally, twice daily. (emphasis to the patient that this must be taken on an empty stomach)

However, to increase compliance **Lymecycline** 408 mg, orally, daily may be a better first choice

Second choices
**Erythromycin** 500 mg, orally, twice daily
Or
**Doxycycline** 100 mg, orally, daily

Third choice
**Trimethoprim** 300 mg, orally, twice daily (unlicensed dose – check FBC 2 x year and warn re possibility of drug reaction)

Females with moderately severe acne, seborrhoea and a pre menstrual flare may benefit from Dianette (Ethinyloestradiol 35 micrograms, cyproterone acetate 2 mg), Co-cyprindiol, non-proprietory. This would also serve as a contraceptive should the patient require Isotretinoin.

If there is no response of inflammatory acne to the above then patients should be referred for consideration of Isotretinoin. An alternative for some females is spironolactone.

**Severe nodulo-cystic acne**
Those with severe inflammatory acne and especially those with significant scarring should be referred immediately for consideration of Isotretinoin. They should be started on an oral antibiotic whilst awaiting their appointment.

Acne Variants

**Macrocomedones**
These comprise large ‘whiteheads’ and usually affect the chin and cheeks. They do not respond to conventional treatment and require cautery before receiving isotretinoin otherwise a sever inflammatory response can occur.

**Acne Fulminans**
Sudden occurrence of severe inflammatory acne on the trunk and face with associated fever arthritis and lethargy. These patients require urgent referral and often require systemic steroids followed by isotretinoin.

**Acne Conglobata**
Usually found in males with clustered blackheads, sinus tracts, tender lesions and usually extensive scarring. Hidradenitis can sometimes co-exist. Treatment is difficult and patients should be referred immediately to secondary care.

**Acne Excoriee**

Usually affects young females who tend to have mild acne which has been ‘picked’. In most a simple explanation of the deleterious effect of this is all that is required but in some patients this forms part of a spectrum including dermatitis artefacta/dysmorphophobia. Treat with the less irritant topical agents.

**Persistent acne in the adult.**

If an adult over 25 continues with acne even mild to moderate in severity then consider referral for specialist advice as these patients often require isotretinoin.

**WHEN AND WHERE TO REFER**

**Refer Early**

- Severe acne
- Moderate acne which is only partially responded to treatment and starting to scar
- Inadequate response to at least 2 systemic antibiotics given for a minimum of 4 months each
- Patients with associated and severe psychological symptoms regardless of severity of acne

Patients with acne who have failed two full courses of oral antibiotic treatment combined with appropriate topical treatment, all patients with severe nodulo-cystic, conglobate acne and acne occurring in the adult should be referred for consideration of Isotretinoin.

*Include FBC, biochemical profile and lipids with referral and a negative pregnancy test within 7 days of the appointment if Isotretinoin is to be considered in a female of childbearing age to minimise appointments required.*

Fig 1 Comedonal Acne

Fig 2 Papules and Pustules
Fig 3 Nodules
ACTINIC KERATOSIS (SOLAR KERATOSIS)

DEFINITION

Dysplastic epidermal cell changes with superficial redness and scaling of the skin in sun exposed areas. Some can be hyperkeratotic. Squamous cell carcinoma (SCC) risk is low with an annual incidence of transformation estimated at < 1%. The risk is higher in immunocompromised patients.

CAUSE

Sun induced.

CLINICAL FEATURES

- Often multiple, discrete red scaling lesions on sun exposed areas.
- Commonly found on backs of hands, face and scalp (especially in men with male pattern baldness), Fig 1
- May develop into a cutaneous horn. Fig 2

DIFFERENTIAL DIAGNOSIS

1. Seborrhoeic keratosis/basal cell papilloma, these are usually larger and often pigmented and not restricted to sun exposed areas.
2. Bowen's disease, often larger, erythematous and scaly.
3. Squamous cell carcinoma – usually and indurated or nodular lesion which may ulcerate.
4. Viral warts are usually more hyperkeratotic with less redness.

INVESTIGATIONS

- None required
- If SCC is suspected refer urgently via 2 week pathway

MANAGEMENT

1. Emollient and observation - an option if there are not very many lesions and the patient is educated to return if lesions change or become nodular.
2. Sun protection – avoidance, sunscreens, hats and clothing.
3. Liquid nitrogen cryotherapy, one freeze/thaw cycle of 10 seconds using the ‘C’ nozzle of the Cryac® usually sufficient.
4. Efudix cream (5 Fluorouracil), useful especially if multiple lesions with field change.
   - Apply once or twice daily to affected areas for 3 weeks – can cause quite marked local irritation or inflammation to the lesions and surrounding skin. In the 4th week, 1% Hydrocortisone or Nystaform-Hydrocortisone ointment can be applied to reduce the inflammation.
5. Topical Diclofenac (3% gel, Solaraze). This is to be applied twice daily for 3 months usually not such a brisk inflammatory reaction as with Efudix but the prolonged treatment is sometimes difficult for compliance. Efficacy probably less than Efudix.

6. 5% Imiquimod (Aldara). Useful for resistant lesions or field change not responding to Efudix. Use 3 times weekly for 6 weeks. Causes significant morbidity.

7. 2% Imiquimod (Zyclara). Use for 2 weeks, 2 weeks off and then a further 2 weeks. Morbidity possibly less then for 5% cream and may be used as alternative to Efudix.

8. Actikerall (Fluorouracil and salicylic acid) paint - for isolated hypertrophic keratotic lesions.

9. Ingenol gel (Picato) – new 3 day treatment regime. Causes inflammation similar to Efudix. Short treatment regime may help with compliance. Currently under evaluation.

10. Curettage for single or particularly keratinous lesions. Tissue must be sent for histology.

11. Photodynamic therapy for widespread lesions

**Treatment ladder:**

- Thick and unusual – curettage & cautery for histology
- Cryotherapy with liquid N₂
- Actikerall paint
- Ingenol gel
- Imiquimod
- 5-Fluorouracil (Efudix) cream
- Diclofenac gel 3% use bd for 12 weeks
- Very thin lesions – may resolve with regular application of emollient.

Keratin horns may be curetted as above if no indurated base but tissue must be sent for histology as even these lesions often show early SCC at the base histologically.

**WHEN AND WHERE TO REFER**

- Extensive actinic changes
- Doubts about diagnosis
- Failure to respond to one or two treatments of cryotherapy or to a course of Efudix cream
- Suspicion of malignancy
- Immunosuppressed patients (e.g. post transplant, cytotoxic drugs)
• Referral should be made to any of the Consultant Dermatologists

Fig 1 Actinic Keratoses

Fig 2 Cutaneous Horn
ALOPECIA

DEFINITION

Alopecia is the general term for hair loss. This is often arbitrarily subdivided into localised and generalised, scarring and non-scarring forms.

CAUSE

1. Non Scarring
   The commonest causes are alopecia areata and male pattern hair loss. Other causes include telogen effluvium (secondary to pregnancy, systemic disease, emotional trauma etc) drugs and cytotoxic agents, endocrine abnormalities (hyper and hypo-thyroidism) and generalised skin disease e.g. exfoliative dermatitis/psoriasis. Rarer causes include congenital syndromes (hair shaft defects, loose anagen syndrome etc) trichotillomania and tinea capitis

2. Scarring Hair Loss
   The commonest cause is hair loss associated with the specific skin diseases; discoid lupus and lichen planus. A particular variant of lichen planus affects perimenpausal women, the scarring alopecia affecting only the frontal and later the temporal areas of the scalp; so called frontal fibrosing alopecia. Less common causes include localised scleroderma, trauma, burns and inflammation associated with fungal infection (kerion) or severe bacterial folliculitis.

CLINICAL FEATURES

- Alopecia areata
  An auto-immune disease which presents as single or multiple areas of discrete areas of alopecia with a normal scalp. Round the edge, exclamation mark hairs are usually identifiable which confirm the diagnosis and also imply that hair loss is active. In the re-growing phase, hair starts to re-grow centrally as fine downy white hair which gradually thickens and takes on the normal colour of the patient's terminal hair. Loss of all the hair on the head is termed alopecia totalis and loss of all head and body hair is alopecia universalis.

- Androgenic alopecia
  In males this usually starts with temporal recession, loss of hair over the vertex and then gradual merging of the two to leave total loss of hair over the crown. In females the frontal margin is often maintained with hair loss gradually progressive behind it. In both male and female pattern alopecia the hair on the parietal and occipital scalp usually remains normal.

- Telogen Effluvium
  General hair loss (with normal scalp) usually follows 2 to 3 months after systemic illness, major emotional shock and childbirth.

- Tinea capitis
  This is usually found in children and whilst tinea infection in adults is not impossible to find it is quite rare and is usually due to infection from an animal based Dermatophyte fungus. This produces an area of localised alopecia with scaling and hairs often broken off at different lengths.

- Trichotillomania
Self inflicted hair loss, usually female and may be part of an obsessive compulsive disorder. Areas of hair loss seem bizarre and broken hairs are all of the same length.

DIFFERENTIAL DIAGNOSIS

1. Chronic Discoid Lupus
2. Lichen Planus

Both these conditions cause erythema and scaling of the scalp which if untreated causes scarring with permanent hair loss.

INVESTIGATIONS

- These are usually unhelpful.
- If tinea capitis is suspected, samples must be sent for mycology.
- If scarring alopecia is present scalp biopsies should be carried out to exclude discoid lupus, lichen planus etc.
- Persistent folliculitis should be swabbed to guide antibiotic therapy.
- Hormonal investigations are usually not required in androgenic alopecia unless associated with other signs suggestive of "virilisation"
- Patients with generalised non-scarring alopecia of unknown cause should have a full blood count, iron studies and thyroid function measured; as abnormalities of any can cause generalised hair thinning or loss.

Unfortunately treatment for most types of alopecia is limited and management in many cases involves sitting down with the patient and explaining the natural history and realistic expectations for the individual condition.

Important to remember that all males and females experience some degree of hair thinning and loss as they get older.

MANAGEMENT

- Tinea capitis should be treated with systemic anti-fungal agents.
- Discoid lupus/lichen planus should initially can be treated with topical steroid. If these fail to help then intralesional steroids (if localised) or second line agents such as antimalarials are required to prevent further scarring and permanent hair loss.
- Treatment of androgenic alopecia is unsatisfactory. Regaine (topical Minoxidil) will sometimes help patients but is not NHS-prescribable and any benefit is gained is rapidly lost when treatment is stopped which is often harder for the patient to cope with than the gradual loss they would have otherwise had. Oral Cyproterone maybe helpful in females and Finasteride may possibly with time become a helpful treatment in males (not available on the NHS).
- Treatment for alopecia areata is generally unhelpful but sometimes a topical steroid will reduce progression and help stimulate re-growth. Intralional triamcinolone given regularly, every month, may stimulate regrowth. Systemic steroids have occasionally been given for widespread alopecia areata but any benefit is usually lost as soon as they are stopped. Treatment for alopecia totalis and universalis is
usually unhelpful. Some academic centres try topical immunotherapy with varying degrees of success.

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- 
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WHEN AND WHERE TO REFER

1. Patients where the diagnosis is unclear.
2. Patients where a specific dermatosis is proving difficult to manage.
3. Patients with scarring alopecia for diagnosis, biopsy and aggressive treatment.
BASAL CELL PAPILLOMAS  
(SEBORRHOEIC KERATOSES/WARTS)

DEFINITION

A benign epidermal tumour that has no relation to sebaceous glands.

CAUSE

Usually thought to be ‘age related’ but:

- Sometimes follows an inflammatory dermatosis (William’s warts)
- Very rarely associated with internal neoplasm (sudden eruption of many itchy lesions – Lesser Trelat syndrome)

CLINICAL FEATURES (Fig 1)

- May be single but often multiple
- Have "stuck on" appearance
- Colour varies from yellow through brown to near black
- May be flat, raised or pedunculated
- Usually occur over age 50
- Surface may have greasy scaled appearance

DIFFERENTIAL DIAGNOSIS

- Usually easily recognised
- Rarely can be confused with pigmented basal cell carcinoma or importantly with a malignant melanoma

INVESTIGATIONS

Nil except in case of diagnostic difficulty.

MANAGEMENT

Most can be left alone with reassurance only. Treatment is usually only warranted if lesions are symptomatic because repeated trauma or recurrent infection. Treatment is usually by curettage & cautery under local anaesthesia (which gives histological confirmation). Cryotherapy may be effective for relatively flat lesions.

WHEN AND WHERE TO REFER

Only for diagnostic difficulty to dermatology clinic.
BENIGN MELANOCYTIC NAEVI (MOLES)

DEFINITION

Melanocytic naevi arise as a result of proliferation of melanocytes, the cells in the skin that produce pigment. They can be classified histologically as junctional, compound and intradermal naevi or as congenital or acquired.

CAUSE

Benign melanocytic naevi or moles, are extremely common. Acquired naevi occur after birth, they become more common during childhood and adolescence and then reduce in prevalence during adult life.

Risk factors:

- Sun exposure - increases the number of acquired naevi in childhood.
- Immunosuppression (drugs, chemotherapy) - an increased number of naevi is seen in children who have been immunosuppressed.
- Increased number of benign moles increases risk of melanoma.
- Genetic factors: Family history of melanoma.

Evolution of a melanocytic naevus

- **Junctional naevus** - in childhood the melanocytes of the naevus are situated at the junction between the epidermis and the dermis. These tend to look dark but small, flat and symmetrical.
- **Compound naevus** – during maturation melanocytes may migrate down to the dermis and form nests of cells. This combination of junctional cells plus dermal cells is called a compound naevus. These moles tends to be protuberant, dome shaped and can have a warty papillomatous surface. Fig 1
  
  This migration may be accompanied by growth of the naevus and a deepening in colour. This phenomenon usually causes a great deal of parental anxiety and precipitates a visit to the GP and often an urgent referral to a dermatologist for an opinion.

- **Intradermal naevus** - the junctional cells cease proliferating and the dermal cells only remain. The mole tends to lighten in colour and is dome shaped. Fig 2

CLINICAL FEATURES

The appearances of naevi are variable. The colour may vary from a deep brown, blue or black to flesh coloured. It is normal for naevi to increase in number, size and thickness during late childhood and adolescence. It is normal for naevi to have hair growing through them. It is normal for naevi to disappear in later life. Other normal phenomena include;

**Halo naevus.** A white halo developing around a naevus is a regular occurrence in adolescence and is not a worrying feature. In an adult, the development of a halo should be viewed with more suspicion Fig 3..

**Meyerson’s naevus.** An area of eczema around a naevus.
**Naevus spilus or speckled naevus.** Large, maybe a few cms, with multiple areas of speckled pigment within it.

**Naevus en cocarde.** There are concentric rings of pigment around a central darker junction naevus.

**Blue naevus.** Pigment is in the lower dermis, it is an optical effect giving the naevus a deep blue colour. Always uniform in colours and does not change. Fig 4

**DIFFERENTIAL DIAGNOSIS**

1. Freckle  
2. Dermatofibroma  
3. Basal cell papilloma.

**INVESTIGATIONS**

- Full skin examination  
- Dermoscopy

**MANAGEMENT**

- Reassurance – the overwhelming majority of naevi listed above are not premalignant. There is no indication on medical grounds to excise these lesions. Patients should be advised that naevi are a normal feature of the skin.  
- Self examination - parents and patients should be educated to examine their moles (ABCD checklist (see melanoma chapter).  
- Photography – of moles if multiple.  
- Sun protection advice.  
- Protuberant naevi at frictional sites that are repeatedly traumatized can be removed by shave excision (all specimens must be sent for histology, be sure the lesion is benign).

**WHEN AND WHERE TO REFER**

- Doubt in the diagnosis.  
- History of change in size, shape and colour, spontaneous bleeding or altered sensation.  
- If concerned about diagnosis of melanoma, refer on 2 week referral pathway.

**CONGENITAL MELANOCYTIC NAEVUS**

These are present at birth. Approximately 1% of newborns have congenital melanocytic naevi (CMN).

**Features:**

- Size – larger that acquired naevi.  
- Colour and texture – getting darker, raised, papillomatous, hairy with age Fig 5.  
- Giant lesions (>20 cm in adulthood) are at risk of developing melanoma.
Neurocutaneous melanosis - risk correlates to the size of the naevus and presence of satellite lesions. Usually asymptomatic but can have neurological symptoms of delayed development or seizures.
- Refer to dermatologist for long term follow up if lesion is large.
- Photography.
- MRI scan only if neurological symptoms develop.

**ATYPICAL MELANOCYTIC NAEVUS**

Atypical melanocytic naevi (AMN) have clinical and histological features somewhere between a benign mole and a melanoma. They occasionally progress to melanoma.

**Features:**
- Family history + of atypical moles and melanoma and sun exposure
- Irregular in shape, colour and border
- Largish (>7 mm) lesions

**Atypical mole syndrome**

- Be aware of this condition
- Large number (>50)
- Morphology - large irregular looking moles
- Family history of multiple moles and melanoma
- Risk of developing melanoma.
- Photography
- Should be referred to a dermatologist for skin surveillance.
Fig 1 Papillomatous compound naevus

Fig 2 Skin coloured intradermal naevus

Fig 3 Halo naevus
Fig 4  Blue naevus

Fig 5  Congenital melanocytic naevus

Fig 6  Atypical mole syndrome
BENIGN SKIN LESION

Only refer benign skin lesions to Dermatology if there is a problem with diagnosis. We do not routinely remove benign lesions for cosmetic reasons unless there is doubt about diagnosis or they are significantly symptomatic.

If there is no diagnostic doubt, there is a good Community Minor Surgery Service and GPs should refer benign lesion to this service.

Examples of typical benign lesions:

**Seborrhoeic keratosis Fig 1**
- Tends be multiple in middle aged and elderly
- Variable appearance from subtle tanned dry macule - brown / black warty plaques
- ‘Stuck on’ appearance

**Dermatofibroma Fig 2**
- Occurring in younger patients but can occur at any age
- Usually on lower limbs, upper arms and the back
- Firm dermal nodule
- ‘Dimpling’ sign when pinched Fig 3
- Can be raised or protuberant

**Neurofibroma**
- Soft skin coloured nodules

**Angioma, haemangioma (Campbell de Morgan spots)**
- Vascular
- Red in colour
- Multiple, well defined
- Dermoscopy shows multiple red or purple lacunae

**Fibroepithelial polyps (skin tags)**

**Intradermal naevus**

See Benign Melanocytic Naevi chapter
Fig 1  Seborrheic keratosis

Fig 2  Dermatofibroma

Fig 3  Dermatofibroma (dimpling sign)

Fig 4  Angioma
BOWEN'S DISEASE

DEFINITION
A persistent, non elevated, red scaly occasionally crusted plaque usually found on the extremities. It is an intra-epidermal carcinoma that has a potential, possibly 20%, risk for malignant change.

CAUSE
Probably due to sunlight, but is certainly commoner than expected on sites of previous trauma. Historically was associated with arsenic exposure.

CLINICAL FEATURES
Can occur anywhere on the skin's surface and also on mucosal surfaces. Usually presents as a red scaly and usually asymptomatic area. Removal of the scale usually leaves a moist reddened surface. Lesions usually do not bleed and may often be hyperkeratotic or crusted. If ulceration occurs this is a possible sign of the development into an invasive carcinoma.

DIFFERENTIAL DIAGNOSIS
Lichen simplex, nummular dermatitis and psoriasis. However Bowen's disease is usually a single scaly patch, whereas the others usually present as multiple lesions.

INVESTIGATIONS
Biopsy or referral may be necessary in equivocal cases.

MANAGEMENT
- Treatment should only be offered if confident in the diagnosis or the lesion has been proved by biopsy.
- Cryotherapy, but this should be undertaken with care on the lower legs.
- Curettage and cautery will yield specimens for histological examination, but may be slow to heal with poor cosmetic result on the lower legs of females.
- Topical 5 Fluorouracil cream can be very effective, applied in the conventional manner twice a day for 21 days then followed by a steroid/antibiotic combination to reduce inflammation and the risk of secondary infection.
- Excision is usually not necessary, the lesions often too large for excision and primary closure and destruction of the epidermis is all that is necessary anyway.
- Photodynamic therapy may be particularly useful on the lower legs of the elderly where other modalities may cause ulceration.

WHEN AND WHERE TO REFER
Referral should be made:

1. If there is doubt over the diagnosis, or
2. If the lesion is ulcerated, thickened or bleeding therefore indicating the development into invasive squamous cell carcinoma.
BULLOUS ERUPTIONS

DEFINITION

Bullous eruptions can be divided into three broad groups:

- congenital and inherited bullous disorders
- bullous reactions
- acquired immunological bullous disorders.

