YOUNG ADULTS (20 - 35 YEARS)

A large number of skin diseases reach their peak of expression during the young adult years, and these individuals also represent a large and growing proportion of the South African population, in common with other developing countries.

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Young adults are especially distressed by skin conditions that are uncomfortable, disfiguring or contagious. This group has also been severely affected by HIV/AIDS, with a consequent increase in the frequency and severity of the HIV-associated dermatoses. Some of these conditions have been discussed in other sections of this review.

ACNE KELOIDALIS

Inflammatory papules and pustules located mainly on the nape of the neck and occiput characterise this acne-like condition. It primarily affects black males. The lesions heal with scarring, creating huge keloids if this tendency exists. The cause is unknown, and unlikely to result from repeated shaving or short hair cutting. It may be caused by unusually thick collagen, which traps and disrupts terminal hair follicles. Treatment is not very successful, and large keloids are best excised by a surgeon. Mild cases can be helped by intralesional injection of steroids like Celestone Soluspan into individual papules and scars. Long-term oral tetracyclines, as for acne, can also be beneficial, as can topical antibiotics like erythromycin and clindamycin.

ACNE VULGARIS

There has been a recent tendency for acne to persist beyond the teens, or even occur for the first time in adulthood, called persistent adult acne. Adult acne has the same pathogenesis as ordinary acne, but is often more resistant to treatment; individuals are also less likely to leave the condition untreated. The principles of treatment are as for teenage acne. It is important to exclude drug causes such as lithium, phenytoin and other anticonvulsants, steroids and cyclosporin. Widespread misuse of potent topical steroid creams for acne and other facial conditions will aggravate acne in the long term, and cause other side-effects like atrophy and telangiectasia.

CHLOASMA

This is an acquired tan-brown symmetrical discolouration of facial skin on the forehead, nose, cheeks, upper lip and chin (Fig. 1). It is much more common in women, and is thought to be due to a combination of sun exposure and hormonal changes such as pregnancy and hormonal contraceptives. It is darker and more disfiguring on darker skins. Chloasma is very difficult to treat: sun avoidance and strong sunscreens are important to prevent worsening of the condition. Bleaching agents like 2 - 4% hydroquinone are widely used. There are many proprietary compounds that contain mild bleaches: kojic acid, vitamin C and

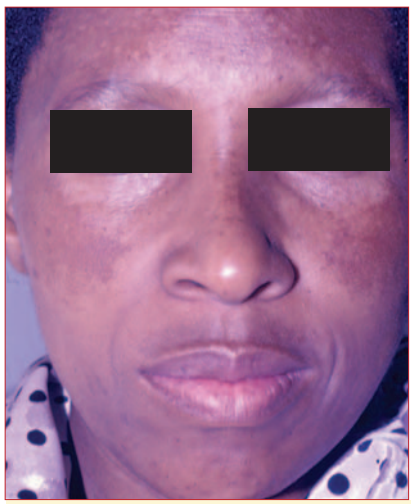

Fig. 1. Chloasma.

liquorice extract. Retinoid creams like tretinoin (Renova, Retin-A), azelaic acid (Skinoren) and glycolic acid products can also be tried. In fair-skinned people, certain light therapy devices can alleviate chloasma.

CONDYLOMATA ACUMINATA

These are genital warts caused particularly by human papillomavirus (HPV), types 6 and 11. They appear initially as small, raised, flesh-coloured or pale papules with an irregular surface, on the skin or mucous membranes of the penis, vulva, vagina, anus and surrounding areas (Fig. 2). Infection can be latent or subclinical, enhancing the risk of unexpected spread. Without prompt treatment, or in the presence of immunosuppression, the lesions can grow to a huge size, obscuring the normal anatomy. There is also increased risk of malignancy, especially carcinoma of the cervix, and squamous carcinoma of the genital skin. Treatment is generally difficult and the principles are the same as in infants (see article on infants, pp.).

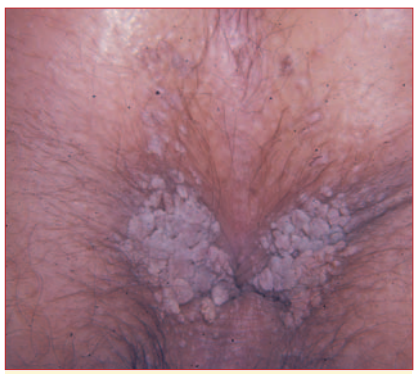

Fig. 2. Condylomata acuminata — perianal.