The first group are all extremely rare and a general practitioner with an average list size may only see one or 2 cases in their working lifetime. Such diseases will include conditions such as epidermolysis bullosa and are not in the remit of this booklet. Any neonate, baby or child with blisters or bullae should be referred immediately for a specialist opinion.

The second group are the most common and will include all conditions resulting in a bullous reaction. This may be secondary to infection e.g. bullous impetigo or rarely (and usually in children) staphylococcal scalded skin syndrome or an external cause such as insect bites, a thermal or chemical burn, phytophotodermatitis (a phototoxic reaction to photosensitizing chemicals in plants such as rue or hogweed) or a bullous drug reaction (see section on drug eruptions).

The third group comprises the immunological bullous diseases in which auto antibodies are produced against specific target antigens within the skin. The site of these target antigens dictates where the split in the skin will occur and thus the disease pattern. Most GPs will have patients with bullous pemphigoid and will come across patients with pemphigus and dermatitis herpetiformis and so these will be dealt with in more detail.

BULLOUS PEMPHIGOID

CAUSE

This is the most common of the immuno bullous diseases and is seen largely but not invariably in the elderly. The cause is unknown but the fact that the disease is more common in those with neurological disorders might raise the possibility that autoantibodies are formed which cross react with both tissues.

CLINICAL FEATURES (Fig 1 & 2)

Large tense unilocular blisters occur on an erythematous often urticated base, favouring the flexural aspect of the limbs, the groin is frequently involved. Can be localised usually on the lower leg. The condition is usually very itchy. Mucosal lesions are rare.

DIFFERENTIAL DIAGNOSIS

The diagnosis is usually obvious in classic cases but if the presentation is localized must be differentiated from bullous impetigo, acute eczema, shingles and other external causes of localised blistering.
INVESTIGATIONS

Referral for a specialist opinion is mandatory to confirm the diagnosis clinically, histologically and immunologically as treatment will require steroids and often immunosuppressant drugs in a population where comorbidities might make these agents difficult.

TREATMENT

The mainstay is usually with topical and/or systemic steroids. If control requires significant doses long term then steroid sparing agents such as azathioprine, methotrexate, ciclosporin, mycophenolate mofetil, dapsone or doxycycline may be needed.

PEMPHIGUS VULGARIS (Fig 3)

Pemphigus is an extremely rare condition and there are many different clinical variants of pemphigus depending on the level of the split within the epidermis (pemphigus vulgaris, pemphigus foliaceus etc).

CAUSE

The disease is due to antibodies to various intercellular antigens, the profile of which determines the clinical phenotype of the patient regarding skin and oral involvement.

CLINICAL FEATURES

It is very rare for pemphigus to present as blisters and the condition usually presents as erosions of the skin. The mucous membranes are invariably affected. Trauma to the skin may induce blistering, the epidermis may be sheared off very easily (Nikolsky sign).

INVESTIGATION

Pemphigus is a severe illness with a considerable morbidity. Treatment is with aggressive immunosuppression and so specialist referral is essential to obtain a definite diagnosis before treatment.

TREATMENT

Treatment is with high dose steroids often combined with immunosuppressive drugs such as azathioprine, methotrexate, mycophenylate mofetil, cyclophosphamide etc.

DERMATITIS HERPETIFORMIS

CAUSE

This condition is due to the deposition of IgA antibodies along the dermo-epidermal junction and it is usually associated with gluten sensitive enteropathy.
CLINICAL FEATURES

Dermatitis herpetiformis an extremely itchy skin disease consisting of small blisters usually on the knees, elbows, buttocks and shoulders. As it is intensely itchy, due to scratching it is unusual to see blisters.

INVESTIGATION

As treatment is with a specific agent it is important to establish the diagnosis with a skin biopsy histological and immunological confirmation. If confirmed, patient will usually be referred to gastroenterology for jejunal biopsy.

TREATMENT

This is with dapsone. In addition, most patients will be started on a gluten free diet.

EPIDERMOLYSIS BULLOSA AQUISITA (mimics porphyria cutanea tarda) and LINEAR IGA DISEASE (blisters around the neck and groin with the ‘string of pearls’ sign and usually in children) are rare diseases that are sometimes only discovered after biopsy.

WHEN AND WHERE TO REFER

Once common infectious, drug and external causes of localized blisters have been excluded most patients with significant blistering should be referred urgently to secondary care for prompt diagnosis and instigation of the appropriate therapy.

Fig 1 Localised bullous pemphigoid

Fig 2 generalised Bullopus pemphigoid
Figure 3 Pemphigus
CHONDRODERMATITIS NODULARIS HELICIS CHRONICUS (CDNH)

DEFINITION

Painful raised nodule or papule on the ear – usually the pinna in males and ante tragus in the female.

CAUSE

Possibly pressure effect but probably idiopathic.

CLINICAL FEATURES

- Non-pigmented, raised nodule or papule on the ear may ulcerate.
- The lesions are usually extremely tender commonly causing pain in bed when lying on the affected side.

DIFFERENTIAL DIAGNOSIS

1. Actinic keratosis: these usually have less substance to them and are more scaly and are not usually painful.
2. Basal cell carcinoma: differentiation can be difficult at times but these usually have a rolled edge with central depression and telangiectatic vessels around. Basal cell carcinomas are not usually as exquisitely tender.
3. Squamous cell carcinoma: usually on the back of the ear, tenderness and pain a late feature.

INVESTIGATIONS

None appropriate in primary care.

MANAGEMENT

- Reassurance. If the diagnosis is clear cut reassurance may be all that is necessary.
- Liquid nitrogen cryotherapy, this can sometimes make the lesions smaller or even cause them to regress completely.
- Haelan tape applied to affected area. May take 6-8 weeks for any benefit
- Pressure relieving pillow.

WHEN AND WHERE TO REFER

1. If diagnosis in doubt.
2. If very painful and no response to liquid nitrogen or haelan tape.
3. Referral is suggested to any Consultant Dermatologist where treatment options will include curettage, excision of the underlying inflamed cartilage and occasionally intralesional steroid injections.
DRUG REACTIONS
(Adverse Cutaneous Drug Reactions [ACDR])

DEFINITION

The incidence of drug reactions is high. Due to under-reporting the true figure is unknown but has been estimated at 2-3% of drug treatments with 1:40 of GP consultations being related to ACDRs. The incidence increases with age but this may just reflect the fact the elderly are more likely to be on multiple medications.

CAUSE

There are many different types of reaction and any drug can cause any of them. Reactions usually occur within 4-14 days of starting the drug and settle soon after stopping but the time scale may be shorter or longer (e.g. thiazides causing subacute cutaneous lupus). Common drugs causing reactions include:

- Antibiotics (usually mild but sometimes acute (see AGEP)
- Sulpha drugs (antibiotics, sulphasalazine, sulphonylureas, diuretics (e.g. hydrochlorothiazide)
- Allopurinol
- NSAIDs
- Diuretics
- Antiepileptics

Common Drugs Reactions include:

**Annular erythema**
An easily recognised pattern with an expanding ring of palpable erythema. There may be just one lesion or many on the trunk and limbs. Cimetidine, penicillin and anti malarials classically give this type of reaction.

**Cutaneous Ulcers**
Nicorandil (Fig 1) can cause perianal/peristomal ulcers. Skin ulcers can occur in patients on hydroxycarbamide. Warfarin and heparin can both cause necrosis.

**Erythema Nodosum (Fig 2)**
Causes may include oral contraceptives and antibiotics.

**Exanthematous Reactions**
The most common type of reaction is the exanthematous or morbilliform type. Antibiotics, particularly the penicillin group, are the most frequent cause and it may occur up to 2 to 3 weeks after the patient has stopped taking the causative drug.

**Fixed drug eruption**
A reaction that recurs on the same site each time the drug is taken. Characteristically the lesions occur within 12 hours of taking the drug and consist of well demarcated, oval, erythematous and oedematous plaques. It may blister and usually there is post inflammatory hyperpigmentation when the lesion resolves. Any drug can cause this
reaction but antibiotics (tetracycline’s and sulphonamides) and NSAIDs are frequent culprits. Occasionally it can result from OTC preparations, laxatives and so a detailed history is needed.

**Lichenoid eruption**
This type of drug reaction resembles the clinical and histological appearance of lichen planus, however a lichenoid drug reaction tends to be more extensive than in idiopathic lichen planus and mucous membrane lesions are unusual. Lichenoid eruptions may occur some months after the drug was started. The drugs most likely to cause this type of reaction include beta blockers, thiazide diuretics, gold and antimalarials.

**Lupus Erythematosus Like**
Occasional triggers include the thiazide diuretics (SCLE) and minocycline (used less commonly because of this side effect).

**Purpura**
Some drugs can cause thrombocytopenia, so present as purpura. Also many drug eruptions would have a purpuric element to them.

**Urticaria**
The vast majority of cases of urticaria are idiopathic but occasionally this can be due or exacerbated by aspirin, non steroidal anti inflammatory drugs and ACE inhibitors. Urticaria usually occurs within 24 to 48 hours of taking the causative drug.

**Vasculitis (Fig 3)**
This purpuric rash is palpable and may also blister. It is most marked on lower extremities. Most cases are due to ‘bugs and drugs’ and settle spontaneously. More severe cases may have internal involvement and may present with abdominal pain and haematuria.

Severe Drug Eruptions with significant morbidity/mortality include:

**Anaphylaxis**
- Usually severe and may be fatal. The reaction is rapid. There is usually urticaria, angio-oedema, bronchospasm and vaso motor collapse.
- The most common causes are contrast media for x-ray examination and antibiotics (penicillin, cephalosporins, sulphonamides, tetracyclines), aspirin, NSAIDs and ACE inhibitors.

**Acute Generalised Exanthematous Pustulosis (AGEP)**
- Rapid onset of erythema with pustulation within 24 hours of drug administration. Often starts on face/flexures disseminating rapidly.
- Common causes are penicillins, macrolides, quinolones, antimalarials, but a host of other drugs have been implicated. The eruption usually settles on drug withdrawal but fatalities have been reported.

**Drug Rash with Eosinophilia and Systemic Symptoms (DRESS)**
- Generalised eruption associated with fever, malaise and organ involvement (hepatitis, nephritis, pneumonitis). Blood eosinophilia is significant.
- Commonest causative agents include the anticonvulsants, allopurinol, minocycline and sulphasalazine and the eruption may take several weeks to develop.
• The condition has a significant morbidity and requires systemic steroids often for several weeks.

**Erythroderma and exfoliative dermatitis**
• A severe life threatening condition. May occur de novo or may complicate any drug eruption. Very dangerous in the elderly due to problems with temperature regulation, fluid balance, risk of infection and high output cardiac failure.
• Common causes include sulphanamides, isoniazid, penicillin, phenytoin, captopril and cimetidine.

**Erythema multiforme and Stevens Johnson’s syndrome (Fig 4)**
• The target lesion is the classic sign associated with this but many other lesion types may be present including macules, papules, urticaria, vesicles, bullae and purpura.
• The rash is usually found on the distal extremities and lesions on the soles of the feet and palms of the hands maybe diagnostic.
• Erythema multiforme is most commonly triggered by herpes simplex infections but sulphonamides and anti epileptics particularly phenytoin may be triggers.
• Stevens Johnson's syndrome occurs when fever, malaise, myalgia and arthralgia and mucous membrane involvement accompany severe erythema multiforme. In Stevens Johnson’s syndrome the area involved is <10% with greater involvement classified as overlap or Toxic Epidermal necrolysis if >40% involvement.
• Common causes include allopurinol, sulpha drugs, anticonvulsants (carbamazepine, lamotrigine, phenytoin) and ‘Oxicam’ NSAIDS (peroxicam, meloxicam) and Rituximab

**Toxic Epidermal Necrolysis (TEN) (Fig 5)**
• This is the severe form of the erythema multiforme/Stevens Johnson’s syndrome spectrum. In TEN there are usually systemic symptoms of fever and malaise before the mucocutaneous eruption occurs.
• There is invariably mucous membrane involvement with erosions in the mouth, nose, conjunctiva and genital mucosa, associated with widespread epidermal loss. This is a severe life threatening disease and carries a significant mortality risk.

**Photo sensitivity Reactions (Fig 6)**
• Photosensitive reactions may be either photo toxic or photo allergic.
• Photo toxic reactions are more common, usually presenting as sunburn-like eruptions occurring within 24 hours of taking the drug. Common causes include tetracyclines and phenothiazines.
• Photo allergic reactions require a period of sensitisation before the reaction occurs and may extend beyond the light exposed sites. Sensitisation may occur either via contact or systemic administration. Common causes include thiazide diuretics, sulphonamides and griseofulvin..
• Other less common reactions may include pigmentation reactions, acneiform and pustular eruptions, bullous eruptions, erythema nodosum, lupus erythematosus like syndromes, scleroderma like reactions, pseudolymphomatous eruptions and oral lesions..
MANAGEMENT

Most drug eruptions are self evident but it is important to remember that some may relate to over the counter medication, herbal tonics and health supplements many patients will not realise might be responsible. It is also important to realise that some reactions may take longer than normal to evolve (e.g. thiazides in LE) or subside (e.g. drugs causing pseudoporphyria).

The suspect drug must be stopped and most reactions will subside without sequelae. The ultimate diagnostic proof is rechallenge though this may be dangerous, therefore it is usually not performed.

WHEN AND WHERE TO REFER

- When there is doubt in the diagnosis
- In all severe cases of drug reactions particularly DRESS, AGEP, erythema multiforme/Stevens Johnson's syndrome/TEN, severe photosensitive reactions and lichenoid reactions.
- All cases of erythroderma, exfoliative dermatitis and toxic epidermal necrolysis must be referred for urgent opinion and management.
- Referral for allergy testing to a particular drug is often unrewarding and difficult to interpret.

Fig 1 Nicorandil Perianal Ulcer

Fig 2 Erythema Nososum
Fig 3 Vasculitis

Fig 4 Stevens Johnson Syndrome
Fig 5 Toxic Epidermal Necrosis (TEN)
ECZEMA - ATOPIC

DEFINITION

Eczema and dermatitis are terms which are interchangeable. It describes an itchy and dry skin condition. The history, age of onset, clinical distribution and prognosis help to determine the aetiology of the eczema.

- **Atopic** – inherited “baby eczema” often associated with asthma and hay fever. Positive family history. Fig 1
- **Discoid** – well rounded patches which can be confused with psoriasis. Sometimes associated with atopy.
- **Pityriasis alba** - subtle hypopigmented slightly scaly patches, usually on cheeks of darker skin children.
- **Asteatotic** – due to dryness of the skin as the patient ages. Fig 3
- **Seborrhoeic** – cause of cradle cap (infantile), facial and flexural rash, and secondary to yeast infection. Fig 4
- **Varicose** – on lower legs with varicose veins. Susceptible to contact allergies from topical treatments.
- **Vesicular/Pompholyx** – affects palms and soles, usually endogenous in origin and very difficult to treat (beware unilateral rashes in these areas – think of fungus). Fig 5
- **Contact** (*allergic or irritant*) caused by contact with an allergen or continual contact with irritants.

All of these types of eczema can present as acute or chronic (weeping, or dry and thickened/lichenified ).

CLINICAL FEATURES OF ATOPIC ECZEMA (AE)

- Common disease affecting up to 20% of children.
- Typically presents early at around 3 months of age.
- Tendency to spontaneous improvement; 20% by the age of 2 years, 30% by 3 years and complete clearance by teens in 80-90%.
- Involvement of the face in infancy with a adoption of flexural distribution Fig 2 by the age of 18 months. Others areas such as wrists, hands and feet and trunk in more severe cases.
- Association with asthma and hay fever
- Skin infections with:
  - Staphylococcus aureus is common. S.aureus can act as a ‘super antigen” to exacerbate AE with frank infection.
  - Viruses e.g. Herpes simplex causing eczema herpeticum Fig 6  
    Pox virus causing molluscum contagiosum

DIFFERENTIAL DIAGNOSIS

1. Seborrhoeic eczema
2. Others (see above)
3. Cases not involving the typical sites can cause diagnostic difficulties. Sometimes only the passage of time can differentiate between childhood atopic and
seborrhoeic eczema (better prognosis, children growing out within 12-18 months of age).

INVESTIGATIONS

In majority of patients, no investigations are required.

- Raised IgE in the patients or a first degree relative indicates atopy.
- Skin swabs for bacterial infection.
- Viral swabs (use special transport medium) if eczema herpeticum is suspected. Treat on clinical grounds rather than wait for virology results.

MANAGEMENT

EDUCATION

- Education should include advice about: -
  - Causation if possible.
  - Emphasis that in overwhelming majority of cases atopic eczema is not caused by an allergy.
  - Dietary manipulation should only be done under the supervision of a consultant allergist and a dietician
  - Chronic nature of the condition, lack of quick easy remedies and cure.
  - Discussion of realistic aims of treatment and prognosis.
  - Positive approach to encourage compliance, emphasising the tendency for spontaneous improvement with age.
  - Skin care routine such as how to use any topical medication that is prescribed.
  - Bathing is not harmful. Frequency should be discussed and a bath emollient considered.
  - Keeping fingernails short to avoid damage done by scratching and consideration of the use of cotton gloves at night.
  - Avoid aggravating factors – irritants, allergens, emotional stress and infection.
  - Clothing – avoid wool next to the skin, cotton clothing preferable.
  - Recommend National Eczema Society if appropriate.

PLAN OF CARE FOR INCREASING SEVERITY OF ECZEMA

Severe

2) EMOLLIENTS (LIGHT TO GREASY)
3) TOPICAL STEROIDS (MILD TO VERY POTENT)
4) ANTIBIOTICS
5) DRESSINGS
6) TOPICAL IMMUNOMODULATORS
7) SYSTEMIC
**EMOLLIENTS**

Dry skin is invariable in atopic eczema and emollient is a priority. The liberal use of emollients may reduce the need for topical steroids.

**Bath additives**
- Some contain antipruritics such as Balneum plus®
- Some contain antiseptics such as Emulsiderm® and Oilatum

**Soap substitute**
- Avoid soaps, shower gels and bubble baths. Encourage cream cleansers e.g., Diprobase, Cetraben, Dermol

**Emollients**
- Compliance – check and encourage.
- Individual variation in response, preference and tolerance to emollients. Emollients should be as greasy as tolerated. Patient choice is important to encourage compliance.
- Ointments are better for dry eczema and creams for more weepy areas. Large quantities of emollients need to be prescribed as they are needed regularly. A Skin care plan (enclosed) can help to encourage patients to use their emollients regularly.
- Topical
  - The following are listed in order of lightness to greasiness
    - Dermol 500®
    - Aveeno cream®
    - Diprobase cream®, Cetraben
    - E45 cream®, Doublebase gel
    - Emulsifying ointment, Hydrous ointment (Oily cream), Epaderm® and Hydromol ointment
    - White soft paraffin/Liquid paraffin (50/50)

They should all be used as often as possible and the less greasy ones can be used as soap substitutes.

**TOPICAL STEROIDS**

This is the mainstay treatment for inflammatory component of eczema. Topical steroid usage – used intermittently in the appropriate strength for the degree of eczema and its area on the body. Once eczema is under control, topical steroid can be stopped and patients should continue only with emollients until the next flare.

**TABLE OF POTENCY**

<table>
<thead>
<tr>
<th>Level</th>
<th>Example</th>
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</thead>
<tbody>
<tr>
<td>Mild</td>
<td>1% Hydrocortisone</td>
</tr>
<tr>
<td>Moderately Potent</td>
<td>Clobetasone 0.05% (Eumovate)</td>
</tr>
<tr>
<td></td>
<td>Flurandrenolone (Haelan)</td>
</tr>
<tr>
<td>Potent</td>
<td>Betamethasone ointment 1% (Betnovate)</td>
</tr>
</tbody>
</table>
1% Hydrocortisone does not cause skin atrophy and can be safely used on the face and flexures.

Ointments are more effective and contain fewer additives and preservatives.

Face – Hydrocortisone should normally be used. If more potent treatment is required, consider calcineurin inhibitors or referral.

Trunk and limbs – mild to moderate steroid should control most cases of eczema when prescribed in appropriate amounts (see Finger tip unit chart). Potent steroid may be needed to gain control and then to reduce to mild strength.

The potency should be kept to the lowest that is effective.

Avoid repeat prescriptions of potent steroid preparations.

TOPICAL STEROID + ANTIBIOTIC

Active or chronic eczema can be complicated by super added infection. It can explain deterioration in the condition and reduce response to steroids. Take a skin swab if in doubt.

This can usually be overcome by using a combination of an antibacterial/steroid preparation or systemic antibiotics if widespread. These should be used for short term (2 weeks at a time).

Consider:

- Antiseptic bath oils e.g. Oilatum Plus, Emulsiderm, Dermol preparations
- Steroid antibiotic combination
  - Fucidin H® - mild. For general eczema
  - Daktacort® - mild. Particularly for seborrhoeic eczema
  - Trimovate® - moderately potent.
  - Fucibet® - potent
  - Betnovate C® - potent
  - Synalar-C - potent

Sometimes for very wet, oozy eczema, diluted potassium permanganate can be helpful as a soak or bath. It can be prescribed as crystals (Permitabs®).

Recurrent infections – take nasal swabs from patient and family members. If positive, treat with Naseptin / Bactroban nasal ointments.

ORAL ANTIBIOTICS

- Systemic antibiotics useful against the commonest pathogen, Staph.aureus, are flucloxacillin and erythromycin. An average course would last 7-10 days according to disease severity.
• Systemic Aciclovir – effective against Herpes virus causing eczema herpeticum. Viral swabs can be taken, treat on clinical grounds rather than wait for virology results. If in doubt, particularly in children, refer urgently to secondary care.

CALCINEURIN INHIBITORS

These immunomodulators are not steroid based and are safe to use on the face and eyelids. Avoid using when there are signs of infection (bacterial and viral). Irritation with itching and burning can occur at initial use but soon subside. Can be used 2x/week for maintenance.

• Tacrolimus ointment – 0.1% (equivalent to a potent steroid but irritates initially). 0.03% is licensed for children over 2 years.
• Pimecrolimus 1% cream. Equivalent to a moderate potency steroid but better tolerated. Licensed for use over the age of 2.

ANTI-HISTAMINES

• Sleep management - an antihistamine with sedative properties (e.g. hydroxyzine, trimeprazine) particularly at night can be helpful. Can reduce the itch/scratch cycle. No evidence that non-sedative antihistamines benefit patients with eczema.

DRESSINGS

These are valuable ways to treat chronic unresponsive or relapsing areas of eczema, particularly if they are very itchy. Patients need to be motivated and this type of treatment should not be used if areas are obviously infected.

Wet wraps

• Oily bath
• Copious application of emollient or sparing application of steroid preparation
• Application of warm, wet Tubifast® / Clinifast garments followed by a layer of dry Tubifast®. These are best used overnight but can be used 24 hourly when eczema is at its worst. Avoid when infected.

Paste bandages

• Examples are Viscopaste, Steripaste, Ichthopaste and Zipzoc
• Apply topical medicaments to areas involved
• Cover with paste bandage
• Cover with Tubifast® to hold in place
• Best left on for at least 24 hours

DIETARY TREATMENT

• Role of food allergy is controversial and exaggerated.
• History of causal association of eczema flare and ingestion of food. Withdrawal of food group (for 6 weeks) and flare up with re-challenge is helpful. History of worsening of eczema within hours of ingestion. This history is much more important than the results of blood or skin tests for “allergies”.
• In “severe life ruling” eczema with a good history, exclusion diets may very occasionally be considered with dietetic supervision.
• Investigations (e.g. RAST, IgE) or skin tests (skin prick test and patch tests) are unhelpful.
• The use of soya formula from birth does not prevent or relieve eczema.

WHEN AND WHERE TO REFER

• Childhood eczema not responding to emollients and mild steroids with frequent flares, parental anxieties or difficulties with compliance.
• Erythrodermic eczema.
• Patch testing to detect cases of contact allergic dermatitis when suspected.
• Consideration of inpatient treatment and second line immunosuppressive therapy.

REFERENCES/BIBLIOGRAPHY

Support Group:
The National Eczema Society
Hill House
Highgate Hill
London
N19 5NA

Tel: 0870 2413604
www.eczema.org
Name: ..........................................................................................................................

Appointment date: ...........................................................................................................

Next appointment: .............................................................................................................

Nurse/doctor contact tel no: ..............................................................................................

Treatment: ........................................................................................................................

The fingertip unit method

FTU = Fingertip unit (adult)
1 FTU = 1/2 g of cream or ointment.
Measurement based on 5mm nozzle.

<table>
<thead>
<tr>
<th>Age Group</th>
<th>FACE &amp; NECK</th>
<th>ARM &amp; HAND</th>
<th>LEG &amp; FOOT</th>
<th>TRUNK (front)</th>
<th>TRUNK (inc buttocks)</th>
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<td>3</td>
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<td>31/2</td>
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<tr>
<td>6-10 years</td>
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<td>21/2</td>
<td>41/2</td>
<td>31/2</td>
<td>5</td>
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<tr>
<td>Adult</td>
<td>21/2</td>
<td>3</td>
<td>1</td>
<td>6</td>
<td>2</td>
</tr>
</tbody>
</table>
The total amount to apply for two weeks treatment twice daily

**Adults**

- Face and Neck 36g
- Trunk Front 96g
- Back 90g
- One Arm 48g
- One Hand 17g
- One Leg 81g
- One Foot 25g

**Children**

<table>
<thead>
<tr>
<th>AGE</th>
<th>Face and Neck</th>
<th>Arm and Hand</th>
<th>Leg and Foot</th>
<th>Trunk (front)</th>
<th>Trunk (back) Inc. Buttocks</th>
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<tr>
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<td>35</td>
<td>63</td>
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"Skin Care Plan for eczema"

**MOISTURISER:**

<table>
<thead>
<tr>
<th>Name of Treatment</th>
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<th>For how long?</th>
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<tr>
<td>Moisturiser</td>
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<td>In the Bath</td>
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<tr>
<td>Soap Substitute</td>
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</tbody>
</table>

**STEROIDS (inc combinations with antibiotic and/or antifungal):**

| For the Body      |            |             |
| For the Face      |            |             |
| For ...............|            |             |

**Prescribing Quantities**

(NB it is easy to underestimate the amount of topical preparation to apply)

For twice-daily application for 1 week an adult requires:

<table>
<thead>
<tr>
<th>Site</th>
<th>Quantity</th>
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</thead>
<tbody>
<tr>
<td>Face &amp; Neck</td>
<td>30g</td>
</tr>
<tr>
<td>Hands</td>
<td>30g</td>
</tr>
<tr>
<td>Scalp</td>
<td>25g</td>
</tr>
<tr>
<td>Arms or Legs</td>
<td>100g</td>
</tr>
<tr>
<td>Body</td>
<td>100g</td>
</tr>
<tr>
<td>Groins &amp; Perineum</td>
<td>25g</td>
</tr>
<tr>
<td><strong>Whole Body</strong></td>
<td><strong>300g</strong></td>
</tr>
</tbody>
</table>
Fig 1  Childhood atopic eczema

Fig 2  Flexural lichenification

Fig 3  Asteatotic eczema
Fig 4  Seborrhoeic dermatitis

Fig 5  Pompholyx (microvesicular) eczema

Fig 6  Eczema herpeticum
DISCOID ECZEMA (NUMMULAR DERMATITIS)

DEFINITION

A common endogenous itchy eczematous eruption most commonly in adult men occurring in discrete coin shaped lesions predominantly on the arms and legs.

CAUSE

Largely unknown, more frequent in the winter months many patients will report an exacerbation with stress and the lesions are often secondarily infected. A cause is rarely found and drugs or an allergic cause is unusual. Surprisingly the condition seems to be confined to managerial and professional classes and the more middle aged or elderly male. In younger patients, male and female, may be a manifestation of atopic eczema.

CLINICAL FEATURES

- By definition the lesions are circular. Fig1
- They may be in several stages of development some oozing and weeping, some dry and scaly. Fig 2
- They are usually itchy and lesions often occur symmetrically on either side of the body. The arms and legs are usually involved, the trunk may also be involved. Fig 3

DIFFERENTIAL DIAGNOSIS

1. Must be differentiated from psoriasis and tinea corporis. Psoriasis would be more widespread and have nail and scalp involvement.
2. Tinea corporis will have a typical scale at the edge of the lesion and will have a positive mycology.

INVESTIGATIONS

Swab for bacteriology. Skin scrapings if tinea corporis is suspected.

MANAGEMENT

- Surprisingly difficult, this condition needs a potent steroid, probably a steroid/antibiotic combination, must be in an ointment base.
- Treatment is often prolonged.
- Any secondary infection must be treated appropriately.
- Copious emollients are needed, Emulsifying ointment, Oily cream etc and these should be used instead of soap and frequent bathing should be avoided.
- Occlusive bandaging may speed recovery. Bandaging with Zinc paste bandages or Ichthopaste bandages is advised. These should be applied over the topical steroid and left on for 24 hours before changing.
- Recurrence is a problem and prolonged treatment is sometimes needed.

WHEN AND WHERE TO REFER
1. When there is doubt in the diagnosis.
2. Failure to respond to treatment.

Fig 1  Discoid coin shaped lesion

Fig 2  Weeping and crusty lesion

Fig 3 Symmetrical limb distribution

HAND ECZEMA

DEFINITION

Eczema (dermatitis) confines mostly to the hands with limited involvement of other parts of the body.

CAUSE

Common and can be debilitating condition
• Endogenous e.g. Atopic, discoid, stress, possibly increased sweating.
• Exogenous (irritant -v- allergic).
• Irritant – repeated exposure to irritants causing damage to the skin. Examples of irritants are water, chemicals such as detergents, solvents, foods, physical agents such as extreme of temperatures.
• Allergic – due to Type IV hypersensitivity e.g. Nickel, fragrances, chromate, acrylate glues (see Patch Test chapter).
• Reactive (id) – dermatophyte infection e.g. Tinea pedis.
• Occupational - must be considered in all cases. Can be a significant cause.

CLINICAL FEATURES

Hand eczema is very common particularly amongst those involved in wet work.

There are several different types of hand dermatitis:
• Pompholyx - intensely itchy microvesicles that occurring on the palms and sides of fingers. Some coalesce to form large blisters. Fig 1
• Hyperkeratotic palmar eczema Fig 2- thick areas of scale on the palms and soles associated with fissures. Differential diagnosis is psoriasis Fig 3.
• Wear and tear dermatitis.
• Fingertip eczema. Fig 4
• Discoid eczema.
• Recurrent focal palmar peeling. (keratolysis exfoliativa) Fig 5
• Ring eczema – irritant cause, dorsum of hands also affected.
• Chronic acral eczema.
• ‘Wet’ (blistering and weeping) or ‘dry’ (hyperkeratotic and fissured).phases can occur in the same patient.

HISTORY

To include personal history of atopy, occupational history, domestic duties, hobbies, gardening, sports, etc.

DIFFERENTIAL DIAGNOSIS

Other skin condition can mimic hand eczema. Examine the rest of the patient’s skin including the feet for clues.

• Psoriasis.
• Infection (especially if unilateral, consider tinea). Fig 6
• Scabies.

INVESTIGATIONS

• Examine the rest of the patient.
• Skin scrapings, swabs.
• Referral for patch testing.

MANAGEMENT

General hand care advice
• Patient information leaflet.
• Avoidance of trigger factors e.g.
  - Avoidance of wet work, detergents extremes of temperatures
  - Use of soap substitutes
  - May necessitate change in occupation
• Proper hand drying.
• Hand protection.
• Emollients – applied frequently after every hand wash, be guided by patient’s choice. See Wirral formulary.
• Gloves for house and occupational work e.g. Cotton, household rubber, vinyl (PVC), nitrile, gardening.

THERAPIES

• Topical steroids – use appropriate potency (usually at least moderately potent). Hydrocortisone is rarely helpful. Ointment base is more moisturizing. Avoid oral steroids (rebound phenomenon). Consider Diprosalic in hyperkeratotic eczema.
• Occlusion – cotton gloves at night helps. Polythene (with clingfilm or plastic gloves) for a limited period.
• Potassium permanganate soaks helpful for wet, weeping phase.

• Haelan tape for fissures.

WHEN AND WHERE TO REFER

• Severe cases unresponsive to treatment and necessitating potent steroids under occlusion.
• Consideration of second line therapies e.g. azathioprine, retinoid (acitretin, alitretinoin), ciclosporin.
• Occupational cause.
• Patch testing  Fig 7– in suspected allergic contact aetiology.

Fig 1  Microvesicular eczema
Fig 2  Hyperkeratotic palmar eczema

Fig 3  Palmar psoriasis (well defined plaques)

Fig 4  Fingertip dermatitis in a hairdresser

Fig 5  Keratolysis exfoliativa (superficial peeling)
Fig 6 Tinea manuum mimicking hand dermatitis (unilateral asymmetrical distribution)

Fig 7 Example of patch testing
SEBORRHOEIC DERMATITIS/ECZEMA

DEFINITION

Erythema and scaling of naso-labial folds, eyebrows, ears and anterior chest. Often associated with dandruff.

CAUSE

Malassezia yeast.

CLINICAL FEATURES

- Scaling and erythema mainly of the face, Fig 1 but often the anterior chest wall too. Eyebrows and nasolabial folds Fig 2 are predominantly affected.
- Dandruff and scaling in scalp. Fig 3
- Blepharitis and otitis externa may be associated.
- Other skin folds may also be affected e.g. axillae, infra mammary, crural and gluteal folds. Fig 4
- Tends to recur following treatment.

DIFFERENTIAL DIAGNOSIS

1. Atopic eczema although the distribution is usually different.
2. Psoriasis on the face
3. Rosacea, this predominantly affects the cheeks, nose and chin. There is no scaling and usually presents with erythema with or without pustules. Rosacea and seborrhoeic dermatitis occur together quite often
4. Sudden severe development of or exacerbation of seborrhoeic dermatitis may be a sign of early HIV infection.

INVESTIGATION

None required.

MANAGEMENT

- There is no 'cure' and treatment may need to be long term.
- To the face and chest, Daktacort or Canesten HC cream.
- Trimovate cream to chest and flexural areas.
- For the scalp, tar shampoo, Selsun shampoo or Ketoconazole shampoo.
- Often worth using Ketoconazole shampoo as a facial wash and to the chest and flexural areas. Ketoconazole can be used regularly for a few weeks then often worth using once a week or so as maintenance.
- Topical calcineurin inhibitors, Tacrolimus and Picroloimus have been shown to be effective in facial seborrhoeic dermatitis.
WHEN AND WHERE TO REFER

1. If diagnosis in doubt.
2. If it does not respond to a combination of the above creams and shampoos.

Fig 1

Fig 2  Nasolabila involvement

Fig 3  Scaling of scalp

Fig 4 Flexural (axillary eruption)
VARICOSE (GRAVITATIONAL) ECZEMA

DEFINITION
An eczematous reaction of lower legs secondary to venous insufficiency. It may lead to lipodermatosclerosis and venous ulceration.

CAUSE
Venous insufficiency due to:

- Previous deep vein thrombosis.
- Valvular incompetence associated with varicose veins.

CLINICAL FEATURES

- Age – adults and elderly.
- Presence of varicose veins. Fig 1
- Itch.
- Eczematous rash (red, scaly ± weeping) on lower legs around the gaiter area. Fig 2
- Increased skin pigmentation (haemosiderin discolouration) around the ankles or foot. Fig 1
- Often associated with oedema.

Venous ulcers Fig 3 or scars (atrophie blanche) from healed ulcers

DIFFERENTIAL DIAGNOSIS

- Other types of eczema of the lower legs e.g. asteatotic, lichen simplex.
- Other causes of peripheral oedema.
- Cellulitis – often incorrect diagnosis; usually unilateral, painful, tender systemic symptoms.

INVESTIGATIONS

- Often none are necessary.
- Oedema – if marked, then investigate systemic causes.
- Doppler’s – to exclude arterial vascular disease and to assess for suitability of compression.

MANAGEMENT

- Emollients – ointments are better as greasier with fewer preservatives.
- Topical steroids - usually moderate /potent strength (betamethasone 0.1% or 0.025%) as ointments.
- Infection – treat streptococcal infection with oral antibiotics, other organisms may be colonizers.
- Combination steroid-antibiotic preparation e.g. Betnovate – C or Trimovate can be helpful in the short term.
- Be aware of increased risk of sensitization causing allergic contact dermatitis with prolonged use of topical antibiotics e.g. Fucidin. Fucibet.
- Doppler to assess ABPI.
Support stockings if eczema is mild and little/no oedema.
Bandaging if  a) leg ulceration.
    b) significant eczema.
    c) peripheral oedema.
Compression with 4 or reduced 4 layer bandage if ABPI normal (≥ 0.8).
Paste bandages (viscopaste, Zipzoc) are sometimes useful if eczema is very dry and irritated.
District nurses to apply and supervise bandaging.
Longterm – Class I/II support stockings indefinitely.

WHEN AND WHERE TO REFER

To Practice or District Nurse run Leg Ulcer clinics if associated ulcer.
Consultant Dermatologist if;
   a) Diagnosis in doubt.
   b) Failure to respond to routine management.
   c) patch testing if suspected secondary allergic contact dermatitis.

Fig Varicose veins with haemosiderin discoulouration

Fig 2 Eczematous rash around the gaiter area

Fig 3 Venous ulcer
Fig 4  Lipodermatosclerosis (inverted champagne legs)
FUNGAL SKIN INFECTION  
(Tinea Corporis, Tinea Pedis, Tinea Manuum, Tinea Capitis, Tinea Faciei, Tinea Versicolor, Tinea Incognito)

DEFINITION

Fungal infection of the skin in which growth is confined to keratinized tissue such as the epidermis, nail and hair.

CAUSE

Dermatophyte group of fungi includes the Trichophyton, Epidermophyton and Microsporum.

CLINICAL FEATURES

- Site - depending upon the site infected.
  Corporis  body  Fig 1
  Pedis   feet  Fig 2
  Manuum  hands  Fig 3
  Capitis  scalp Fig 4
  Faciei  face  Fig 5
  - Trunk, limbs, face - classically presents as annular scaly lesions, described as 'ring worm'.
  - Scalp - variable degree of hair loss, scaling and inflammation in the scalp or beard area.
  - Kerion – inflammatory boggy plaque with pustules on hair bearing area, usually from animal source. Fig 6
  - Feet - pruritic dermatitic reaction on the feet and in particular in the toe webs
  - Hands - unilateral scaling and erythema of the palm.
  - Incognito (loss of annular identity) due to inappropriate use of topical steroid.
  - Dystrophic nails (see Nails chapter).

DIFFERENTIAL DIAGNOSIS

Any annular rash such as:

- Granuloma annulare – not scaly, not itchy
- Nummular / discoid eczema – usually coin rather than ringed shaped, very itchy
- Psoriasis – can take an annular form
- Lichen planus

INVESTIGATIONS

- Wood's light examination showing fluorescence
- Mycology – skin scrapings, nail clippings, hair plucks – these should always be taken before commencing treatment for anything other than simple tinea pedis as antifungal agents are perhaps the agents used most inappropriately in clinical practice.

MANAGEMENT
- Topical anti-fungal - majority of skin infections will respond to topical agents. Topical treatment will need to be maintained for at least one month.
- Systemic anti-fungal (oral Terbinafine or itraconazole) – see BNF for duration of treatment.
- Scalp tinea – topical treatments do not work. Use oral Itraconazole or Terbinafine. These have largely replaced griseofulvin. Although Terbinafine is not licensed in children, it is considered the drug of choice for tinea capitis, to be used under specialist supervision (as per the BNF).

WHEN AND WHERE TO REFER

- Uncertain diagnosis
- Confirmation of diagnosis or where
- Equivocal mycology
- Immunocompromised patient
- Contemplating systemic treatment in a child
- Advice regarding the treatment schedule in extensive disease

Fig 1 Tinea corporis

Fig 2 Tinea pedis (athlete's foot with scaling in the web space)
Fig 2  (unilateral serpinginous rash on foot)

Fig 3  Tinea manuum (unilateral annular rash)

Fig 4  Tinea capitis (moth eaten appearance)

Fig 5  (obvious annular patches on the face)
Fig 5  Tinea faciei (mimicking eczema but note the serpiginous edge along the jawline)

Fig 6 Kerion in beard area
HIRSUTISM

DEFINITION

Hirsutism is the excessive growth of terminal hair in a male sexual growth pattern. Hypertrichosis is the excessive growth of hair that is not in a male sexual growth pattern.

CAUSE

Hirsutism in women is usually idiopathic in nature or related to end-organ hypersensitivity to androgens which is probably hereditarily determined. Other causes which may need to be ruled out include polycystic ovarian disease, adrenal hyperplasia, Cushing’s syndrome, adrenal tumours, ovarian tumours, pituitary tumours and hypothyroidism and these should always be considered.

Various drugs can also cause hirsutism (androgens, Danazol, Progesterone). Hypertrichosis is usually genetic or racial in origin. However some drugs can cause it; acetazolamide, corticosteroids, cyclosporin, diazoxide, interferon, minoxidil, phenytoin and psoralens.

CLINICAL FEATURES

- Hirsutism by definition is an excess of hair in a male pattern distribution and the commonest areas are therefore the face, neck, anterior chest, lower abdomen, tops of the feet etc.
- Patients with underlying hormonal problems often will have evidence of menstrual irregularities, male pattern alopecia and acne.
- Hypertrichosis usually presents with excess hair on normal sites, often fine vellus hair, occasionally long terminal hair in some congenital conditions.