DISCOID LUPUS ERYTHEMATOSUS (DLE)

This form of lupus erythematosus is not uncommon, mainly affecting women. In most cases the condition occurs independently of systemic lupus erythematosus (SLE), but can occur as part of, or progress to, SLE. Typical lesions start as itchy, painful, reddish, scaly swellings or plaques on sun-exposed areas: the face, lips, chest, upper back and hands. The lesions heal centrally leaving depigmentation (Fig. 3). Plaques on the scalp are associated with scarring alopecia, which is permanent. The diagnosis is normally confirmed on skin biopsy, and SLE is excluded through history and examination and testing for antinuclear antibody (ANA), although this may be positive in low titre with DLE. The condition is more common with HIV. DLE is resistant to therapy, and the resultant scarring is permanent. Mild cases are treated with sun avoidance, sunscreens and potent topical steroids: clobetasol (Dermovate, Dovate) or betamethasone dipropionate (Diprosone, Diprolene). More extensive cases are treated with low to medium doses of systemic steroids: prednisone, together with chloroquine (remember retinal toxicity).

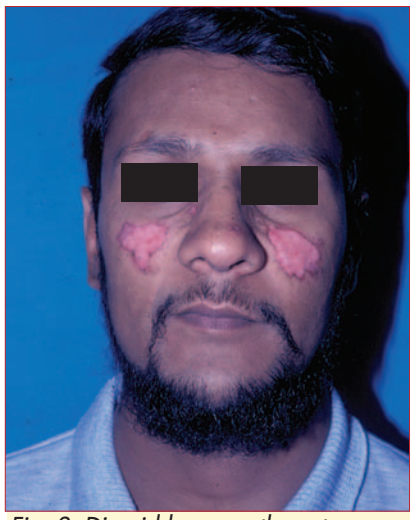

Fig. 3. Discoid lupus erythematosus.

DYSHIDROTIC ECZEMA

This common and potentially severe form of dermatitis affects the hands and feet, but can spread. It starts with intensely itchy small vesicles on the palms and/or soles, and the sides of the fingers or toes (Fig. 4). These enlarge, coalesce and rupture to leave red, fissured, scaly and painful areas. The condition usually occurs intermittently, but can persist. Dyshidrotic eczema can represent hand and foot involvement in atopic dermatitis, when the usual trigger is a contact irritant such as frequent hand washing, soaps, detergents and solvents. It can also occur independently and be due to allergic contact dermatitis, confirmed on patch tests. The condition can also be triggered by psychogenic factors like stress, but this is difficult to prove. Finally dyshidrotic eczema can be an allergic reaction to a distant focus of fungal infection, especially tinea pedis, giving rise to the id reaction. All types tend to be resistant to therapy, and a treatable cause gives the best results. Failing this, milder cases should be treated with a potent topical steroid cream like clobetasol propionate or mometasone, together with a course of oral antibiotics. More severe cases are better treated with a short course of oral steroids, such as prednisone 20 - 60 mg daily for 5 - 10 days.

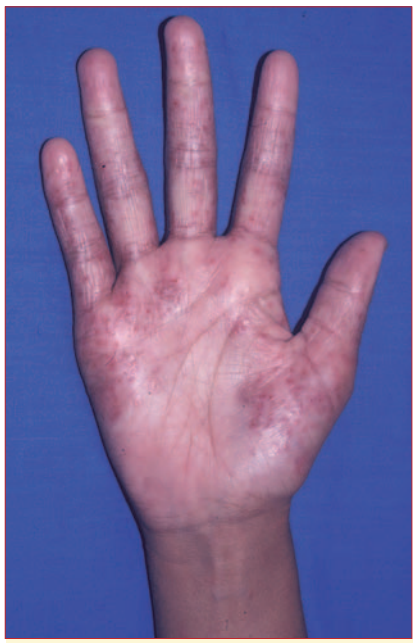

Fig. 4. Dyshidrotic eczema.

DYSPLASTIC NAEVI

These are acquired melanocytic naevi (moles) that are larger than usual and slightly irregular in shape, border and colour, with features suggestive of melanomas (Fig. 5). They increasingly appear from the late teens into the twenties, often with a family history of similar moles. Sun exposure probably plays a major role in converting a benign melanocytic naevus into a dysplastic naevus. Their natural history is not clear, but some may become melanomas, hence the importance attached to these lesions. Some recom-

mend elective removal of all suspected dysplastic naevi, but this is seldom practical. Regular examination is prudent, often by means of epiluminescence microscopy (Molemax machine), to highlight more suspicious lesions, or those which are changing: these are then excised surgically.

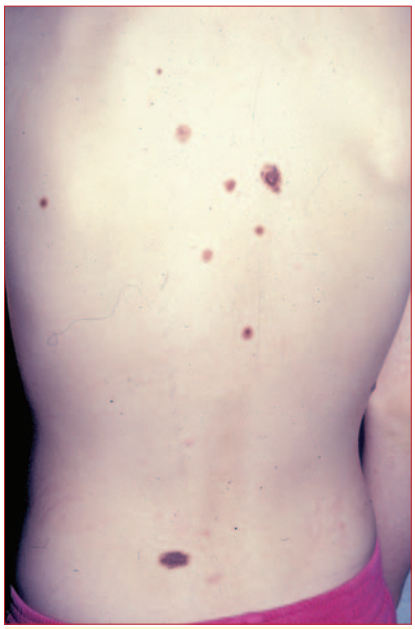

Fig. 5. Dysplastic naevi.

FOLLICULITIS

Folliculitis occurs when small pustules occur within the hair follicles covering most of the skin (vellus hairs). One type of folliculitis is a simple superficial bacterial infection of the hair, mainly with *Staphylococcus aureus*. This is usually mild and self-limiting, and can be triggered by friction, shaving, waxing or scratching. More severe cases are treated with a topical antibiotic like mupirocin (Bactroban cream), fusidic acid (Fucidin cream), erythromycin (Ilotycin, Zineryt) or clindamycin (Dalacin T). Oral antistaphylococcal antibiotics like cloxacillin can also be used. Another type is caused by a commensal fungus, *Malassezia*, and presents as itchy follicular papules and pustules on the upper chest, back and arms (Fig. 6). This is best diagnosed on biopsy, and is treated with antifungal shampoos like ketoconazole (Nizshampoo) or econazole (Pevaryl foaming solution), or with oral antifungals like itraconazole (Sporanox). This fungal folliculitis, also known as pityrosporum folliculitis, can closely resemble acne. Another type of folliculitis occurs exclusively in HIV infection, and is due to eosinophil infiltration of the vellus hair follicle. This so-called eosinophilic folliculitis is exceptionally itchy, and untreatable. Fortunately the condition usually remits spontaneously. Potent topical steroids, antihistamines, antifungals and ultraviolet B phototherapy may help. All types of folliculitis are more common in HIV, and following cancer chemotherapy.

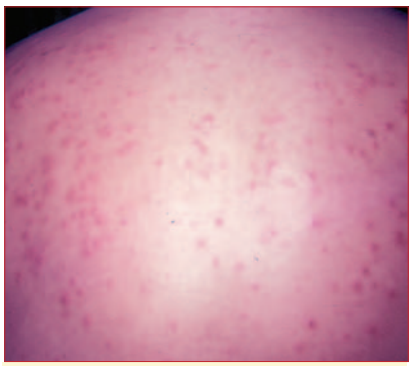

Fig. 6. Pityrosporum (fungal) folliculitis.

HERPES SIMPLEX

Human herpesviruses 1 and 2 cause the common cold sore (recurrent labial herpes) and genital herpes. Frequent recurrences are a hallmark of the condition, often with 2 - 4 attacks per month. Each attack is preceded by mild paraesthesia, followed by the appearance of grouped erythematous papules, vesicles and pustules in the nasolabial or genital region (Fig. 7), which crust and resolve in a few days. Triggers include sun exposure, trauma, fever, menstruation and stress. Antiviral creams like acyclovir (Zovirax, Activir) can be helpful if applied early and often. Antiviral tablets like acyclovir (Zovirax, Lovire and many other generics) 200 mg 5 times daily for 5 days will shorten the length and severity of the attack. Alternatives include valacyclovir (Zelitrex) 500 mg bd for 5 days and famciclovir (Famvir) 125 - 250 mg tds for 5 days. Preventive therapy with long-term oral antivirals can be used if necessary, and will inhibit transmission. Titrate down to the lowest effective dose which suppresses outbreaks: acyclovir 200 - 400 mg bd, valacyclovir 500 mg daily, famciclovir 125 - 250 mg bd. HIV or another cause of immune suppression results in progression to painful, chronic ulcers.