DIFFERENTIAL DIAGNOSIS

1. Idiopathic hirsutism should always be differentiated from an underlying hormonal problem by careful history and examination and investigation as necessary.
2. As a general guideline patients without acne, male pattern hair loss or menstrual irregularities are unlikely to have an underlying hormonal problem and investigation in this group is usually not warranted although a serum testosterone may reassure the doctor and patient.

INVESTIGATIONS

Serum testosterone and sex hormone binding globulin is all that is required in most patients, if raised refer to Endocrinologist for further investigation.

MANAGEMENT
• Isolated hirsutism can be treated by physical methods which include shaving (there is no evidence to support the myth that shaving makes hair grow back thicker, darker and quicker), depilatory creams, bleaching, electrolysis or laser treatment.
• Hormonal manipulation can be undertaken (especially in those with polycystic ovary disease) and a contraceptive pill including Cyproterone (Dianette) with or without added Cyproterone is sometimes used.
• Spironolactone has anti androgen properties and is occasionally used.
• Vaniqa cream used in conjunction with other means of depilation may reduce the rate of regrowth. Treatments with these agents need to be long term and response is slow.

WHEN AND WHERE TO REFER

1. Endocrinologist if suspicion of underlying hormonal problem.
2. Beauty Salon if electrolysis is patient choice for treatment (not available under NHS locally).
3. Laser centre for laser hair removal if treatment of choice (not currently fundable on NHS locally, case may be taken to complex cases panel for funding if patient has biochemically proven PCOS).
LATEX ALLERGY

DEFINITION

A term used strictly to describe a type 1 immediate allergic reaction to the latex protein resulting in a contact urticarial reaction with swelling occurring within minutes of exposure. It can be associated with other immediate-type reactions e.g. wheezing and anaphylaxis.

CAUSE / SOURCE

Rubber /latex containing products e.g. rubber gloves (domestic and medical), balloons, condoms, catheters, other medical devices. Cross reactions with: Kiwi fruit, bananas.

CLINICAL FEATURES

- Itching, swelling and urticaria at sites of contact with latex product within minutes of exposure. Fig 1
- If severe, may develop wheezing, rhinitis, hypotension and anaphylaxis.
- Patients often have a history of multiple surgical procedures in the past.

DIFFERENTIAL DIAGNOSIS

Other allergen causing type 1 reaction e.g. peanuts

INVESTIGATIONS

- RAST latex.
- Skin prick test. Fig 2

MANAGEMENT

Refer to Dermatology for assessment, investigation and management advice.

WHEN AND WHERE TO REFER

As above.

‘Rubber’ allergy

Generally term used to describe a type IV delayed allergic contact reaction resulting in DERMATITIS. This is a result of an allergy to rubber additives (accelerators and vulcanisers e.g. thiuram, mercaptomix, carbamates) used in the processing of rubber. This is a common cause of dermatitis on the hands (e.g. from rubber gloves) and feet (from rubber soles).

Investigation - Patch test
Fig 1 Contact urticarial from rubber gloves

Fig 2 Positive skin prick test to latex
LEG ULCERS (VENOUS)

DEFINITION
An ulcer is an area of discontinuity of epidermis persisting for 4 or more weeks. When occurring on the lower leg, a venous ulcer is usually a result of venous hypertension due to varicose veins or as a calf muscle pump insufficiency.

CAUSE
There are multiple causes of leg ulceration and it must not be assumed that a leg ulcer is due to venous disease. Appropriate history taking, examination and investigation are mandatory. Exclusion of other causes is a prerequisite to appropriate management. Diagnosis is usually by clinical features, doppler measurements if venous or arterial; aetiologies are suspected and occasionally by biopsy.

- Venous ulcer occurs as of venous hypertension and consequent damage to the microvascular system in the lower leg.
- Trauma to lower limb - 50% will give history of a DVT, phlebitis, previous surgery or fracture to the leg or pulmonary embolus.
- Varicose veins – incompetent superficial or communicating veins of the leg.

OTHER CAUSES OF LEG ULCERS
- Vascular
  - Arterial – arteriosclerosis
  - Vasculitis – SLE, R.A., P.A.N.
    - Lymphatic
    - Capillaritis
- Neuropathic
- Diabetes
- Haematological
  - Polycythaemia rubra vera
  - Sickle cell anaemia
  - anti-phospholipid syndrome
- Traumatic
  - Burns, cold injury, pressure sores, radiation, factitious
- Neoplastic
  - BCC, SCC, MM, Bowen’s disease
- Other
  - Pyoderma gangrenosum,
  - Necrobiosis Lipoidica
  - Connective tissue disease e.g. Rheumatoid
  - Tropical ulcers
  - artefacta

CLINICAL FEATURES
- Site – gaiter area of the lower leg.Fig 1
- Varicose veins with venous flares will be present in the majority of cases.
- Eczema and haemosiderin pigmentation around the ulcer and extending up the leg.
- Lipodermatosclerosis- hardening of skin secondary to fibrosis giving the ‘inverted champagne bottle’ appearance’, erythema and inflammation. Fig 2
- Atrofie blanche – white indented scarring. Fig 3
- Palpable foot pulses.

Concomitant arterial disease must be excluded as must diabetes, inflammatory arterial disease, connective tissue disease, rheumatoid disease and malignancy.

**DIFFERENTIAL DIAGNOSIS**

- Arterial ulcers.
- Mixed arterial / venous ulcers.
- Malignant ulcers.
- See above.

**INVESTIGATIONS**

- Doppler assessment of the foot must be done and ankle brachial pressure index (ABPI) calculated before compression therapy. Fig 4
- Swabs
  a) Leg ulcers are going to be colonised by skin and bowel flora.
  b) Antibiotic treatment is not required unless clinical signs of infection (pyrexia, pain, spreading erythema.
  c) β-haemolytic streptococcus infection (often causes cellulitis) is probably an exception and should always be treated, usually with systemic antibiotics according to sensitivities. Likewise staphylococcal infection often causes pain and should usually be treated systemically according to sensitivities.
- Full blood count, biochemical profile and fasting glucose, etc when relevant to exclude others causes

**MANAGEMENT**

Initially by District Nurses

- Elevation of legs
- Emollient ± topical steroid ointment
- Compression bandaging - an ABPI of 0.8 or greater

Compression bandages must be properly applied to produce active counter pressure, with graduated compression, maximal at the ankle to enhance venous return.
NB do not compress without prior Doppler to exclude arterial disease. Four layer compression should be the ultimate aim. After the ulcer has healed, compression hosiery should be considered to prevent recurrence.

**Support stockings**
Below knee is often all that is realistic
Grades are I – III depending on compression (I=low, III=high)
They can be difficult to put on and often require application aids

- Mobilization
- Weight loss and good nutrition

**PRACTICAL POINTS**

- If there is obvious oedema of the leg, the ulcer is unlikely to heal.
- A weeping ulcer requires elevation and Potassium Permanganate soaks.
- Crepe bandages and Tubigip™ do not apply adequate support or pressure.
- Slough on an ulcer should be removed by forceps and scissors. It is not necessarily a sign of infection but will delay healing.
- Contact allergic dermatitis is a common complication and may be caused by rubber, lanolin, preservatives and topical antibiotics.
- No dressings of any type have been proven to enhance ulcer healing rates. There is evidence from clinical trials that occlusive dressings may relieve pain. Gels and hydrocolloid dressings absorb exudates. See Wirral dressings formulary for ‘dressings' which may be used in specific clinical scenarios usually after assessment by Tissue Viability nurses.
- It is useful to monitor healing with measurements and photographs.

**WHEN AND WHERE TO REFER**

**Dermatology referral**

- Ulcers where an inflammatory aetiology is considered (e.g., pyoderma gangrenosum).
- Ulcers complicated by severe eczema
- If contact dermatitis is suspected
- If malignancy is suspected.

**Surgical referral (vascular)**

- Ulcers with an ABPI of less than 0.7 should be referred to a vascular surgeon
- Consider operative intervention for varicose veins once leg ulcers are healed.
- Presumed venous ulcers not healing after 12 weeks compression should be referred for a vascular opinion.
- Ulcers in diabetic patients should receive a vascular opinion in the first instance.
Fig 1 Gaiter area

Fig 2 Lipodermatosclerosis with inverted champagne legs

Fig 3 Atrophie blanche (scarring)

Fig 4 Doppler ABPI measurement
LICHEN PLANUS

DEFINITION

A relatively common condition characterised by multiple usually itchy flat topped papules on the skin and a white lacy pattern on the mucosal surfaces. Genital and perianal mucosa may also be involved.

CAUSE

- Largely unknown
- Viral
- Immunologically mediated - similarity with the pathology of graft verses host disease raises the possibility of a lymphocytic reaction against the keratinocyte.

CLINICAL FEATURES

- Itchy eruption
- Duration – weeks to months. Some may last years.
- Morphology: Violaceous polygonal flat-topped papules. Fig 1 Wickham’s striae (white lacework pattern).Fig 2
- Sites: Any skin but predilection for - Flexor aspects of wrists. Fig 1 - Lumbar region.
- Ankle.
- Other sites: - Scalp (lichen planopilaris, frontal fibrosing alopecia; see alopecia chapter).
- Nails.
- Mucous membranes (oral buccal and/or anogenital) – Wickham’s striae, erosive lichen planus.
- Koebner’s phenomenon Fig 3– linear papules within injury sites e.g. scratches, scars.
- Post inflammatory hyperpigmentation Fig 4– very common, especially in dark skinned patients when it can be intense and prolonged. Topical steroids do not help this phase.
- Variants:
  Hypertrophic – persistent, thickened, lower legs. Fig 5
  Annular - multiple small annular lesions, penile involvement.
  Linear – Koebner.
  Bullous.
  Erosive (mucosal gingivitis, vaginitis) – risk of SCC in longstanding ulceration.
  Lichen planopilaris, follicular (see Hair chapter).

DIFFERENTIAL DIAGNOSIS

Lichenoid reactions induced by drugs.

INVESTIGATIONS

- Usually none is needed.
- Skin biopsy if in doubt.
Liver function - association with primary biliary cirrhosis or hepatitis.

**MANAGEMENT**

Aim of treatment is to control the symptom of itch. The natural progression and prognosis of LP is not altered by treatment.

- Steroid
  2. Systemic (oral Prednisolone) in severe, extensive and rapidly evolving cases (requires several weeks treatment).
  3. Intralesional triamcinolone – for localized hypertrophic lesions.
- Antihistamines (sedative) will help to reduce the itch and improve sleep.
- Second line agents (for secondary care consideration)
  e.g. PUVA, Dapsone, Azathioprine, oral retioinds, Cyclophosphamide, Hydroxychloroquine or Cyclosporin

**WHEN AND WHERE TO REFER**

- If diagnosis in doubt
- Not responding to simple treatments
- For consideration of second line agents
- Hair involvement resulting in scarring alopecia
- Nail involvement causing nail destruction
- Mucous membrane involvement

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Fig 1 Violaceous polygonal papules on flexor wrist

![Fig 1 Violaceous polygonal papules on flexor wrist](image1)

Fig 2 Wickham’s striae (white lacy pattern)

![Fig 2 Wickham’s striae (white lacy pattern)](image2)
Fig 3  Koebner phenomenon (linear lesions in a scratch)

Fig 4  Post inflammatory hyperpigmentation especially in dark skin

Fig 5  Hypertrophic lichen planus
LOCAL HYPERhidrosis (EXCESSIVE SWEATING)

DEFINITION
Excessive local sweating usually of the axillae, palms or soles that leads to embarrassment.

CAUSE
- Idiopathic, but organic disease (especially thyrotoxicosis, Hodgkin's Disease, acromegaly and tuberculosis) should be considered.
- Anxiety often coexists leading to a vicious cycle.

CLINICAL FEATURES (Figs 1,2 &3)
All too obvious leading to problems with clothing, footwear, manual tasks and interpersonal relationships. Scores highly on reduction in quality of life assessments. If feet colonised with corynebacterium may lead to malodour and characteristic appearance (pitted keratolysis)

DIFFERENTIAL DIAGNOSIS.
Exclude organic cause, as above, if clinical suspicion.

INVESTIGATIONS
Nil required.

MANAGEMENT
- Topical therapy: Aluminium chloride hexahydrate (Drichlor or Anhydrol Forte) initially once daily at night then reduced in frequency according to response. Apply to dry skin (not recently shaved; stinging). May cause irritant dermatitis (especially in axillae – combine with mild topical steroid). Advise patients to build up duration of application, not to apply the treatment for a whole night initially, build up to this.
- Oral anticholinergic drugs. Oxybutinin 2.5mg three times a day, increasing according to response to a maximum of 7.5 mg three time a day. Dry mouth and blurred vision possible side effects.
- Iontophoresis: A low dose electric current is passed through the skin two or three times a week. Used for hand and foot hyperhidrosis. This is now available in the Dermatology Unit at Clatterbridge Hospital.
- Botulinum toxin - only for specialist units to perform. Licensed for Axillary hyperhidrosis only.
- Surgery: Cervical (for palmar) sympathectomy may be tried if the above measures are ineffective.

WHEN AND WHERE TO REFER
- Topical measures can be prescribed on FP10.
• The dermatology unit performs iontophoresis for hands and feet (if successful patient purchases machine) and Botox injections for axillae (maximum of one treatment per year).

Fig 1 Hyperhidrosis - Axillae

Fig 2 Hyperhidrosis - Hands

Fig 3 Hyperhidrosis – Feet
MALIGNANT MELANOMA

DEFINITION

Malignant melanoma is a potentially aggressive form of skin cancer arising from the pigment producing cells in the skin (melanocytes). Though still relatively uncommon with an annual incidence of approximately 1 per 15,000 of the population the incidence of this cancer continues to rise.

CAUSE

- Ultraviolet exposure - Intermittent exposure e.g. recreation exposure and childhood sunburns correlate closely with the tendency to develop melanoma.
- Increasing evidence of link with sunbed use.
- Risk factors: Fair skin, freckles
  Multiple naevi
  Family history of melanoma
  History of episodes of sunburn

CLINICAL FEATURES

There are six clinical types of melanoma:

- In situ melanoma or lentigo maligna Fig 1
- Superficial spreading malignant melanoma Fig 2
- Nodular melanoma Fig 3
- Lentigo maligna melanoma
- Acral lentiginous Fig 4 and subungual melanoma Fig 5
- Unclassifiable melanoma

Early detection and treatment will influence the Breslow thickness and hence prognosis.

The prime criteria for diagnosis are:

<table>
<thead>
<tr>
<th>MAJOR</th>
<th>MINOR</th>
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<tbody>
<tr>
<td>Increase in size</td>
<td>Itching</td>
</tr>
<tr>
<td>Irregular edge and shape</td>
<td>Inflammation</td>
</tr>
<tr>
<td>Irregular pigmentation</td>
<td>Crusting &amp; bleeding</td>
</tr>
<tr>
<td>Diameter &gt; 7 mm</td>
<td>Diameter &gt; 7 mm</td>
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</tbody>
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The ABCD(E) algorithm

Asymmetry - in shape
Border - irregular
Colour - mixed, red, brown, black, blue, flesh coloured
Diameter – over 6mm
Enlargement – history of growth

If a pigmented lesion has 2 or more of the above features, refer for specialist opinion.
DIFFERENTIAL DIAGNOSIS

Compound naevus, basal cell papilloma (seborrhoeic wart), haemangioma, angio fibroma and dermatofibroma.

INVESTIGATIONS

Dermatoscope in skilled hands.

MANAGEMENT

- Refer ALL suspicious moles to the Skin Cancer Unit for assessment.
- Biopsy – NOT recommended for suspicious moles in the GP surgery.
- Surgical excision – full, with margin initially of at least 2mm is the treatment of choice.

NICE and Skin cancer IOG recommend that ALL suspected melanomas should be referred on a 2 week cancer referral pathway to a member of the Local Skin Cancer MDT.

WHEN AND WHERE TO REFER

- Refer via 2 week skin cancer pathway using electronic referral proforma.
- Diagnosis.
- Treatment- full excision on the day of first consultation, wide local excision at a later date depending on histological diagnosis and depth of excision.
- Discussion at skin MDT.
- Referral to Specialist Skin MDT (SSMDT) and sentinel lymph nodes biopsy if appropriate. Current guideline is for malignant melanoma with Breslow thickness of 1mm or more
- Follow up as appropriate.
Fig 1 Lentigo maligna

Fig 2 Superficial spreading Malignant melanoma

Fig 3 Nodular melanoma
Fig 4 Lentigo maligna melanoma

Fig 4 Acral lentiginous melanoma

Fig 5 Subungual melanoma (note Hutchinson sign)
MOLLUSCUM CONTAGIOSUM

DEFINITION

A common virus infection usually affecting children.

CAUSE

This is due to infection with one of the pox viruses; the molluscum contagiosum virus. Infection is more common and more widespread in atopic individuals.

CLINICAL FEATURES

- The individual lesion is a small smooth papule with a central umbilicated centre. Gentle expression of the lesion will result in a plume of “cream cheese” like material from the umbilicated centre.
- Lesions are often multiple due to auto inoculation and groups of lesions may well be complicated by surrounding eczema which is usually responsible for any itching, the lesions themselves usually being asymptomatic.
- Occasionally lesions may become secondarily infected or traumatised by scratching.

DIFFERENTIAL DIAGNOSIS

1. Viral warts
2. Folliculitis

INVESTIGATIONS

None required.

MANAGEMENT

- Education of the patient and especially the parents is often all that is required, reinforcing that the lesions will spontaneously remit in usually 6 to 9 months. This will imply that immunity has developed and that hopefully therefore new lesions will not develop. (Emphasise this as an advantage of not treating!)
- Occasionally Bactroban or Crystacide cream may be helpful for infected lesions.
- Specific treatment requires "traumatising" lesions in someway either by gentle expression, freezing with cryotherapy or curettage. All of these can be traumatic to the patient and the treatment of choice therefore is "masterly inattention".
- Small 'pock mark' scars may follow the healing of infected lesions.

WHEN AND WHERE TO REFER

- Molluscum does not usually warrant hospital referral.
NAIL DISORDERS, (onychodystrophies and onychomycosis)

DEFINITION

Onychomycosis. Infection of the finger and/or toe nails with either dermatophyte fungus, Candida species or moulds (common reason why ‘fungal nails’ fail to respond to treatment.. Nail bed may be infected by bacteria usually Pseudomonas or viral, either herpes simplex or the Orf virus.

Onychodystrophy. Abnormality of the nail plate due to an underlying skin, general medical problem or trauma.

CAUSE

- May be part of a generalised infection particularly if dermatophyte fungus or in the immunocompromised patient particularly, AIDS. Candida and Pseudomonas infection is common in those who do a lot of wet work e.g. bar workers.
- Herpes virus infection of the finger nail area used to be occupational hazard of health workers prior to universal wearing of latex gloves.
- Orf is seen in farm workers particularly in the lambing season.
- Onychodystrophy can be caused by a multitude of conditions; skin diseases such as psoriasis, eczema, lichen planus, alopecia areata, Darier’s disease, congenital abnormalities, habit picking, over zealous use of nail varnish remover and allergic contact dermatitis reaction to false nails.
- Drugs, hydroxycarbamide can cause a nail discolouration. NSAIDs have been reported causing photo-onycholysis

CLINICAL FEATURES

- The nails are usually dystrophic, thickened, may be separating from the nail bed and they may be discoloured.
- The classical feature of fungal nail infection is a yellowish white discolouration of the nail (mould infection can mimic this).
- Superficial white onychomycosis present as small white/yellow patches on the nails.
- A Candida infection causes pain and swelling of the finger pulp and a whitish discharge from the lateral nail fold.
- A Pseudomonas bacterial infection will often present as a very dark green or black discolouration under the nail.
- Herpes simplex infection is painful with a red swollen nail plate sometimes with blistering.
- Orf infection presents as a single painful nodule on or around the nail apparatus.
- Psoriatic nails show distal onycholysis, salmon patch, oil spot sign and pitting.
- Eczematous nails show general nail dystrophy and onycholysis.
- Lichen planus of the nails shows thickening and longitudinal ridges and nail pterygium.
- Alopecia areata has small ‘pepper pot’ pitting.
- There are many well recognised nail changes associated with underlying medical problems e.g. with renal failure, liver failure, chest disease, thyroid disease, lupus etc.
DIFFERENTIAL DIAGNOSIS

1. Many of the chronic papulo-squamous skin disorders have nail manifestations, chronic plaque psoriasis, chronic eczema, lichen planus all have nail dystrophies.
2. Over zealous use of nail varnish and nail varnish remover can also lead to a nail dystrophy, particularly onycholysis.
3. Chronic picking of the nail fold causes a persistent nail dystrophy – Christmas tree sign.