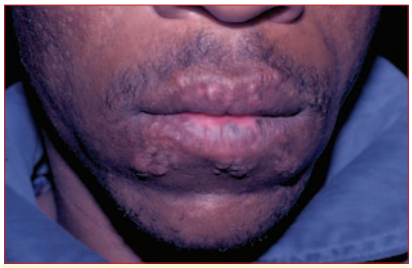

Fig. 7. Herpes labialis.

HYPERHIDROSIS

Hyperhidrosis is excessive sweating, objectively apparent and which interferes with daily activities, usually on the palms, soles and axillae and triggered by heat, stress or anxiety, although there may be no obvious stimulation. Hyperhidrosis can cause loss of confidence, and depression, and should not be trivialised. A simple but effective treatment of axillary hyperhidrosis is aluminium chloride hexahydrate antiperspirant roll-on (Driclor). Irritation and contact dermatitis are possible side-effects. An effective and long-lasting alternative is Botox injection. Palmoplantar hyperhidrosis is treated with either iontophoresis (galvanic current applied to hands and/or feet immersed in water), Botox injection or sympathectomy, generally laparoscopic and successful. However, compensatory hyperhidrosis at distant sites can occur as a complication of sympathectomy.

KAPOSI'S SARCOMA

This vascular neoplasm due to proliferation of abnormal endothelial cells has become distressingly common in young HIV-positive adults, whereas it was previously seen as a disease of the middle-aged, or those on immunosuppressive therapy for renal transplants. It presents insidiously as purplish macules, patches and plaques anywhere on the skin surface, but usually affects the face or lower extremities. These progress into indurated plaques and nodules, and lesions can coalesce into huge fungating masses with associated severe lymphoedema

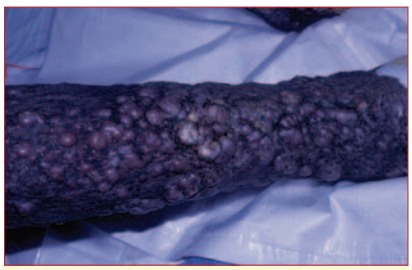

Fig. 8. Kaposi's sarcoma.

(Fig. 8). Lesions are often painful and can involve mucous membranes causing impaired function. Flexion contractures of limbs are frequent. Internal organ involvement is common, even in the absence of skin lesions, and includes lymph nodes, lung, gastrointestinal tract and bone marrow. Organ involvement eventually causes death. Kaposi's sarcoma can occur at any stage of HIV infection, sometimes even in the absence of profound immunosuppression. Skin biopsy confirms the diagnosis, and treatment depends on extent, severity, general condition and symptoms caused by the neoplasm. Localised symptomatic disease responds best to irradiation, and widespread disease, or internal organ involvement, is treated with chemotherapy. Unless the underlying HIV is treated with antiretrovirals the treatment is at best palliative. Kaposi's sarcoma is closely, and perhaps causally, associated with human herpesvirus 8 infection.

KELOID

Keloids are scars that proliferate beyond the original site of injury or burn. These itchy and often painful lesions are commonest in sites exposed to continuous straining forces, such as the upper chest, back and arms. They are also common on the face and earlobes (Fig. 9). There is a strong hereditary and racial predisposition to keloids. Occasionally they occur without any antecedent trauma, particularly on the chest or breasts. Treatment is difficult, and small lesions are generally injected with potent steroids like Celestone Soluspan. Large lesions can be excised, but adjuvant therapy like steroid injection or a short course of radiation is needed to prevent recurrence, which can be worse than the original condition. Silicone sheets (Cica-care) applied for several hours daily can prevent or improve some keloids.

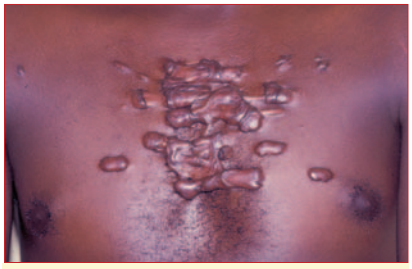

Fig. 9. Keloid — chest.

PERIORAL DERMATITIS

Erythematous papules, tiny pustules and scaling occur around the mouth, spreading to the nasolabial fold, usually in healthy young women, resembling acne and seborrhoeic dermatitis. Cosmetics, toothpaste, steroid creams and emotional stress have all been implicated in the aetiology. Treatment with any oral tetracycline such as tetracycline HCl 500 mg bd, lymecycline (Tetralysal) 300 mg daily, doxycycline 100 mg daily or minocycline 100 mg daily for a few months is effective. Alternatives include topical antibiotics like erythromycin, clindamycin and metronidazole.