INVESTIGATIONS

Nail clippings must **ALWAYS** be taken and sent for mycological examination before treatment is commenced. Distal nail clippings are not adequate; the more proximal that nail clippings can be taken, the more likely one is to get a positive and relevant mycological diagnosis.

MANAGEMENT

- Mycologically proven dermatophyte nail infection requires oral anti-fungal treatment with either oral Terbinafine taken for 2 to 3 months for finger nail infection and up to 4 to 6 months for toe nail infection. Alternatively Itraconazole either 200 mg for 3 months or pulse treatment 200mg twice daily for 7 days repeated after a 21 day interval, 2 cycles for finger nails, 3 cycles for toe nails.
- Superficial white onychomycosis may respond to topical Amorolfine
- Candida nail infection will require oral Itraconazole.
- Infection with moulds (Scopulariopsis brevicaulis or Scytalidium dimidiatum) is very frustrating, may respond to prolonged application of Amorolfine.
- Treatment for bacterial infection will depend on the bacteria cultures. Psudomonas infection of the nail bed can be treated with Ofloxacin eye drops.
- Herpes simplex will require oral Aciclovir.
- Orf is a self limiting disorder, however topical antiseptics are useful to prevent secondary infection.
- Oral antifungal agents should only ever be prescribed when one has positive mycology and not prescribed empirically. N.b. Terbinafine will exacerbate/precipitate psoriasis in a susceptible patient
- Treatment of nail dystrophies secondary to generalised skin or systemic disease will depend on the causative illness.

WHEN AND WHERE TO REFER

Nail problems often difficult to diagnose and manage, so may require referral for diagnosis and advice for treatment in difficult cases.

Advice on management of onychomycosis in children

On occasions a nail biopsy will be necessary to obtain a definite diagnosis.
NON MELANOMA SKIN CANCERS
(Basal cell carcinoma (BCC) and Squamous cell carcinoma (SCC))

DEFINITION
Malignant tumours of the epidermis. Basal cell carcinoma believed to be a tumour of a pluripotential cell of the infundibulum of the pilosebaceous unit. Squamous cell carcinoma the malignant tumour of the keratinocyte.

CAUSE
Ultraviolet exposure - probably long term exposure rather than short term intermittent. BCCs can occur de novo in the skin.

SCCs invariably arise on pre-existing actinically-induced lesions such as an actinic keratosis or Bowen's disease. SCCs may also be induced by other means than ultraviolet light; chronic exposure to oils, workers in the rubber industry or on a site of pre-existing trauma e.g. ionizing radiation and chemical or physical trauma such as burns and chronic wounds.

DIFFERENTIAL DIAGNOSIS
- Benign lesions such as sebaceous hyperplasia, intradermal naevus.
- Pre-malignant lesions e.g. actinic keratosis, Bowen’s.
- Malignant lesions e.g. lymphoma, sarcoma, keratoacanthoma, amelanotic melanoma.

BASAL CELL CARCINOMA (Rodent Ulcer)

CLINICAL FEATURES
- Nodulocystic type can look like pale, protuberant moles on the face. Telangiectasia and translucency may give more of a clue. Fig 1
- Beware odd scars on the face with no history of trauma – these may be infiltrating morphoeic BCCs. Fig 2
- BCCs tend to bleed easily. They can ulcerate and then appear to heal but there will always be some residual lesion between ‘flares’.
- Some look like the red scaly patches of eczema or psoriasis but are always well defined, usually solitary and sometimes have a very fine thread like raised edge – superficial BCC. Fig 3
- Pigmentation may be present in a BCC, making differentiation from a melanoma difficult. Fig 4
- Common sites include – head and neck, back and legs.

NICE and Skin cancer IOG recommends that all but small low risk BCCs (<1cm on trunk and limbs) should be referred to secondary care for treatment.
MANAGEMENT

Depends on the site and type of lesion, age and comorbidities of the patients. Also on patient’s choice and sometimes previous experience.

- Excision (including Mohs micrographic surgery in rare cases)
- Radiotherapy
- Curettage and cautery
- For superficial BCCs;
  - Cryotherapy
  - Imiquimod or Efudix cream
  - Photodynamic therapy
- MDT discussion when appropriate

WHEN AND WHERE TO REFER

- Diagnosis
- Discussion whether medical treatments appropriate in clinical setting.
- Selection of treatment.
- Discussion of treatment options with Multidisciplinary Team (MDT) for skin cancer.
- Once patient has had a BCC they have a 30-50% of having a further BC in the following 5 years.
- NB: BCCs are not included in the 2 week wait referral for skin cancer.

SQUAMOUS CELL CARCINOMA (SCC)

CLINICAL FEATURES

- Papule or nodule which may be scaly with central ulceration to give a crateriform appearance. Fig 5
- Cutaneous horn usually with erythematous indurated base. Fig 6
- Arising from pre-existing actinically damaged skin.
- Slow growing (well differentiated) to rapidly growing (poorly differentiated).
- Bleeds easily.
- Can be painful and tender.
- Occurs mainly in the over 70’s on sun exposed skin.
- 5% may metastasise to regional lymph nodes.

NICE and Skin cancer IOG recommend that ALL suspected SCCs should be referred on a 2 week cancer referral pathway.

MANAGEMENT

- Surgical excision
- Radiotherapy

WHEN AND WHERE TO REFER

- Refer via 2 week skin cancer pathway using electronic referral proforma
- Diagnosis
• Selection of treatment: surgery or radiotherapy
• Discussion of treatment options at MDT for skin cancer
• Follow up as appropriate

Fig 1 Nodular BCC (pearly nodule)

Fig 2 Morphoeic BCC

Fig 3 Superficial BCC (note the pearly edge) at 12 o’clock margin

Fig 4 Pigmented BCC
Fig 5  Crateriform SCC

Fig 6  SCC – cutaneous horn on an indurated base
PATCH TESTING

DEFINITION

A form of skin testing to investigate allergic contact dermatitis and to identify allergens which cause Type IV delayed allergic reactions.

CLINICAL FEATURES

Indications of patch testing (P/T)

1. Eczemas where contact allergy is suspected or is to be excluded.
2. Eczemas failing to respond to treatment as expected.
3. Chronic hand and foot eczema.
4. Persistent eczema at specific sites e.g. Face, eyelids, ears and perineum.
5. Varicose eczema which is resistant to conventional treatment and medicaments.

METHOD

Patch testing involves three visits to the Dermatology department:

- Monday (D1) - patches are applied to the patient’s back and these are left in situ for 48 hours.
- Wednesday - patches are removed, the first readings are taken and the back is marked.
- Friday (D4) - a dermatologist reads and interprets any reactions elicited by the patches. A positive reaction is shown as a small patch of eczema corresponding to a specific allergen. The patient is counselled on allergen avoidance and information leaflets given.

Patch test batteries

In addition to the Standard battery, we have a large number of patch test batteries which can be tailored to the patient’s clinical history e.g. Hairdressing, cosmetic, dental, acrylates, flavourings, plants, leg ulcer, etc.

Occupational (industrial) dermatitis – we can patch test patients to materials from the workplace. These have to be delivered to the Dermatology department 1-2 weeks prior to patient’s appointments. A dermatologist examines these together with the COSHH to assess the suitability for patch testing. The materials are then diluted accordingly.

Patch testing is labour and cost intensive on nurse, doctor and patient time. Dilution of materials and additional allergen batteries have cost implications. It is also uncomfortable for the patients.

The greatest expertise is required for the reading and especially interpreting the relevance (or otherwise) of reactions and putting them into context for the patient.
WHEN AND WHERE TO REFER

Any patient with suspected contact dermatitis (see above) should be referred to routine dermatology clinic for initial assessment for the need for patch testing as well as the batteries to be tested.

Patch testing is NOT used to investigate type 1 reactions e.g. Rhinitis, asthma, anaphylaxis, and food ‘allergy’ or intolerance which should be referred to the allergy services. Allergy tests are also or little or no use in urticaria.
PHOTOSENSITIVE RASHES

DEFINITION

A group of skin conditions associated with an abnormal response to ultraviolet (UV) radiation.

Photodermatoses can be divided into:
1. Photosensitive (allergic).
2. Photoaggravated.

UVB causes sunburn. UVA which penetrates glass is responsible for most of the photodermatoses.

CAUSE

Photosensitive dermatoses
These are immunologically mediated.
Idiopathic: Polymorphic light eruption (PLE) and Juvenile spring eruption.
Actinic prurigo.
Chronic actinic dermatitis (CAD).
Solar urticaria.
Hydroa vaccineforme.

Metabolic: Porphyria.

Exogenous: Drug-induced.
Photocontact.
Phytophotodermatitis.

Genetic: Xeroderma pigmentosum.

Photo-aggravated dermatoses
These are pre-existing skin conditions that can be worsened with UV light. Examples are lupus erythematosus (LE), dermatomyositis, herpes simplex, Darier’s disease, pellagra, rosacea.

CLINICAL FEATURES

- **Age** – genodermatoses such as xeroderma pigmentosum and erythropoietic porphyria present in infancy and early childhood. Juvenile spring eruption occurs in young boys.

- **Gender** – PLE and lupus erythematosus are more common in young women. Chronic actinic dermatitis is more common in older men.

- **Family history** - important for the porphyrias, genodermatoses.

- **Timing** in relation to **sun exposure** - solar urticaria (type 1) occurs within minutes, PLE many hours. Chronic actinic dermatitis is a type IV reaction and sufferers may not be aware that their symptoms are worse in the sun because of slow and delayed onset. Symptoms made worse after application of sunscreen suggest a photocontact allergic dermatitis to the sunscreen. Window glass - most glass only blocks UVB. Some special laminated windows (e.g. car windscreens) also protect against UVA.
- **Timing** of the eruption in relation to season - PLE is typically prominent in the spring and improves later in the summer. Chronic actinic dermatitis and phytodermatitis are more prevalent during the summer months.

- **Distribution** of the rash – occurs on sun exposed areas of the skin, mainly on the head and V of neck and forearms. On the face there is sparing of the ‘shadowed’ areas (submental, below nose, behind ears, underneath a fringe). In chronic cases, this pattern is sometimes blurred. Dorsa of hands are less commonly involved because of ‘hardening / weathering’ effected. N.b. light can pass through thin clothing.

- **Symptoms** – depends on the rash. Itch is common in actinic dermatitis, actinic prurigo, solar urticaria and PLE. Burning pain is typical for erythropoietic protoporphyria

- **Drug history** – consider ALL drugs including OTC, systemic prescribed and topical medication. Drug causing photoallergic reactions include quinine, thiazides, NSAID. Other drugs can cause a phototoxic (non-allergic) reaction e.g. doxycycline, amiodarone.

**DIFFERENTIAL DIAGNOSIS**

- To distinguish between photo allergic and photoaggravated dermatoses.
- Airborne dermatitis – dermatitis is distributed to exposed skin with no sparing of the ‘shadowed’ areas.
- Contact dermatitis to e.g. sunscreens, psoralen, fragrance.

**INVESTIGATIONS**

- Careful history.
- Autoimmune screen (ANA, ENA) to exclude lupus.
- Porphyria screen.
- Patch test / photopatch test.
- Photo testing - only in specialised centres (Manchester).

**MANAGEMENT**

- Remove culprit photosensitive (if present).
- Sun avoidance (seeking shade, avoid mid day sun).
- Sun protection (high SPF 50+ and 5* UVA, clothing, hat).
- Drugs - depending on cause e.g. anti-malarials for lupus.

**WHEN AND WHERE TO REFER**

- Patient not responding to simple sun protection measures.
- Uncertain diagnosis.
- Investigations.

**Polymorphic light eruption (PLE)  Fig 1**

- More common young women.
- Caused by UVA, therefore sunbeds can trigger, high SPF UV block only moderately helpful. Need to use maximum 4* or 5* rating sunscreens
• Occurs in early spring.
• Face is usually spared because of ‘hardening / weathering’ effect.
• Exclude Lupus.
• Management:  1. Prevention - Sun avoidance. 
   Desensitisation (UVB).
   2. Treatment - Topical steroids ± anti-histamines.

Juvenile spring eruption – condition of young boys. Fig 2

Actinic prurigo  Fig 3
• Occur in all ages (one third in children).
• More common in darker skin patients.
• Intensely itchy papules in sun exposed areas e.g. face, neck, upper limbs.
• UVA and UVB involved.
• Treatment: Sun avoidance and protection.
  Topical steroids.
  Anti-malarials.

Chronic actinic dermatitis (CAD)  Fig 4
• Affects men usually over 50 years.
• Chronic lichenified dermatitis on sun exposed skin of face, V of neck, upper arms and hands.
• Very UV sensitive, even through clothing, glass, on dull days and in the winter.
• Patch test to exclude photo contact e.g. chrysanthemum and fragrance.
• Treatment: Sun avoidance.
  Topical steroids
  .azathioprine, ciclosporin.

Solar urticaria  Fig 5
• Rare.
• Immediate reaction (within 30 minutes) - stinging, urticaria, swelling of sun exposed areas.
• Short lasting (min - hour).
• Treatment: Sun avoidance.
  Anti-histamines.
  Desensitisation.

Porphyria
• Porphyria cutanea tarda (PCT).
• Skin fragility and blisters on back of hands and arms, milia, Fig 6, abnormal LFT, drugs, alcohol, post menopausal women, particularly on HRT.
• Erythropoietic porphyria (EPP).
• Painful burning sensation of sun exposed skin early in infancy.
Fig 1 Polymorphic light eruption

Fig 2 Juvenile eruption

Fig 3 Actinic prurigo

Fig 4 Chronic actinic dermatitis
Fig 5 Solar urticaria

Fig 5 Porphyria cutanea tarda
PITYRIASIS ROSEA

DEFINITION

A benign self limiting condition with a characteristic eruption.

CAUSE

Probably unknown but circumstantial and also laboratory evidence pointing towards a viral aetiology. HHV 6 & 7 have been implicated but the condition does not appear to be contagious.

CLINICAL FEATURES

Patients may occasionally report mild prodromal symptoms such as mild upper respiratory tract symptoms. The condition may occur in clusters and is common in the winter months.

A herald patch (a large 2 cm to 5 cm reddish/brown patch usually occurring on the trunk, exhibiting a sharply demarcated scaly border) precedes the main rash by 2 to 3 weeks. The secondary eruption consists of crops of oval to round erythematous patches which have a peripheral collarette of scale which classically lie along the lines of the ribs and giving the classic "Christmas tree" pattern. The face, the forearms and legs maybe affected, particularly in children. In 10% to 20% of cases oral lesions can occur which may consist of ulcers, erosions or plaques in the mouth. The rash is usually itchy and self limiting. The herald patch usually lasting for 3 to 4 weeks and the secondary eruption for a maximum of 3 months.

DIFFERENTIAL DIAGNOSIS

1. The eruption maybe mistaken for a fungal infection (scaly edge)
2. Pityriasis versicolor
3. Acute psoriasis
4. A drug reaction
5. Classically secondary syphilis (often involves the palms)

INVESTIGATIONS

None needed.

MANAGEMENT

No treatment will fundamentally alter the course or duration of this disease but symptoms may be helped by emollients, antipruritics, mild topical steroids or, if the rash is very florid phototherapy. The condition usually confers lifelong immunity.

WHEN AND WHERE TO REFER

- The condition is usually easy to diagnose on history and examination.
- Atypical forms do exist and these may require referral.
PITYRIASIS VERSICOLOR

DEFINITION

A yeast infection of the skin leading to areas of scaly, altered pigment of the skin.

CAUSE

A group of lipophilic yeasts of the malassezia family (Pityrosporum ovale). They form part of the normal flora of the skin.

CLINICAL FEATURES

- Skin condition of young adults.
- Insidious onset.
- Virtually asymptomatic, occasionally irritable after sweating.
- Distribution: upper trunk, shoulders, arms, neck.
- Widespread macules and patches which may be hypopigmented, light brown / fawn coloured or pink. Fig 1. May coalesce to form a large area of altered pigmentation. Tend to look darker on light skin Fig 2 and paler on dark skin. Fig 3
- More noticeable after sun exposure (macules do not tan) e.g. after a sunny holiday or sunbed use.
- Light surface bran-like scale.

DIFFERENTIAL DIAGNOSIS

Any condition with pigmentary disturbance.
- Vitiligo – complete loss of pigmentation with depigmentation rather than hypopigmentation
- Pityriasis rosea – truncal scaly rash, reactive viral aetiology
- Tinea corporis / incognito

INVESTIGATIONS

- Skin scrapings examined with a Parker’s ink / KOH solution will show hyphae and yeasts cells (‘spaghetti and meatballs’) appearance.
- Mycology is rarely successful in culturing the yeast.

MANAGEMENT

Topical treatment
- Ketoconazole shampoo used 2 to 3 times a week as a body wash leaving on for 10 minutes for 2-3 weeks can be effective.
- Selenium sulphide (Selsun) painted to the affected areas and left on overnight then washed off. It can be very irritating and has a very pungent odour and compliance can be poor.
- Topical Imidazole creams such as Miconazole or Econazole are effective when only a small area is involved.

Systemic treatment - more widespread or recurrent cases
• Oral Itraconazole 200 mg daily for 14 days is effective. Oral Terbinafine does NOT work.
• Pigmentary disturbance will last for a considerable time and is not a sign of failed treatment.
• Recurrence is very common.

WHEN AND WHERE TO REFER

• Uncertain diagnosis

Fig 1 Pink macules of P versicolour

Fig 2 Hyperpigmented patches

Fig 3 Hypopigmented patches
PRURITUS

DEFINITION

Generalised itching.

CAUSE

- Local causes. Scabies, eczema, lichen planus, dermatitis herpetiformis, mycosis fungoides, urticarial dermographism, pre-bullous eruptions.
- Systemic causes: Iron deficiency, liver disease, chronic renal failure, underlying malignancy e.g. lymphoma, polycythaemia, pregnancy, thyroid disease, old age, "psychogenic".
- Aquagenic pruritus is a rare phenomenon where the patient gets an intense itch when in contact with water.

CLINICAL FEATURES

- Itching of the skin which can be persistent and extremely unpleasant.
- There may be no local rash in which case think of the systemic causes.
- It is worth looking carefully for scabies burrows.
- Eczema (especially atopic eczema) may be relatively mild in appearance yet cause significant itching.

DIFFERENTIAL DIAGNOSIS

As above, see causes.

INVESTIGATIONS

Take a full history and perform a full physical examination. If no obvious local cause check; full blood count, ESR or plasma viscosity, ferritin (even if full blood count normal) U&Es, liver function tests, TFTs, chest x-ray and abdominal ultrasound.

MANAGEMENT

- Treat any local causes as detailed in the appropriate guidelines.
- Generalised pruritus with no obvious underlying cause responds poorly to antihistamines although sedating antihistamines may help in some cases, especially when worst at night.
- Non sedating antihistamines have no benefit in non specific pruritus.
- It is important to keep the skin well hydrated with emollients and use a soap substitute as detailed in the Wirral formulary.
- Low dose tricyclic antidepressants as for chronic pain can sometimes be helpful.

WHEN AND WHERE TO REFER

1. If there is a rash and the diagnosis is in doubt.
2. In the absence of a rash, refer to the appropriate specialists if investigations reveal any underlying cause.
3. In the absence of an obvious cause if there is no response to the above management suggestions then refer to any Consultant Dermatologist.
PRURITUS ANI

DEFINITION

Intense itching localised to the perianal skin. Maybe persistent or intermittent.

CAUSE

Multifactorial, having excluded surgical causes, haemorrhoids, fissures, skin tags, mucous secreting tumours, proctitis, anal carcinoma etc. then dermatological conditions have to be considered;

1. The perianal manifestation of a generalised skin disease, e.g. psoriasis, seborrhoeic dermatitis, lichen planus etc.
2. Skin disease localised to the perianal skin e.g. lichen sclerosus, lichen simplex chronicus, hidradenitis suppurativa.
3. Contact dermatitis, (a) allergic, due to contact allergy to components of over the counter and prescribed topical preparations, NB: topical anaesthetics, balsam of Peru, ingredients in wet wipes are potent sensitisers. (b) irritant, due to prolonged contact with faeces, particularly elderly or others with faecal incontinence.
4. Local infections; (a) bacterial, perianal streptococcal dermatitis, syphilis, gonorrhoea. (b) viral, warts, molluscum contagiosum, herpes. (c) yeast, candida, probably as a secondary contaminant. (d) dermatophyte fungi – very unusual to get perianal ‘ringworm’. (e) parasites, threadworms.
5. Pre malignant and malignant conditions; AIN, Bowenoid papulosis, extra mammary Pagets.
6. Poor perianal hygiene, too much washing ‘anal polisher’, too little washing, elderly, obese, arthritic.
7. Faecal leakage, often no primary cause found for pruritus but patient suffers from a small amount of faecal leakage onto the perianal skin. Faces very irritant, leakage may be a consequence of; surgery, pregnancy, obesity, excess flatus, alcohol, smoking, spicy foods and drugs.
8. Rare causes, Nicorandil ulcer, acrodermatitis enteropathica, ankylostoma infestation, Behcets, dermatitis herpetiformis.