PITTED KERATOLYSIS

Pitted keratolysis is a bacterial infection of the weight-bearing areas of the soles. Predisposing factors are male sex, occlusive footware, excessive sweating and frequent contact with water. Shallow pits occur on a macerated background; a foul odour is apparent (Fig. 10). Usually asymptomatic, the condition can coexist with tinea pedis. The major organism implicated is a species of *Corynebacterium*. Treatment includes attention to predisposing factors, together with oral or topical antibiotics like erythromycin, clindamycin or fusidic acid. Imidazole antifungal creams are also effective because of their Gram-positive antibacterial action.

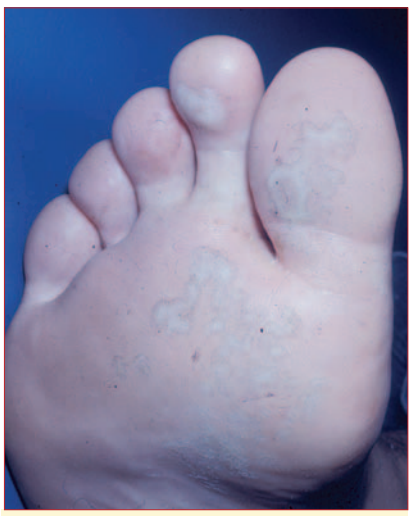

Fig. 10. Pitted keratolysis.

PITYRIASIS ROSEA

Pityriasis rosea is a self-limiting papulosquamous eruption of presumed viral origin, primarily affecting young adults. Commoner at season change, the rash starts with an annular, scaly, erythematous patch resembling ringworm, usually on the trunk. Several weeks later a disseminated eruption occurs, consisting of small pinkish macules and papules that form oval scaling patches. These patches lie parallel to the skin creases, mainly on the trunk and proximal extremities, in the so-called fir tree distribution (Fig. 11).

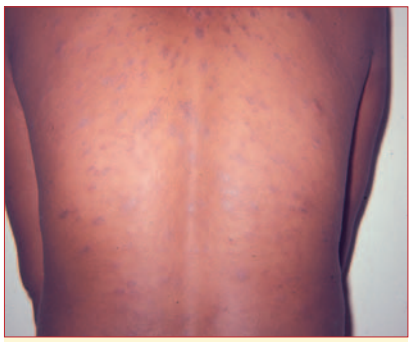

Fig. 11. Pityriasis rosea.

The whole eruption, which may be pruritic, lasts about 2 months. Postinflammatory pigmentation can occur in darker skinned individuals. Pityriasis rosea might be caused by human herpesvirus 7. No specific therapy is required, but topical steroid creams can help the itching, and sun exposure is said to hasten resolution.

PITYRIASIS VERSICOLOR

This superficial fungal infection is caused by the commensal yeast *Malassezia furfur*. The condition is

characterised by fine scaling brownish and whitish macules that coalesce into large patches, affecting mainly the upper trunk and neck (Fig.12). Mild itching may be present. Tanning of the infected skin is inhibited, which may take months to repigment, even after treatment. Its other name is 'beach fungus' since it is often noticed after tanning.

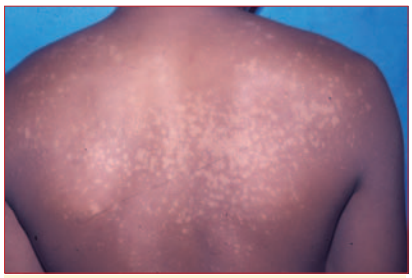

Fig. 12. Pityriasis versicolor — whitish patches.

A variety of treatments are successful, but the condition tends to recur. Antifungal shampoos like Nizshampoo and Pevaryl foaming solution are applied to the whole upper trunk and scalp. They can be left on overnight or rinsed off after 10 minutes, depending on the severity of the infection. The treatment is repeated a few times. Antifungal sprays like terbinafine (Lamisil spray) or imidazole creams can also be used. Oral treatment with itraconazole (Sporanox) 200 mg daily for 1 week or ketoconazole (Nizoral, Ketazol) 200 mg daily for 10 days is simple and effective. Older treatments include 2% sulphur in aqueous cream or selenium sulphide shampoo (Selsun). Selsun is particularly useful as a prophylactic regimen in cases of frequent recurrence: the scalp and upper trunk are treated once or twice monthly by applying the foamed shampoo for 10 minutes before rinsing.