CLINICAL FEATURES

May show the manifestations of the primary skin disease, examine the rest of the body with particular attention to the flexures, scalp, mouth and genitals.

INVESTIGATIONS

Rarely required, bacteriological swab where indicated, patch testing if allergic contact dermatitis suspected (we will do standard battery and perianal battery) skin biopsy in difficult to diagnose cases,

MANAGEMENT

- As for the primary skin disease.
- Advice on diet, lifestyle, smoking, alcohol etc.
• Advise on perianal cleansing, use a soap substitute, bidet if available.
• Avoid OTC creams, particularly anything with ‘caine’ in the title.
• Avoid using wet wipes
• It is quite safe to apply topical steroids above the potency of hydrocortisone.
• Apply steroid and then put a ‘barrier’ cream over it e.g. Aveeno, Cavalon.
• Maintain the barrier cream indefinitely.

WHEN AND WHERE TO REFER

1. When there is need of a diagnosis, and/or initial treatment has failed.
2. Refer to GUM if history suggestive of sexually transmitted disease.
PSORIASIS - CHRONIC PLAQUE

DEFINITION

Chronic skin disease characterised by well demarcated red scaly plaques commonly involving the elbows, knees and scalp. May have an associated arthritis. Now known also to be associated with an increased risk of type 2 diabetes, metabolic syndrome, inflammatory bowel disease and cardiovascular disease.

CAUSE

Bimodal presentation. Those presenting in younger age group most likely and those in older age group least likely to have a family history. Local trauma, acute illness, stress and drugs including beta blockers (particularly atenolol), lithium, terbinafine and anti malarials may precipitate the initiation or worsening of symptoms. Care should therefore, be taken when prescribing these drugs in patients with psoriasis or a family history of it.

CLINICAL FEATURES

Well demarcated red scaly plaques commonly on knees, elbows, scalp and trunk (Fig 1). Flexural psoriasis may often look more erythematous with less obvious scale but a maintained well demarcated edge. Pustular lesions may be present on the palms and soles (Fig 2). There may be a family history and if both parents have psoriasis 50% of children are likely to develop it. There may be associated pitting of the nails. Between 2% and 5% may also have an associated sero-negative arthritis.

DIFFERENTIAL DIAGNOSIS

The classical lesions are usually not difficult to diagnose. Lesions in the flexures, on the palms, soles and scalp may be more difficult to differentiate from eczema.

INVESTIGATIONS

None needed.

MANAGEMENT

As outlined in the NICE guidance (2012) this should include;

- Providing point of access to patients and families to aid access to information and advice about the condition and services available at each stage of the care pathway.
- Assessment of disease severity and impact and when to refer to specialist care.
- Identification of comorbidities (e.g. cardiovascular disease)

Therapy. This comprises:

- Topical therapy
- Second line and third line treatments (via secondary care) when topical therapy alone is unlikely to provide adequate control (e.g. extensive disease >10% surface
area, score of moderate or greater on static Physicians Global Assessment or conditions where topical therapy ineffective such as nails).

TREATMENT LADDER FOR TOPICAL TREATMENT (NICE 2012)

A) Trunk and limbs
   i) Potent topical steroid once daily plus vitamin D analogue once daily (one in morning and one in evening) for up to 4 weeks.

   ii) If no improvement at 4 weeks or satisfactory control not reached by 8 weeks offer vitamin D or Vitamin D analogue twice daily.

   iii) If satisfactory response not obtained in after 8-12 weeks offer either a potent topical steroid twice daily for up to 4 weeks or a coal tar preparation twice daily.

   iv) If the above cannot be used or a once daily preparation would improve adherence offer a combined product containing calcipotriol and betamethasone dipropionate daily for up to 4 weeks.
      - Potent topical steroid bd for 1 month
      - If no response topical steroid and Vitamin D analogue

B) Face, flexures and genitals
   i) Short term mild or moderate corticosteroid once or twice daily for maximum of 2 weeks.

   ii) If no response or risk of steroid induced side effects offer calcineurin inhibitor twice daily for up to 4 weeks.

C) Scalp
   i) Potent corticosteroid daily for up to 4 weeks.

   ii) If no satisfactory control at 4 weeks consider different formulation such as shampoo or mousse or descaling agents containing salicylic acids, emollients and oils before steroid application.

   iii) If unsatisfactory after 4 weeks offer combined calcipotriol/betamethasone add gel daily for up to 4 weeks or (if cannot use steroids or only mild disease) vitamin D or Vitamin D analogue.

   iv) If no clearance after 8 weeks try either very potent steroid up to twice daily for 2 weeks or a coal tar preparation once or twice daily or refer for specialist opinion.

The preparations mentioned above will include:

Vitamin D and Vitamin D analogues

- Ass Dovenex® 50micrograms/g (ointment 120g, scalp application 60ml) Apply once or twice daily to skin or twice daily to scalp.
- Calcitriol (Silkis®) 3 micrograms/g ointment 100g. Apply twice daily to no more than 35% of body surface area. Max: 30g per day.
- Tacalcitol (Curatoderm®) 4micrograms/g ointment 30g. Apply once daily, preferably at bedtime. Max: 40micrograms applied per day.
Vitamin D analogue and steroid

- Betamethasone and calcipotriol 0.05% / 50microgram/g (Dovobet®) ointment, gel
  Apply once daily to no more than 30% of the body surface. The ointment is licensed for psoriasis on the body. The gel is licensed for scalp and body. Usually, no more than 4g (1 teaspoon) is sufficient to treat the scalp.

Scalp preparations

- Polytar® liquid 150mL, 250mL, 500mL Apply once or twice weekly.
- Coal tar 1%, coconut oil 1%, salicylic acid 0.5% (Capasal®) shampoo 250ml Apply daily as necessary.
- Coal tar solution 12%, salicylic acid 2%, precipitated sulphur 4% (Sebco®) 40g, 100g Apply to scalp daily as necessary.
- Coal tar solution 12%, salicylic acid 2%, precipitated sulphur 4% (Cocois® ointment) 40g, 100g Apply to scalp once weekly as necessary. If scalp disorder severe, use daily for first 3 to 7 days. Shampoo off after 1 hour.

- Coal tar preparations

  - Coal tar extract 5%, hydrocortisone 0.5%, allantoin 2% (Alphosyl HC®) cream 100g Apply thinly to affected area twice daily.
  - Coal tar solution 5% (Exorex®) lotion 100mL, 250mL Apply to skin or scalp 2 or 3 times a day.

For secondary care

- Crude coal tar 2%, 5%, 10% in yellow soft paraffin 80g Apply once a day - retain on skin for between 30 minutes and 4 hours, then wash off. Can be used twice a day in severe cases.
- Salicylic acid 2%, coal tar 12% solution in emulsifying ointment (aka SCALP - Salicylic Acid Liquor Picis) (unlicensed) 250g Apply once daily, usually at night, then wash off the following morning.
- Salicylic acid 2% in white soft paraffin 500g Apply once or twice daily to areas of mild hyperkeratosis.
- Salicylic acid 2%, 5%, 10%, 20% in emulsifying ointment (unlicensed) 100g. Apply once daily, usually at night, then wash off the following morning.

**Topical steroids for flexural psoriasis**

Clobetasone butyrate 0.05%, oxytetracycline 3%, nystatin 100,000 units/g (Trimovate®) cream Apply to the affected area twice a day.

Or

Hydrocortisone 1%, miconazole 2% (Daktacort®) cream Apply to the affected area twice a day.

**Use of Dithranol Preparations**

Though not mentioned in the NICE guidance some patients do find this preparation helpful:

- Dithranol (Dithrocream®) 0.1%, 0.25%, 0.5%, 1%, 2% cream 50g. Apply to the affected area daily as needed. Start with 0.1% and titrate upwards every 2 days to
the maximum tolerated dose. Low-strength creams (0.1% to 0.5%) can be left on overnight. High-strength creams (1% and 2%) should be washed off after 1 hour. If burning occurs during this process:

- Eumovate® ointment should be applied to the sore areas. When the soreness has settled, the titration regimen is restarted at the previously tolerated strength.
- To aid compliance it is sensible to prescribe at least the first 3 strengths together to allow the patient to gradually increase the strength.

**THERAPEUTIC TIPS**

The above guidance notwithstanding, there are a huge range of treatments available for psoriasis and the treatment has to be tailored to the patient, the type of psoriasis they have and the site of the psoriasis. Most patients will benefit from some form of emollient to the skin and many patients will have their own favourite preparations. Despite what the textbooks say, psoriasis is usually itchy and the emollients will help in reducing that itch particularly at night. It is still generally agreed that potent flourinated topical steroids do not have a regular role in the management of chronic plaque psoriasis. They may be used under very specific circumstances on specific sites but only under strict supervision. Specific tips include:

Psoriasis patient should be advised on several life style issues, ideal body weight, stop smoking and reduce alcohol consumption. All these can make psoriasis worse and/or hinder treatment.

Many patients with psoriasis will have had psoriasis for many years and they will know what works and does not work for them. They can be 'an expert patient'.

**General principles**

- Ensure patients understand when and how to use their treatments.
- Consider recommending purchasing a 'pre-paid' prescription as patients will often require multiple products for different areas.

**Face**

Can be treated with tar preparations, mild topical steroids, tacalcitol, calcitriol (not calcipotriol).

**Scalp**

- Tar shampoos such as Alphosyl, Polytar or Capasal with added Salicylic acid.
- Tar preparations can be left in for several hours or overnight to be washed out with shampoo.
- If scalp psoriasis extends onto the face calcitriol or mild tar preparation may be used.

**Flexures**

- Dithranol, calcipotriol and tacalcitol should be avoided but calcitriol can be used. If there is 'intertriginous change then a steroid/antibiotic/antifungal mixture can be tried such as trimovate, daktacort, Canestan HC etc.

**Hands and Feet**

- Topical steroids are often the mainstay for these areas. If hyperkaratotic change then worth trying diprosalic or Haelan tape for fissures etc

**MANAGEMENT OF MODERATELY TO SEVERE PSORIASIS**
Inevitably the majority of these cases will be managed in secondary care but we would hope to share care in many cases. Patients on ultraviolet light therapy and PUVA therapy will be under the exclusive care of the hospital but those who are on a second line treatment such as Methotrexate, Hydroxy carbamide or Cyclosporin shared care arrangements may be in place. Regular monitoring of full blood count and liver function tests in those on Methotrexate and the creatinine level and blood pressure of those on Cyclosporin are very helpful in their long term monitoring.

WHEN AND WHERE TO REFER

- Erythrodermic psoriasis - urgent referral to hospital.
- Pustular psoriasis
- Chronic plaque psoriasis unresponsive to the above management.
- Widespread psoriasis (especially small plaque psoriasis). Management at home can be difficult and referral to consider UVB, admission or day care attendance is an option.

REFERENCE/BIBLIOGRAPHY


Fig 1 Psoriasis – Knees

Figure 2 Palo-plantar Pustulosis
PSORIASIS - GUTTATE

DEFINITION

Recent onset widespread small psoriatic lesions.

CAUSE

It commonly occurs 7 to 10 days after a Streptococcal sore throat. It may be the first episode of psoriasis in an individual or may occur in patients who already are known to have psoriasis. Tends to affect children and young adults. Whilst some will go on to develop classical psoriasis, many do not, especially those without a family history.

CLINICAL FEATURES (Fig 3)

Multiple widespread small 1 -2 cm diameter, red, well demarcated lesions with silvery scales affecting most of the trunk and limbs and often sparing the face. Individual lesions may exhibit the koebner phenomenon (lesions in the sites of trauma and scars) which will help diagnostically. It tends not to be itchy and usually resolves spontaneously over a few months.

DIFFERENTIAL DIAGNOSIS

Pityriasis rosea. This will classically have a herald patch which predates the widespread lesions and is larger. Also there is less scaling over the centre of the lesions which tend to be paler.

INVESTIGATIONS

Throat Swab.
ASO Titre.

MANAGEMENT

- Topical tar based preparations such as Exorex lotion (not cream). If itchy a mild topical steroid may be helpful. Also copious emollients for the itch.
- Out patient ultra-violet light. Do NOT advise patient to use a sunbed.
- Reassurance that the lesions will resolve spontaneously with time.
- Penicillin V for a Streptococcal sore throat.

WHEN AND WHERE TO REFER

- If the diagnosis is in doubt.
- If lesions do not resolve within the expected time scale.
- If the lesions are particularly bothersome and do not respond to tar based products then referral for UVB treatment may be considered.
Fig 3 Guttate Psoriasis with Koebner phenomenon
ROSACEA

DEFINITION

This is an inflammatory condition usually affecting the face of middle aged patients comprising erythematous and papular/pustular components. Patients often have a history of flushing and blushing.

CAUSE

Several factors have been claimed to influence onset of the disease including a greasy type skin, excess alcohol, hot drinks and spicy foods. Demodex mites have in the past been implicated in the aetiology but have now been largely discounted. In others sun exposure and emotional stress seem to exacerbate their condition. May be genetic, American dermatologists refer to it as ‘the curse of the Celts’

Can be precipitated by the inappropriate application of topical steroids to the face

CLINICAL FEATURES

- The condition inevitably has an erythematous component made up of flushing and telangiectasia. This is classically distributed over the butterfly area of the nose and cheeks although the forehead and chin can also be involved.
- Within the areas of erythema most patients ultimately develop papules and pustules which "come and go". As with the erythema these usually involve the butterfly area but can involve the forehead and chin but rarely extend off the face.
- Severe longstanding cases may result in a bulbous greasy hypertrophy of the nose (rhinophyma).
- A number of patients with rosacea also develop ocular irritation.

DIFFERENTIAL DIAGNOSIS

1. **Systemic lupus Erythematous**
   No papules or pustules are usually seen in this condition and anti nuclear antibodies are often found.

2. **Seborrhoeic Dermatitis**
   Pustules are uncommon in this condition and the scaling of the eyebrows, nasolabial folds and scalp etc typical of this are unusual in rosacea.

3. **Acne**
   This may well involve the chest and back and have open comedones (blackheads) which are not a feature of rosacea.

4. **Peri-oral ‘dermatitis’**
   This tends to affect the muzzle area and is often triggered by the use of topical steroids

INVESTIGATIONS

None required. In atypical rosacea a biopsy will sometimes help to differentiate this from other differential diagnoses.
MANAGEMENT

a. General Advice
Many patients will note exacerbating factors for the erythema/pustules or both. These include alcohol, hot drinks, spicy foods, sun and wind etc and should obviously be avoided wherever possible in such patients.

b. Topical Therapies
Topical metronidazole products may help mild rosacea though they are rarely as effective as oral agents. They may also be used at the end of a course of antibiotics to facilitate their tapering or withdrawal.

Wirral formulary recommends Metronidazole (Rozex®) 0.75% cream / gel Apply to the affected areas twice daily for 3 to 4 months. If sustained improvement is evident, continue for a further 3 to 4 months.

c. Systemic Therapy
The mainstay of treatment of rosacea is with the tetracycline antibiotics. An initial treatment course should be for at least 3 months. A good response at 3 months would be a 50% to 60% improvement and as a general rule the papules and pustules respond better than the erythematous component. Treatment should be continued until remission and then the patient gradually weaned off treatment over the course of several months.

On subsequent withdrawal some will remain clear and require no further treatment whilst others will relapse and require a further few months of treatment. Another sub group relapse as the dose is reduced or immediately on stopping and this group often need to remain on treatment long term. Approximately 1/3 fall into each of the above categories.

Patients with ocular rosacea will require systemic treatment

Wirral formulary recommended antibiotics are:

- **Oxytetracycline** 500mg, orally, twice daily for 6 to 12 weeks.
  - Or
  - **Lymecycline** 408mg, orally, daily for 6 to 12 weeks
  - **NOTE: Unlicensed indication**
  - Or
  - **Doxycycline** 50mg, orally, daily for 6 to 12 weeks.
  - Or
  - **Metronidazole** 200mg, orally, three times a day for 6 to 8 weeks
  - **NOTE: Unlicensed indication**
  - Or
  - **Erythromycin** 500mg, orally, twice daily for 6 to 12 weeks

A group of patients seem to have very resistant disease and on occasions Isotretinoin (Roaccutane) is helpful although the response is not as reliable as in nodulo-cystic acne.
Flushing is less amenable to therapy but may be helped by propranolol 40mg bd or clonidine 50 micrograms bd. Fixed erythema will sometimes respond to laser therapy and this modality may also be required for severe rhinophyma.

WHEN AND WHERE TO REFER

- Atypical cases where diagnosis is unclear.
- Severe cases unresponsive to treatments outlined above.
- Those who are for consideration for Roaccutane.
- Those with severe erythema or rhinophyma who may require laser therapy.
SCABIES

DEFINITION

A superficial skin infestation with the mite (Sarcoptes scabei var. hominis).

CAUSE

Human scabies is transmitted from person to person by prolonged physical contact e.g. holding hands, lying in the same bed, cuddling children and not by inanimate objects (e.g. not by toilet seats!). Animal scabies does not normally cause significant problems in humans.

Mites burrow through the stratum corneum and fertilized females lay eggs that mature to adults in two to three weeks. The generalised rash and itching of scabies is considered to be due to a hypersensitivity reaction to the mites and their faeces. The number of mites on an individual may be quite small.

CLINICAL FEATURES

- Delay of 4-6 weeks before symptoms occur following infestation.
- Pruritus is the usual primary feature and is especially marked at night.
- Burrows are diagnostic – Finger webs. Fig 1
  Hands on sides, palms.
  Wrists - flexor aspect.
  Ankles and feet (especially in infants).
  Elbows.
- Visualisation: Burrows are fine linear tracts visible to the naked eye via a hand lens. Fig 2
  Sarcoptes mite measures 0.3 to 0.4 mm is seen as a tiny black dots at the end of a burrow (better still with a dermatoscope).
- Papules and nodules on the penis and scrotum Fig 3 (often causing rubbery papules), also the nipples of the female.
- Distribution: Neck downward.
  Face and scalp in infants and immunocompromised.
- Secondary eruption with papules that may be excoriated, urticarial or eczematised and impetiginised occurs predominantly on the trunk and is non specific.
- Itching in close contacts.

DIFFERENTIAL DIAGNOSIS

- Any itchy rash e.g. dermatitis herpetiformis, lichen planus and atopic eczema.
- Especially if not responding to conventional treatments.

INVESTIGATIONS

Skin scraping of a burrow and identification of a mite under the microscope. Fig 4
MANAGEMENT

- Treat the patient
- Contacts: All members of the household and sexual and close social contacts irrespective of whether they are itching or not.
- Topical scabicides:
  a) Permethrin 5% (Lyclear dermal cream) - 60g needed for a single application for one adult, safe in pregnancy.
  b) Malathion 0.5% aqueous solution (Derbac-M) - 100 ml needed for a single application for one adult.
  c) Benzyl benzoate – irritant, avoid in children, pregnancy and lactation.
- Rub into ALL parts of body (chin down, unless infants and immunocompromised). Brushed under finger and toe nails.
- Left on prescribed period of time – Permethrin 8-12 h, Malathion 24 h.
- Re-application after hand washing.
- Repeat same treatment 1 week later.
- Information sheet to the patient to reinforce instruction (otherwise it will be used wrongly and will fail!).
- Post-scabetic itch - itching may last days or even weeks after the application (due to sensitivity to the dead mites and their debris). Eurax-Hc, Calamine can be helpful for the itch.
  Higher potency topical steroids may be needed to treat the secondary eczema.
- Do not use scabicides as repeated applications:
  Not necessary if use correctly.
  Wrong diagnosis.
  Irritant exacerbating eczema.
- Disinfestation of clothing and bedding is NOT necessary, only ordinary laundering.

WHEN AND WHERE TO REFER

- Recalcitrant cases.
- Diagnosis is unclear or with other severe pre-existing dermatoses.
- Outbreaks in residential or nursing homes may require advice from public health to ensure adequate eradication.

Fig 1  Finger web involvement
Fig 2  Burrow and mite (black dot at the right end of track)

Fig 3  Penile nodules

Fig 4  Scabies mite
URTICARIA

DEFINITION

Recurrent wheals (hives) with individual lesions lasting less than 24 hours.