POLYMORPHIC LIGHT ERUPTION (PLE)

Polymorphic light eruption is the commonest idiopathic photodermatosis worldwide, with a prevalence estimated at 10 - 20%. Usually affecting women, there is often a family history. Itchy, erythematous papules and vesicles occur in certain sun-exposed sites a few days after sun exposure. Relatively intense UV exposure is usually required to evoke PLE, e.g. during a beach holiday. Avoidance of further sun leads to spontaneous healing without scarring. Chronically sun-exposed parts are seldom affected by PLE, which targets mainly the chest, back and forearms. An important differential diagnosis is lupus erythematosus. PLE can be prevented with very strong, broad-spectrum sunscreens, and individual lesions are improved with potent topical steroid creams. The condition tends to improve with age.

PORPHYRIA CUTANEA TARDA

This type of porphyria has become predominant in South Africa, because it is associated with HIV infection. Some cases are inherited, but most are acquired and due to liver dysfunction associated with either iron overload, hepatitis C infection, alcoholic liver disease, SLE or HIV. There is an enzyme defect in the hepatic haem pathway, and porphyrins accumulate in the skin, giving rise to reactive oxygen molecules on exposure to light. Sun-exposed areas, the face and dorsal surface of the hands, are damaged, causing fragility of skin with blisters, scars and milia. Diffuse hyperpigmentation and hypertrichosis (excessive facial hair) occur and the skin has a weather-beaten look (Fig. 13).

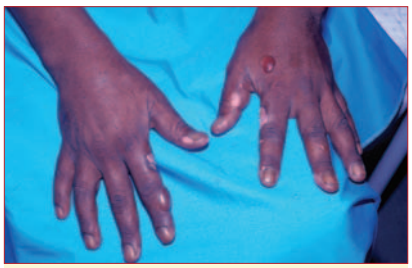

Fig. 13. Porphyria cutanea tarda.

The diagnosis is confirmed by high porphyrin levels in urine. Unless the original liver disease is treatable, the condition tends to be persistent. Avoiding sun and mechanical trauma to the skin are important. Multivitamins and chloroquine have been beneficial in some cases.

PSEUDOFOLLICULITIS

Close shaving of coarse, curly hair causes the hair to bend and penetrate the skin as it grows, causing a foreign body-type reaction with inflamed papules and pustules characteristic of pseudofolliculitis. The condition closely mimics staphylococcal folliculitis, but is not an infection. The most common site is the beard, but other regions with coarse terminal hair can be affected. Pseudofolliculitis occurs primarily in black men. The lesions resolve with often disfiguring postinflammatory pigmentation. Treatment is aimed at avoiding close shaves, using older types of single-blade razors rather than newer multiblade devices. Electric razors are sometimes preferred. Ingrowing portions of hairs should be gently extracted with a needle, and the hair cut with scissors. Acne-type therapy with benzoyl peroxide and oral tetracyclines may give some relief, and 1% hydrocortisone cream can be used as well. If possible shaving should be avoided, but this is often difficult. Definitive therapy is laser epilation.

PYOGENIC GRANULOMA

These benign tumours composed of granulation tissue usually follow a minor injury, and occur especially on the lips, fingers and face. A rapidly growing exudative, exophytic, reddish tumour occurs, which bleeds on the slightest rubbing (Fig.14). The lesions do not resolve spontaneously, and should be removed by curettage and electrodesiccation, or by surgical excision. Histology should always be obtained, as amelanotic melanoma and Kaposi's sarcoma can present in a similar way.

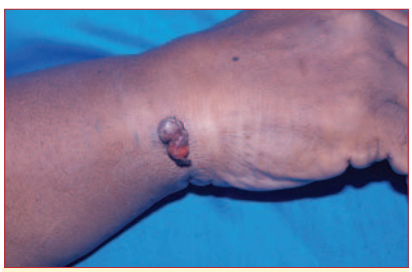

Fig. 14. Pyogenic granuloma.

PYODERMA GANGRENOSUM

Pyoderma gangrenosum is a severe disease where large painful ulcers occur spontaneously at any site on the skin, usually on the leg, starting as pustules which ulcerate and extend peripherally. The edge is violaceous and undermined (Fig.15). The ulcers do heal slowly, but new ones occur. This is a clinical diagnosis, as histological findings are nonspecific; however, other causes of cutaneous ulceration need to be excluded.
Approximately 50% of cases are associated with an underlying disease, particularly inflammatory bowel disease, rheumatoid arthritis and lymphoproliferative disorders. Treatment is difficult, and consists largely of high-dose oral corticosteroids. Other agents used include cyclosporin and thalidomide.