CAUSE

The vast majority of cases are idiopathic and no cause is found. Majority of cases are probably a form of auto-immune disease with antibodies against the Mast cell fixed IgE. Physical urticaria can be triggered by trauma, pressure, changes in temperature or exercise. Urticaria occasionally triggered by drugs e.g. aspirin, codeine, NSAIDs and ACEIs. It is occasionally triggered by foods such as fish, nuts, chocolate and very rarely additives. It has been reported in conjunction with chronic and non specific infections such as streptococcal, campylobacter and parasites.

CLINICAL FEATURES

Recurrent wheals that can appear anywhere. Although the condition may be chronic the fact that each lesion lasts less than 24 hours is diagnostic. There may be associated dermographism (Fig 1). In many cases attacks are relatively short lived but it is common for idiopathic urticaria to persist for several months if not longer.

DIFFERENTIAL DIAGNOSIS

Other causes of pruritus.

INVESTIGATIONS

Food allergy or physical causes for urticaria are usually obvious. Allergy tests such as patch tests and prick tests are of NO value. No investigations are usually necessary unless suggested by the history.

MANAGEMENT

Non sedating antihistamines are the mainstay of treatment and should be given regularly until the condition has been under control for a period before withdrawal. All of the non sedating antihistamines are safe and should be tried in rotation to find the one which suits the patient best. The dose of all the current non sedating antihistamines are safe at twice or three times the normal dose and should be tried before referral.

Non-sedating antihistamines (Wirral formulary)

**Cetirizine** 10 to 20mg, orally, daily (licensed maximum: 10mg daily).

Or

**Loratidine** 10mg, orally, daily [*added by the PCT]*

Or

**Fexofenadine** 180 to 360mg, orally, daily.

Itch is often worst at night in which case a sedating antihistamine can be given in addition at night.
Sedating antihistamines *(Wirral formulary)*

**Alimemazine** *(trimeprazine)* 10mg orally 2 or 3 times daily (maximum 100mg daily in severe cases).

*NOTE: VERY sedating. Exercise caution in use outside of hospital*

*Or*

**Hydroxyzine** *(very sedating)* 25mg orally at night, increase if necessary to 25mg 3 or 4 times daily.

*Or*

**Chlorpheniramine** 4mg, orally, up to four times a day as needed.

If there is inadequate response to a type I antihistamines then a type II antihistamine such as ranitidine (150mg bd) or cimetidine (400mg bd) can be added in.

NB Steroids have no place in the management of chronic urticaria and are limited to the short term treatment of angio-oedema.

Facial oedema as part of chronic urticaria is not life threatening and patients can be reassured.

**WHEN AND WHERE TO REFER**

- If the diagnosis in doubt.
- If there is failure to respond to regular use of antihistamines in full doses when other treatments such as ciclosporin, dapsone, warfarin, methotrexate etc might be considered.
- Patients with allergic reactions to foodstuffs, be stings etc are best referred to immunology

**ANGIOEDEMA (without urticaria)**

Whilst angioedema is common in patients with urticaria (and is usually not life threatening to these patients) it can sometimes occur in the absence of urticaria. Such patients may have:

ACE inhibitor related angioedema

This usually occurs within 3 weeks of starting the drug though then timescale can be longer.

Patients will be intolerant of the other ACE inhibitors and sometimes Angiotensin11 inhibitors.

**C1 ESTERASE INHIBITOR DEFICIENCY**

- Most cases start before puberty though onset can be delayed until adult life
- Most patients are hereditary though a small proportion can be secondary to lymphoproliferative or autoimmune disorders.
- Diagnosis is by demonstrating low C1 esterase levels and function

**SPONTANEOUS ANGIOEDEMA**

- Occasional cases may not satisfy the above and have idiopathic angioedema.
• Differential to exclude might include contact allergies to hair dyes, lip products and toothpastes, food allergy and fixed drug eruptions.

TREATMENT

• Stop any offending drug.
• Treat with antihistamines, short courses of oral steroids plus epipen if clinically indicated.

Fig 1 Urticaria - Dermographism
VASCULAR BIRTHMARKS

SALMON PATCH

- Stork mark (nape of neck) or angel’s kiss (forehead).
- Present at birth.
- Pale pink /salmon macular discoloured patch.
- Facial lesions tend to fade.
- Nape of neck lesion persist into adult life in 50% of patients.
- No treatment required.

STRAWBERRY BIRTHMARKS

- 1% of all births.
- Present at birth or appearing soon after.
- Single or multiple with 60% in the head and neck region. Fig 1
- Can superficial, deep or mixed.
- Three phases of development
  1. Proliferative phase: occurs in the first 6-9 months. Can grow rapidly and ulcerate especially at sites of friction and be painful. Fig 2
  2. Stabilisation phase.
  3. Involution phase: usually between 6 – 18 months of life. Spontaneous change include loss of the brilliant red colour, flattening, softening and shrinkage of the lesion.
- 90% will regress completely without any intervention by the age of 9.
- Residual skin changes include atrophy, altered texture or telangiectasia.
- Reassurance - most do not require treatment.
- Refer if:
  a) Cosmetically important area.
  b) Interfering with function – breathing, feeding, vision.
  c) Complication of ulceration or bleeding.
  d) Very large lesions.
  e) Areas of necrosis due to platelet consumption Kasabach Merrick syndrome

PORTWINE STAINS

- Present at birth.
- Colour varies from deep pink to dark red to purple.Fig 3
- Persists throughout life.
- Grows with the patient.
- Commonly on the face in the trigeminal nerve distribution.
- Tendency to darken and thicken with age. Fig 4
- Cosmetic problem.
- Amenable to laser treatment and cosmetic camouflage.
Fig 1  Strawberry birthmark

Fig 2 Ulcerated strawberry birthmark during proliferative stage

Fig 3  Port wine stain

Fig 4 Thickening of port wine stain
**VIRAL WARTS**

**DEFINITION**

Viral warts are an extremely common viral infection of the skin due to the human papilloma virus and will remit spontaneously given time.

**CAUSE**

Human papilloma viral infection of the epidermis.

**CLINICAL FEATURES**

- Present as single or multiple papillomas.
- The majority of lesions occur on the palms of the hands or the soles of the feet and occasionally on the knees and face.
- Plane warts present as flat areas usually on the face.

**DIFFERENTIAL DIAGNOSIS**

Widespread lesions may be mistaken for molluscum contagiosum.

**INVESTIGATIONS**

None required.

**MANAGEMENT**

1. There is a profusion of over the counter wart treatments which if used on a daily basis, appropriately applied should be efficacious with time.
2. Cryotherapy is a logical alternative. Cryotherapy needs to be done regularly and properly. At least one 20 second freeze thaw cycle with the C nozzle of the Cryac™, maybe two freeze thaw cycles for a plantar wart. This to be repeated every three weeks. Resolution may take 6 to 12 treatments.
3. It is useful to combine the cryotherapy with other topical wart treatments, particularly salicylic acid containing products. It is not advisable to treat children with cryotherapy.
4. There are more and more anecdotal reports of the efficacy of ‘Duct tape’ to make it worth a try.

**WHEN AND WHERE TO REFER**

1. Referral to secondary care for the classic verruca vulgaris, verruca plantaris and plane warts is rarely justified..
2. Post-transplant patients or other immunocompromised patients may require secondary care input.
3. Genital warts should be referred to GUM clinic.
VITILIGO

DEFINITION
Localised to widespread area of depigmentation due to the absence of melanocytes and melanin in the skin.

CAUSE
Affects 1-2% of the world's population. Incidence higher in racially pigmented skin. There is often a family history of vitiligo. Probably an autoimmune disease as vitiligo is frequently associated with other autoimmune conditions e.g. autoimmune thyroid disease and pernicious anaemia.

CLINICAL FEATURES
Depigmented macules can occur on any part of the body. Nearly a half of cases will occur in children and teenagers. Areas that are subjected to trauma, dorsum of hands, and the normally hyperpigmented parts of the body, perianal, genital and nipples, are particularly prone to vitiligo. Vitiligo can occur in damaged skin – the Koebner phenomenon. The skin around a patch of vitiligo may become hypopigmented. Hair on the head, beard, eyelashes, eyebrows and body may become involved in the vitiligo process.

DIFFERENTIAL DIAGNOSIS
1. Tinea versicolor; usually confined to the back and chest. Will fluoresce under Wood's light. Hyphae and spores will be seen with a KOH wet preparation
2. Pityriasis alba - usually on the face of children with atopic diathesis.
3. Piebaldism will have been recognised form early infancy.

INVESTIGATIONS
Examination under Wood’s light will confirm. Autoantibody screen with particular reference to thyroid antibodies may be done.

MANAGEMENT
- Try and treat as early as possible in the history of the disease. Older established areas of vitiligo rarely respond to treatment.
- Small, early, localised areas of vitiligo on the trunk and limbs can be treated with potent topical steroids – 0.05% clobetasol propionate.
- For the face there are positive reports on the use of topical calcineurin inhibitors, tacrolimus or pimecrolimus.
- Widespread vitiligo may respond to a prolonged course of TL01 narrow band UVB.
- Advice on photo protection of vitiligo affected skin is vital.

WHEN AND WHERE TO REFER
1. If concerned over using potent topical steroids or prolonged use of calcineurin inhibitors on the face.
2. Refer widespread vitiligo for consideration of TL01 UVB.
ADVICE AND SUPPORT

XERODERMA

DEFINITION

Widespread areas of dry skin often with secondary eczema in the elderly.

CAUSE

- Most patients with atopy have generally dry skin.
- There are several hereditary "ichthyoses" which give varying degrees and distributions of dry skin but these are generally uncommon.
- The commonest presentation occurs in the elderly and is often complicated by "asteatotic eczema".

CLINICAL FEATURES

- Dry skin associated with atopy is usually generalised although it does tend to spare the flexures to some degree.
- The congenital ichthyoses are often more severe and may in some cases also involve the flexures and in some localised forms give significant hyperkeratosis of the hands and feet.
- Ichthyosis of the elderly is usually most marked in the lower legs (often complicated by asteatotic eczema) although the involvement can be generalised.

DIFFERENTIAL DIAGNOSIS

Atopic dermatitis, but distribution is typical and patient may give history of associated asthma and hay fever.

INVESTIGATIONS

1. None are usually required.
2. The rare congenital ichthyoses sometimes may require specialist investigation for definitive diagnosis.

MANAGEMENT

- This rests largely with emollients in large quantities for all types of ichthyosis (see Wirral formulary).
- Different patients will find different preparations helpful and patients should therefore be allowed to try various emollients continuing with the one which "works best for them".

WHEN AND WHERE TO REFER

1. Generally referral is not required.
2. Usual hereditary ichthyoses may require referral to specialist centres if particularly severe.
SECTION 2

TREATMENTS
CRYOTHERAPY (CUTANEOUS CRYOSURGERY)

DEFINITION

The branch of therapeutics that makes use of local freezing for the controlled destruction or removal of living tissue. It has become a widely used therapeutic tool in the treatment of many cutaneous conditions.

MECHANISM OF ACTION

Cellular injury (and subsequent death) is produced by:
1. Ice formation; (intracellular and extra cellular).
2. Osmolality change; (ice formation leads to osmolality changes).
3. Vascular change; (vasoconstriction leads to microthrombi formation and ischaemic necrosis).
4. Host immune system stimulation; (creation or revealing of new antigenic material).

METHODS

Liquid nitrogen has superseded other refrigerants (e.g. nitrous oxide or carbon dioxide - dry ice). Liquid nitrogen spray equipment although initially expensive to purchase is robust with little maintenance cost. It is convenient and simple to use and the same method can be used to treat benign, pre-malignant and malignant conditions. Before using cryotherapy it is advisable to undergo training in the techniques of treatment.

LESIONS AMENABLE TO TREATMENT

Details of time of freeze given below specifically refer to Cryac using C nozzle (Other delivery methods/equipment might vary in their freeze thaw cycle recommendations).

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Beginner or Expert</th>
<th>Time of Treatment (sec)</th>
<th>No. of freeze/thaw cycles</th>
<th>No. of sessions</th>
<th>Repeat Interval (weeks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acne cyst</td>
<td>Expert</td>
<td>5-10</td>
<td>1</td>
<td>2-3</td>
<td>4</td>
</tr>
<tr>
<td>Chondrodermatitis nodularis helicis</td>
<td>Beginner</td>
<td>10-15</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Cutaneous horn</td>
<td>Beginner</td>
<td>10-15</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Dermatofibroma</td>
<td>Beginner</td>
<td>20</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Disseminated superficial actinic keratosis</td>
<td>Beginner</td>
<td>10</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Granuloma annulare</td>
<td>Expert</td>
<td>5-10</td>
<td>1</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Hypertrophic scar</td>
<td>Expert</td>
<td>20-25</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Ingrowing toenail (granulation tissue)</td>
<td>Expert</td>
<td>20</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Lentigines</td>
<td>Beginner</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Milia</td>
<td>Beginner</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Molluscum contagiosum</td>
<td>Beginner</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Myxoid cyst (digital)</td>
<td>Expert</td>
<td>20-30</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Seborrhoeic keratosis</td>
<td>Beginner</td>
<td>10</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>Level</td>
<td>Treatment 1</td>
<td>Treatment 2</td>
<td>Treatment 3</td>
<td></td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
<td></td>
</tr>
<tr>
<td>Skin tags</td>
<td>Beginner</td>
<td>10-15</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Solar (Actinic keratosis)</td>
<td>Beginner</td>
<td>10</td>
<td>1</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Viral warts (hands/fingers)</td>
<td>Beginner</td>
<td>20</td>
<td>1</td>
<td>6+</td>
<td>3</td>
</tr>
<tr>
<td>Viral warts (plain)</td>
<td>Beginner</td>
<td>5</td>
<td>1</td>
<td>3-4</td>
<td>4</td>
</tr>
<tr>
<td>Viral warts (feet)</td>
<td>Beginner</td>
<td>20</td>
<td>1-2</td>
<td>6-12</td>
<td>3</td>
</tr>
</tbody>
</table>

Lesions NOT to treat:
- Naevi (moles) and undiagnosed pigmented lesions
- Malignant lesions unless directed by a member of the skin cancer MDT

Complications: Pain, blisters, secondary infections, tendon damage, pigmentary changes.

Extra equipment: - Cryotherapy dispenser, liquid nitrogen storage.

WHEN AND WHERE TO REFER
- Lesions where diagnosis uncertain
- Medicolegal aspects: Consent
DERMATOLOGICAL THERAPY

TOPICAL THERAPY

A number of general principles are as true today as they have been over the centuries.

VEHICLE

Weeping/exudative eruption:-
Use a cream or a lotion (non occlusive and will allow evaporation to occur). Also consider the use of astringents such as potassium permanganate or aluminium acetate soaks.

Dry/Scaly eruption:-
Use an ointment (occlusive base will help to retain "moisture" and thereby improve efficacy). Ointments (containing no preservatives) are also likely to be better tolerated in those with allergies to medicaments or preservatives.

INFECTION

- Local infection - use topical antibiotic.
- Generalised infection - use systemic antibiotic.
- As a general rule keep courses short to minimise the development of resistance.

EMOLLIENTS

- All patients with dry/lichenified rashes should use liberally, both topically onto the skin and when bathing. Regular use often reduces the amount of steroid needed in general, especially in children.
- Patient preference and therefore compliance varies. Rotate the emollients until you find one the patient is happy with.
- Prescribe bath oils and emollients in large amounts i.e. 500g tubs/500ml pumps NOT 15g tubes!

Emollient preparations – general advice

Suitable quantities of emollients to be prescribed for specific areas of the body (assuming the emollient is used twice daily for one week) are:

<table>
<thead>
<tr>
<th>area of the body</th>
<th>creams / ointments</th>
<th>lotions</th>
</tr>
</thead>
<tbody>
<tr>
<td>face</td>
<td>15–30g</td>
<td>100mL</td>
</tr>
<tr>
<td>both hands</td>
<td>25–50g</td>
<td>200mL</td>
</tr>
<tr>
<td>scalp</td>
<td>50–100g</td>
<td>200mL</td>
</tr>
<tr>
<td>both arms or both legs</td>
<td>100–200g</td>
<td>200mL</td>
</tr>
<tr>
<td>trunk</td>
<td>400g</td>
<td>500mL</td>
</tr>
<tr>
<td>groins and genitalia</td>
<td>15–25g</td>
<td>100mL</td>
</tr>
</tbody>
</table>

An average adult requires 25 g to cover whole body once.

NOTE: These quantities will need to be increased significantly for patients with severe exacerbations of skin disease when emollients will need to be applied 5–6 times a day.
Wirral formulary options for greasier emollients include:

- Oily cream (hydrous ointment BP) 500g
- 50% liquid paraffin / 50% white soft paraffin 500g
- Emulsifying ointment 500g
- Epaderm ointment 125g, 500g, 1,000g
- Diprobase ointment 50g, 500g
- Hydromol ointment 125g, 500g, 1,000g

Wirral formulary options for emollient creams include:

- Cetraben cream 500g
- Doublebase gel 500g
- Diprobase cream 500g
- E45 cream 50g, 125g, 350g, 500g
- Epaderm cream 50g, 500g
- Hydromol cream 50g, 100g, 500g
- Nutraplus (urea 10%) cream 100g
- Oilatum cream 40g, 150g, 500ml, 1,050ml
- Oilatum Junior cream 150g, 350ml, 500ml, 1,050ml

Wirral formulary options for emollient lotions include:

- E45 lotion 200ml, 500ml
- QV lotion 250ml

Patients with chronic skin conditions often require total emollient therapy (i.e. a mixture of emollients including one to use as a soap substitute or bath oil).

Wirral formulary options for emollient soap substitutes/bath oils are:

- Dermalo bath emollient 500mL
- Emulsifying ointment 500g
- Hydromol (bath emollient) 350mL
- Oilatum Emollient (bath emollient) 250mL, 500mL
- Oilatum Gel 150g (soap substitute)
- QV bath oil 200mL, 500mL [PCT suggest]
- Aqueous cream (use as a soap substitute ONLY) 100g, 500g

**NOTE:** Aqueous cream is NOT an effective moisturiser and can irritate some patients’ skin. It must be washed off after application.

The following preparations should only be prescribed on the advice of a dermatology specialist or when other emollients have been tried and failed:

- Dermamist spray 250ml
- Emollin spray 150ml, 240ml
- Aveeno cream 100mL, 300mL

If the emollient products listed proves unsuitable for a particular patient, clinicians should choose a cost-effective alternative.
TOPICAL STEROIDS

Strength (depends on site)

<table>
<thead>
<tr>
<th>Site</th>
<th>Acute Eruption</th>
<th>Reduce To</th>
</tr>
</thead>
<tbody>
<tr>
<td>Face</td>
<td>Moderate</td>
<td>Mild</td>
</tr>
<tr>
<td>Trunk/Limbs</td>
<td>Potent</td>
<td>Moderate</td>
</tr>
<tr>
<td>Scalp</td>
<td>Very potent/potent</td>
<td>Potent</td>
</tr>
<tr>
<td>Hands/Feet</td>
<td>Very potent</td>
<td>Potent</td>
</tr>
</tbody>
</table>

In children use weakest possible to obtain effect. 1% Hydrocortisone is relatively safe in children and can usually be used without problem (the problem is often to get the parent to use enough!).

All scalp preparations except Betnovate lotion and Synalar gel contain alcohol and so will 'sting' if put on broken skin.

Wirral formulary recommendations are:

Mild potency
- **Hydrocortisone 1% cream / ointment [0.5% removed]**

Moderately potent
- Clobetasone butyrate (Eumovate®) 0.05% cream / ointment
- Betamethasone butyrate (Betnovate-RD®) 0.025% cream / ointment
- Fludroxcortide (Haelan®) 0.0125% cream / tape (4 microgram/cm²)
- Fluocinolone acetonide (Synalar 1-in-4®) 0.00625% cream / ointment

Potent
- Betamethasone valerate (Betnovate®) 0.1% cream / ointment / lotion / scalp application
- Fluocinolone acetonide (Synalar®) 0.025% cream / ointment / gel
- Betamethasone dipropionate (Diprosone®) 0.05% cream / ointment
- Fluocinonide (Metosyn®) 0.05% ointment
- Betamethasone valerate (Betesil®) plasters — for initiation by dermatologists ONLY for chronic lichenified eczema

Very potent
- Clobetasol propionate (Dermovate®) 0.05% cream / ointment / scalp application

How Much To Use?

Suitable quantities of corticosteroids for two weeks' treatment are:

- Trunk: 100g to 200g
- Arms and legs: 100g to 200g
- Whole body: 300g to 400g

Average adult requires 20-25 g to cover whole body once
Finger tip unit - 1 "sausage" from tip to DIP finger crease is correct amount to cover 2 "hand prints" of same patient. Prescribe enough!
SKIN SURGERY

DEFINITION

Many simple benign skin lesions lend themselves to easy removal by an adequately trained general medical practitioner. Following recent government initiatives, the Calman Heine report and the National Institute for Health and Clinical Excellence ‘Improving Outcomes for People with Skin Tumours including Melanoma’ (Feb 2006) all suspected skin cancers must be referred to an accredited specialist centre. Biopsies and excisions of suspected cancers, particularly those on the head and neck should not be done out with these centres.

The techniques available to the interested GP include curettage, cautery, shave excision and elliptical excision. Cryotherapy is dealt with in a separate section. The benign lesions that could be dealt with include skin tags (achrocordon), basal cell papillomas (seborrhoeic warts), simple squamous papillomas, viral warts, actinic keratoses, dermatofibromas, epidermal cysts, pilar cysts, benign naevi and in-growing toe nails. As a general rule if you cannot diagnose a lesion clinically then it would be better to refer on rather than excise blindly.

It is generally agreed that one should not biopsy a pigmented lesion, either cut it out in its entirety or refer on for a second opinion. Unless particularly experienced it is probably sensible to avoid operating on lesions on the face. Prior to operating informed consent must be obtained from the patient, the reason for doing the operation must be explained, as must be all possible complications and alternative treatments.

ANAESTHESIA

Nearly all procedures will require some form of anaesthesia. Pre treatment of the area with a topical local anaesthetic e.g. EMLA or Ametop is essential if operating on a child and might be reassuring for the nervous adult. Otherwise use intradermal injection of Lidocaine 1% to 2%. The addition of adrenaline prolongs anaesthesia and will also reduce bleeding at the time of operating.

Adrenaline should never be used on:

- digits
- genitalia
- areas of previous radiotherapy
- and not advised in those with angina or on Beta-blockers

Always allow time for the local anaesthetic to work, usually 2 minutes.

CURETTAGE

Ideal for viral warts, basal cell papillomas and hyperkeratotic actinic keratoses. The ring curette gives an excellent cosmetic result. Haemostasis following curettage can be obtained using silver nitrate, Monsel's solution or cautery by heat or electrocautery.
SHAVE EXCISION

This is the technique of choice for pedunculated naevi and achrocordon. Again haemostasis to the base of the shave can be obtained using silver nitrate, Monsel's solution or cautery.

ELLIPtical EXCISION

Using the classic 3 to 1 ellipse and ensure that the scar lies in or parallel to relaxed skin tension lines. Cut deep enough to:

- ensure complete excision of the lesion, and
- to get to the next anatomical layer to allow neat closure

Haemostasis is obtained with cautery or ligation. Closure of a wound should be in layers with an absorbable subcuticular suture such as Monocryl and monofilament nylon to the surface. Interrupted sutures will create less tension and scarring in the long term. Use the appropriate suture size for the area you are suturing for instance, 60 for the eyelid, lip and cheek, 50 for the forehead, 40 for thin limb skin and the abdomen, and 30 for thick limb skin and the back. Sutures should be removed from the face in 5 to 7 days, the trunk in 10 to 12 days, the arm in 10 to 12 days and the leg in 14 days. It is advisable to reinforce all wounds with Steristrips and/or a Tegaderm dressing after sutures have been removed.

Always

1. warn the patient of post operative scarring and pain
2. infection risk and
3. send ALL removed tissue for histology
SECTION 3 – Educational Resources

British Association of Dermatologists (BAD)
- Excellent Resource for Patient Information Leaflets

Primary Care Dermatology Society (PCDS)
- Clinical resource aimed at Primary Care Physicians. Good images
- Educational material re dermoscopy

New Zealand Dermatology Society
- Concise summaries of clinical information (with images) about most dermatological conditions. Of use to all groups from patients to secondary care clinicians
SECTION 3

PATIENT SUPPORT GROUPS
ACNE SUPPORT GROUP
First Floor,
Howard House,
The Runway,
South Ruislip,
Middlesex HA4 6SE
Tel: 020 8841 8400

Allergy UK
3 White Oak Square
London Road
Swanley, Kent
BR8 7AG
Charity Reg. N 1003726
Tel: 01322 619 898
Fax: 01322 663 480
Helpline: 01322619 864
Email: info@allergyuk.org
Web: www.allergyuk.org

Alopecia Awareness
Alopecia Awareness
90 Plymouth Road
Plympton
Plymouth
PL7 4NB
Tel: 07890 246 398
Website: www.alopecia-awareness.org.uk

Alopecia Help and Advice (Scotland) Limited
Rosemary Gierthy, Chair
9 Bangholm Bower Avenue
Edinburgh
EH5 3NS
Website: www.alopeciascotland.co.uk
Registered Office: 33 Burnbank Road, Grangemouth FK3 8RU

Alopecia UK
Alopecia UK
5 Titchwell Road
London
SW18 3LW
Tel: 0208 333 1661
E-mail: info@alopeciaonline.org.uk
Website: www.alopecia.org.uk
Behcet's Syndrome Society

8 Abbey Gardens
Evesham
Worcester
WR11 4SP

Tel: (0845) 130 7328 (Office)
Helpline: (0845) 130 7329 (Local rate)
Email: info@behcetsdisease.org.uk
Web: www.behcets.org.uk

BHD (Birt-Hogg-Dubé) Foundation

BHD Foundation,
c/o Myrolytis Trust,
26 Cadogan Square,
London.
SW1X 0JP.

Tel: 020 7052 0088
Email: contact@bhdsyndrome.org
Web: www.BHDSyndrome.org

British Association of Skin Camouflage

The British Association of Skin Camouflage
PO Box 3671, Chester
Cheshire, CH1 9QH

Tel: (01254) 703 107
Email: info@skin-camouflage.net
Web: www.skin-camouflage.net

BRITISH ASSOCIATION OF SKIN CAMOUFLAGE
The Executive Secretary
British Association of Skin Camouflage,
PO Box 202,
Macclesfield SK11 6FP
Tel: 01625 267 880
British Porphyria Association
136 Devonshire Road
Durham City
DH1 2BL
Helpline: 01474 369 231
Email: helpline@porphyria.org.uk
Web: www.porphyria.org.uk

Bullous Pemphigoid
Mrs E Evendon
17 Barley Mount
Redhills
Exeter
EX4 1RP
Tel: (01392) 431 362

Caring Matters Now (CMN)
The Congenital Melanocytic Naevus Support Group
Jodi Unsworth
Caring Matters Now
Bridge Chapel Centre
Heath Road
Liverpool L19 4XR
Tel: (0151) 281 9716
Fax: (0151) 281 9717
Web: www.caringmattersnow.co.uk
Helpline: 0845 458 1023

Changing Faces
Mr James Partridge, Executive Director
Changing Faces
Changing Faces Centre
33-37 University Street
London WC1E 6JN
Tel: (0845) 4500 275
Fax: (0845) 4500 276
Email: info@changingfaces.org.uk
Web: www.changingfaces.org.uk

THE CONGENITAL MELANOCYTIC NAEVUS SUPPORT GROUP
Jody Unsworth,
Caring Matters Now
Bridge Chapel Centre,
Heath Road,
Liverpool L19 4XR
Cutaneous Lymphoma Foundation (Formerly Mycosis Fungoides Foundation)

Darier's Disease Support Group
Mrs J Davies
Darier's Disease Support Group
19 St Annes Road
Hakin
Milford Haven
Pembrokeshire
SA73 3LQ
Tel: (01646) 695055
Web: www.dariers.co.nr

DEBRA
Ms Claire Mather
DEBRA
Debra House
13 Wellington Business Park
Dukes Ride, Crowthorne
Berkshire RG45 6LS
Tel: (01344) 771961
Fax: (01344) 762661
Email: debra@debra.org.uk
Web: www.debra.org.uk

Ectodermal Dysplasia Society
Mrs Diana Perry
Ectodermal Dysplasia Society
Unit 1 Maida Vale Business Centre
Leckhampton
Cheltenham
Gloucester
GL53 7ER
Tel: (01242) 261332
Fax: (01242) 261332
Email: diana@ectodermaldysplasia.org
Web: www.ectodermaldysplasia.org

Eczema Outreach (Scotland)

Contact
Magali Speight (Project Manager)
273 High Street
Ehlers-Danlos Support Group
Ms Lara Bloom
Ehlers-Danlos Support Group
P O Box 337
Aldershot
Hampshire GU12 6WZ
Tel: (01252) 690940
Email: info@ehlers-danlos.org
Web: www.ehlers-danlos.org

Gorlin Syndrome Group
Margaret Costello
Gorlin Syndrome Group
11 Blackberry Way
Penwortham
Preston
PR1 9LQ
Tel: 01772 496849
Email: info@gorlingroup.org
Web: www.gorlingroup.org

Hailey-Hailey Disease Society
www.haileyhailey.com
This is an American site which is useful for patients.

HAIRLINE INTERNATIONAL
The Alopecia Patient's Society
Lyons Court,
1668 High Street,
Knowle,
West Midlands B93 0LY
Tel: 01564 775 281

Hereditary Leiomyomatosis and Renal Cell Cancer (HLRCC) Family Alliance
c/o VHL Family Alliance
Graham Lovitt
The Sycamores
19, Stoneleigh Drive
The Herpes Viruses Association
Miss Marian Nicholson
Herpes Viruses Association
41 North Road
London N7 9DP
Tel: (020) 7607 9661 (for professional calls)
Helpline: 0845 123 2305 (for advice and information to public)
Fax: On request
Web: www.herpes.org.uk

THE HERPES VIRUS ASSOCIATION
Marion Nicholson
The Herpes Virus Association
41 North Road,
London N7 9DP
Tel: 020 7609 9061

The Hidradenitis Suppurativa Trust
British Association for Hidradenitis Suppurativa
PO Box 550
Chatham
ME4 9AH

E-Mail: enquiries@hstrust.org
Website: www.ba-hs.org.uk

Hyperhidrosis Support Group
Mrs Julie Halford
Website: www.hyperhidrosisuk.org
email: info@hyperhidrosisuk.org

Ichthyosis Support Group

P. O. Box 1404
Latex Allergy Support Group

Ms Aleks Kinay
The Latex Allergy Support Group
P O Box 27
Filey
YO14 9YH

Email: info@lasg.org.uk
Web: www.lasg.org.uk

LATEX ALLERGY SUPPORT GROUP
The Secretary
Latex Allergy Support Group,
PO Box 27,
Filey Y0149YH
Tel: 01723 890 001

LEPRA Health in Action

Mr T Vasey
LEPRA Health in Action
28 Middleborough
Colchester
CO1 1TG
Essex

Tel: (01206) 216700
Fax: (01206) 762151
Email: lepra@lepra.org.uk
Web: www.lepra.org.uk

Let's Face It

Mrs C Piff
Let's Face It
72 Victoria Avenue
Westgate on Sea
Kent CT88BH

Tel: (01843) 833724
Fax: (01843) 835695
Hours: 9am - 9pm Seven days a week
Email: julialetsfaceit@aol.com
Email: chrisletsfaceit@aol.com
Web: www.lets-face-it.org.uk
London Vulval Pain Support Group
Tel: 07837 533 992 (Please do not leave a message - try again later)
E-mail: londonvps@yahoo.co.uk
Web: www.vul-pain.dircon.co.uk/london

LUPUS UK
LUPUS UK
St James House
Eastern Road
Romford
Essex
RM1 3NH
Tel: (01708) 731251
Email: headoffice@lupusuk.org.uk
Web: www.lupusuk.org.uk

Lymphoedema Support Network
Mrs B Finch
St Luke’s Crypt
Sydney Street
London
SW3 6NH
Tel: 020 7351 0990
Fax: 020 7349 9809
Web: www.lymphoedema.org

Lymphoma Association (LA)
Mrs Ros Redding
Lymphoma Association
PO Box 386
Aylesbury HP20 2GA
Bucks
Helpline: (0808) 808 5555 Mon - Fri 9 am - 5 pm
Office: (01296) 619400 Mon - Fri 9 am - 5 pm
Fax: (01296) 619414
Web: www.lymphomas.org.uk

Marfan Association UK
Mrs Diane L Rust
Chairman/Support Co-ordinator
Marfan Association UK
Rochester House
5 Aldershot Road
Fleet
Hampshire GU51 3NG
Melanoma Action and Support Scotland
Leigh Smith
c/o LTCAS
349 Bath Street
Glasgow
G2 4AA
Tel (mob): 0773 823 1260
Email: leigh@masscot.org.uk
Web: www.masscot.org.uk

Mastocytosis Support Group
The UK Mastocytosis Support Group
Group Leader Irene Wilson
E-mail: winegums@blueyonder.co.uk
Website: www.ukmasto.co.uk

Myositis Support Group
Irene Oakley
Dermatomyositis and Polymositis Support Group
146 Newtown Road
Woolston
Southampton
Hampshire SO19 9HR
Tel: (023) 8044 9708
Fax: (023) 8039 6402
Email: info@myositis.org.uk
Web: www.myositis.org.uk

National Eczema Society
National Eczema Society
Hill House
Highgate Hill
London N19 5NA
Tel: (020) 7281 3553
Fax: (020) 7281 6395
Helpline: 0800 089 1122
Email: helpline@eczema.org
Email: info@eczema.org
Web: www.eczema.org
The Neuro Foundation
Mrs Roberta Tweedy
Chief Executive
The Neurofibromatosis Association
Quayside House
38 High Street
Kingston upon Thames
Surrey KT1 1HL
Tel: (020) 8439 1234
Fax: (020) 8439 1200
Email: info@nfauk.org
Web: www.nfauk.org

Nodular Prurigo International
Ms Rebecca Dittman
Nodular Prurigo UK
136 Bedford Street South
Liverpool
L7 7DB
Tel: 0151 709 1432
Email: info@nodular-prurigo.org.uk
Web: www.nodular-prurigo.org.uk

Pachyonychia Congenita
Ms Siri Lowe
The Pemphigus Vulgaris Network
Flat 26 C
St Germans Road
London SE23 1RJ
Tel: (020) 8690 6462
Website: www.pemphigus.org.uk

The Pseudoxanthoma Elasticum (PXE) Support Group
Miss Elspeth M W Lax, or
Wg Cdr Bernard Lax MBE
15 Mead Close
Marlow
Bucks SL7 1HR
The Psoriasis Association
Helen McAteer
Chief Executive
The Psoriasis Association
Dick Coles House
2 Queensbridge
Northampton
NN4 7BF
Tel: (01604) 251620
Fax: (01604) 251621
Helpline 08456 760076
Email: mail@psoriasis-association.org.uk
Web: www.psoriasis-association.org.uk

THE PSORIASIS ASSOCIATION
7 Milton Street,
Northampton NN37JG
Tel: 01604 711 129
Mail@psoriasis.demon.co.uk

Psoriasis and Psoriatic Arthritis Alliance
Mr David Chandler/Mrs Julie Chandler
PAPAA
PO Box 111
St Albans
Herts AL2 3JQ
Tel: 01923 672 837
Fax:01923 682 606
Email: info@papaa.org
Web: www.papaa.org

Psoriasis Scotland Arthritis Link Volunteers (PSALV)
Janice Johnson
PSALV
54 Bellevue Road
Edinburgh
EH7 4DE
Tel: (0131) 556 4117
Email: janice.johnson5@btinternet.com
Web: www.psoriasisscotland.org.uk
Raynaud's & Scleroderma Association Trust
Mrs Anne Mawdsley MBE
Chief Executive
Raynaud's & Scleroderma Association Trust
112 Crewe Road
Alsager
Cheshire ST7 2JA
Tel: (01270) 872776
Fax: (01270) 883556
Email: info@raynauds.org.uk
Web: www.raynauds.org.uk

Opening hours: 0900 - 1700 Monday to Friday inclusive.
Outside these hours an answering machine is in operation
Free phone: (0800) 917 2494

THE RAYNAUD'S AND SCLERODERMA ASSOCIATION TRUST
112 Crewe Road,
Alsager ST7 2JA
Tel: 01270 872 776
www.raynauds.demon.co.uk

The Scleroderma Society
Mike Richard
CEO
The Scleroderma Society
Bride House
18-20 Bride Street
London
EC4y 8EE
Tel: 0207 7000 1925
Email: info@sclerodermasociety.co.uk
Web: www.sclerodermasociety.co.uk

Shingles Support Society
Mr Nigel Scott
Shingles Support Society
41 North Road
London N7 9DP
Tel: (020) 7607 9661 (Office)
Tel: (Advice) 0845 123 2305
Web: www.shinglessupport.org

SHINGLES SUPPORT SOCIETY
41 North Road,
London N7 9DP
Tel: 020 7607 9661
Skcin
The Karen Clifford Skin Cancer Charity
P O Box 9629
West Bridgeford
Nottingham
NG2 9GY
General enquiries: alannah.beardsmore@skcin.org
Charity Registration No. 1116440

Skin Camouflage Network
SCN
PO Box 276
Newcastle Upon Tyne
NE3 4RZ
Helpline tel: 07851 073 795
Email: enquiries@skincamouflagenetwork.org.uk
Web: www.skincamouflagenetwork.co.uk

The Sturge Weber Foundation UK
Mrs. Jenny Denham,
Burleigh,
348 Pinhoe Road,
Exeter,
Devon, EX4 8AF

Telephone :- 01392 464675
Fax :- 01392 464675
E mail :-support@sturgeweber.org.uk
Web :- www.sturgeweber.org.uk

Telangiectasia Self Help Group
Mrs D M Lawson
Co-ordinator/Organiser
Telangiectasia Self-Help Group
39 Sunny Croft
Downley
High Wycombe HP13 5UQ

Tel: (01494) 528047
Fax None
Email: info@telangiectasia.co.uk
Web: www.telangiectasia.co.uk
Terrence Higgins Trust
Terrence Higgins Trust
314-320 Gray's Inn Road
London WC1X 8DP

Tel: (020) 7812 1600
Direct Line: (0845) 122 1200 (Mon - Fri, 11 - 8)
Helpline: (020) 7242 1010 (12 noon - 10 pm daily)
Fax: (020) 7812 1601
Email: info@tht.org.uk
Web: www.tht.org.uk

Tuberous Sclerosis Association
Mr Chris Johnson
Support Services Coordinator
PO Box 8001
Derby
DE1 0YA
Tel/fax: 01332 290734
Email: development-support@tuberous-sclerosis.org
Web: www.tuberous-sclerosis.org

UK Lichen Planus
Web: www.uklp.org.uk
Email: admin@uklp.org.uk
Founder member; Mrs Bridie Nelson

The Vitiligo Society
Jennifer Viles
Manager
The Vitiligo Society
125 Kennington Road
London SE11 6SF

Tel: (020) 7840 0855
Freephone: (0800) 018 2631
Fax: (020) 7840 0866
Email: Ken125@vitiligosociety.org.uk
Web: www.vitiligosociety.org.uk

VITILIGO SOCIETY
125 Kennington Road,
London SE 11 6SF
Tel: 020 7840 0855
www.vitiligosociety.org.uk
The following is an alphabetical list of local and regional patient support groups for those suffering from skin disease. They are often run by volunteers who suffer from the disease themselves, and many patients find it enormously valuable to have such contact with others both for support and for practical help.

**Eczema Outreach (Scotland)**
Eczema Outreach (Scotland)
273 High Street
Linlithgow
EH49 7EP
Registered Charity SC042392
Tel: 01506 840 395
Free line: 0800 622 6018
Mob / Text: 07 8070 4 8070
Email: info@eczemaoutreachscotland.org.uk
Web: www.eczemaoutreachscotland.org.uk

**Nottingham Support Group for Carers of Children with Eczema**
Amanda Roberts
18 Marlborough Road
Nottingham
NG5 4FG
Tel: 0115 926 9996
Email: enquiry@nottinghameczema.org.uk
Website: www.nottinghameczema.org.uk

**Skin Conditions Campaign Scotland**
c/o LTCAS
Venlaw Building
349 Bath Street
Glasgow
G2 4AA
Tel: 07852299206
Email: Melissa.sccs@gmail.com

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**Skin Care Cymru**
Skin Care Cymru
PO Box 612
Swansea
SA1 9GH
Tel: 07917 572 895
Email: info@skincarecymru.org
Web: www.skincarecymru.org
Registered Charity No. 1131832

**Wessex Cancer Trust - SCIN (Skin Cancer Information Network) MARC'S LINE**
(Melanoma and Related Cancers of the Skin)

Jo Allum / Linda Burt
Clinical Nurses Specialists in Skin Cancer Prevention
Marc's Line Resource Centre
Dermatology Treatment Centre
Level 3
Salisbury District Hospital
Salisbury
Wiltshire SP2 8BJ
Tel: (01722) 415071
Fax: (01722) 415071
Web: www.wessexcancer.org
Email: MARCSline@salisbury.nhs.uk
A full list of the above Patient Support Groups giving details about their aims, grants available etc is listed on the BAD website at

http://www.bad.org.uk/site/575/default.aspx