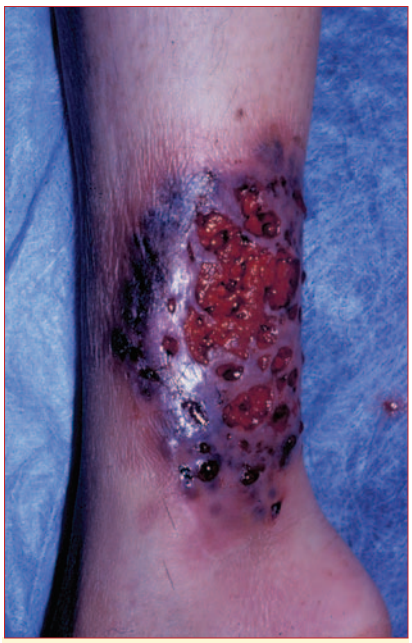

Fig. 15. Pyoderma gangrenosum.

SEBORRHOEIC DERMATITIS

This is one of the commonest forms of dermatitis worldwide, and in its mildest forms includes all cases of itchy dandruff. Men are more often affected than women. Sites of predilection are those rich in sebaceous glands, such as the scalp, face, upper chest and back. On a greasy background, erythema and yellowish scales occur. Itching is mild or not present. The eyebrows, glabellar, nasolabial, paranasal and perioral regions of the face are mostly affected. The eruption can be acute, weeping and secondarily infected on occasions, especially on the scalp. Seborrhoeic dermatitis is a chronic disease that remits and relapses throughout the adult years; it is exacerbated by cold weather, stress and intercurrent illness. In the setting of HIV infection seborrhoeic dermatitis is much more common, and also more severe. Mild cases are treated with mild topical steroid creams like 1% hydrocortisone (Procutan), or slightly stronger agents like hydrocortisone butyrate (Locoid), clobetasol butyrate (Eumovate), methylprednisolone aceponate (Advantan) or diluted betamethasone valerate (Betnovate, Persivate, Lenovate). Signs of dermatitis disappear rapidly, but recur when the cream is stopped. Adjuvant therapy with a non-irritating cleanser to remove excessive oil from the face is helpful. Alternatives to steroid creams include sulphur preparations and antifungal creams like the imidazoles (ketoconazole, econazole, clotrimazole). Weeping dermatitis is treated with oral antibiotics and a short course of oral steroids. Scalp involvement is treated in mild cases with any of the proprietary antidandruff shampoos, including tar, selenium, zinc pyrithione and antifungal shampoos. More severe scalp disease warrants a steroid scalp application such as Locoid crelo, Elocon lotion, Advantan scalp solution or Synalar gel.

SECONDARY SYPHILIS

Secondary syphilis occurs 2 - 6 months after the primary chancre without treatment, resulting from haematogenous dissemination of *Treponema pallidum*. Associated HIV infection accelerates this progression. Skin manifestations are protean, and occur in 80% of cases. Widely distributed brownish red scaling papules occur, also involving the palms and soles (Fig. 16). Later more isolated round, dark lesions with raised edges occur at certain sites, especially around the mouth. Moist, wart-like plaques occur on the genitalia — condylomata lata.

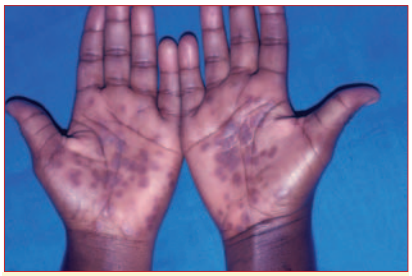

Fig. 16. Secondary syphilis.

Secondary syphilis can mimic guttate psoriasis, pityriasis rosea and lichen planus. Any suspicion should be confirmed with serological testing for syphilis, which will invariably be positive in secondary syphilis. Treatment consists of benzathine penicillin 2.4 million units IMI weekly x 3.

SYRINGOMA

These small, flesh-coloured papules occur around the eyes, and are of cosmetic importance only (Fig. 17). They may be confused with xanthelasmata, which are a yellowish colour.

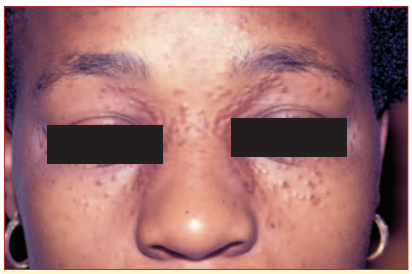

Fig. 17. Syringomata.

Reassurance is the best advice, but the lesions can be excised if desired.

TINEA PEDIS

Tinea pedis or athlete's foot occurs between the toes or on the soles of the feet. It is caused mainly by *Trichophyton rubrum* and *Trichophyton mentagrophytes*. The condition presents as itchy or painful scaling, maceration and fissuring in the toe webs, especially the 4th webspaces.
Infection can spread to the soles and sides of the feet as a red, scaly eruption. Another type of tinea pedis causes small, inflamed blisters on the soles or between the toes (Fig.18). Tinea pedis must be differentiated from macerated soft corns, candidiasis and

dyshidrotic foot eczema. Diagnosis is confirmed through mycological examination of foot scrapings. Tinea pedis responds well to a variety of treatments, but is often recurrent. Risk factors include shared ablutions, use of communal swimming pools, occlusive footware, humid environments and coexistent conditions like varicose veins or diabetes. Terbinafine cream (Lamisil) is a popular treatment, applied once or twice daily for a few weeks. Any of the imidazole antifungal creams like ketoconazole (Nizcreme), econazole (Pevaryl) or clotrimazole (Canesten) can also be used. More severe cases are treated with oral antifungals like ketoconazole (Nizoral, Ketazol), fluconazole (Diflucan, Fluzol, Flucoric), itraconazole (Sporanox) or terbinafine (Lamisil). Griseofulvin is very effective, but high doses are needed. Severe, neglected tinea pedis can become secondarily infected with mixed organisms, and this responds best to regular soaking of the feet in diluted potassium permanganate solution.

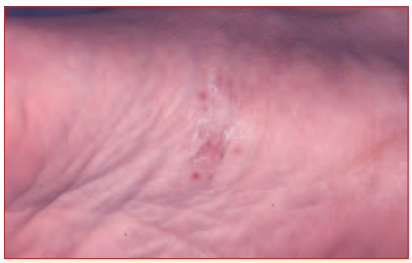

Fig. 18. Vesicular tinea pedis.

URTICARIA

Urticaria is characterised by transient, itchy weals or hives that tend to last less than 24 hours. Mucous membrane involvement, angioedema, can be potentially dangerous. Weals can occur anywhere and are often of bizarre or annular shape, healing with no secondary changes. Acute urticaria is usually caused by allergies to food, medicine or insect stings, and is best managed in medical emergency units because of the risk of laryngeal angioedema or anaphylaxis. Bacterial infections like streptococcal pharyngitis can also cause acute urticaria, especially in children. This responds well to antibiotics. Urticaria which becomes recurrent over many weeks and months is termed chronic urticaria. The causes are usually different from those of acute urticaria, and in most cases remain unknown. Presumed causes include chronic bacterial infection, especially of the teeth, tonsils, throat or sinuses, recurrent urinary tract infections, hepatitis B and HIV, scratching (dermographism) (Fig. 19), pressure, water, cold, sweating or sun exposure, bilharzia and intestinal worms, autoimmune diseases, haematological malignancy or lymphoma, drug and food allergies and emotional stress. It is important to note that most cases of chronic urticaria are not allergic. If a cause is found and treated, the urticaria may disappear. Symptomatic treatment is still required.

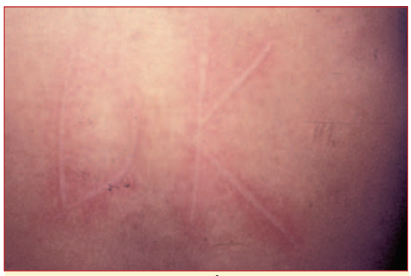

Fig. 19. Dermographism.

If no cause is forthcoming the condition is labelled chronic idiopathic urticaria, and treatment is purely symptomatic and suppressive. Antihistamines remain the mainstay of therapy, and a trial-and-error approach is recommended until the right agent is found. Both older, sedating and newer non-sedating agents can be tried, sometimes in combination. Look out for interactions between antihistamines and other drugs. Oral corticosteroids are also effective, and can be used for short-term control. Unresponsive chronic idiopathic urticaria can be treated with ketotifen, cyclosporin or psoralen plus ultraviolet A phototherapy (PUVA).