Infestations
HIV patients can tolerate heavy burdens of the sarcoptes mite without producing the usual intense pruritis giving rise to disseminated crusted or Norwegian scabies. Infested patients are highly infectious owing to the heavy numbers of mites.

Non-infectious cutaneous manifestations of HIV disease
At least a third of our patients develop seborrhoeic dermatitis. This occurs in the usual distribution, but often with a prominent perifollicular component and extending on to the flanks and thighs. Scrapings may be positive for *Pityrosporum orbiculare*. Topical imidazoles are helpful in the management of early cases, but later more potent steroids are needed. Patients may also have itchy follicular lesions on the face and trunk—sometimes called eosinophilic folliculitis or pruritic papular eruption of AIDS. An unusual response to intrafollicular organisms is probable but treatment aimed at these is unfruitful. Potent topical corticosteroids or UVB treatment is often required because of intense pruritis.

The behaviour of psoriasis in HIV patients challenges our efforts to understand this enigmatic condition. Prevalence is probably not increased, but the condition is more severe. Reiter's-like psoriasis with rupioid lesions and keratoderma blenorrhagica are common. Lesions can progress alarmingly and are often accompanied by severe arthritis. Cytotoxic treatment is hazardous but Acetretin and Zidovudine can be very helpful.

An acquired icthyosis is common as the disease advances which may signify malabsorption of essential nutrients.

Drug rashes are much more common, for example with Co-trimoxazole, Dapsone and Fucidic Acid. Zidovudine can cause longitudinal sub-ungual black, streaks, particularly in coloured patients.

Auto-immune disease
Thrombocytopenia with or without purpura is common. Alopecia areata and totalis, morphoea, lichen planus and granuloma annulare have been reported.

Cutaneous malignancy and HIV disease
Cutaneous lymphoma has been described and is likely to be related to decreased immunity. Malignant melanoma and squamous carcinoma can progress alarmingly. Oral and ano-rectal squamous carcinomas may ensue.

Summary
The dermatologist is in a unique position to play a life-saving role in the early diagnosis and care of HIV patients and can significantly contribute to improving the quality of their lives. Early biopsy with culture for organisms is the key and should precede a course of treatment, rather than be resorted to if it fails. Surprises and atypical presentations are the rule. The exaggerated severity of the common non-infectious skin conditions remain a challenge in this vulnerable group of patients.

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LESSONS FROM A SYMPOSIUM ON DERMATOLOGY HELD IN THE COLLEGE ON 4 MAY 1994*

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In the UK 10 per cent of those who go to their family doctors do so with skin problems. Despite recent advances and improvements the specialty of dermatology continues to challenge students and doctors at all levels of practice and training. The symposium provides a comprehensive overview of dermatology today, from guidance on when to consult a dermatologist, through to an update of the contribution of molecular science to the discipline.

Skin clues to systemic disease
Many systemic diseases first declare themselves in the skin and physicians need to be alert to these clues. All of the largest organs in the body should be inspected in the pursuit of 'Sherlockian dermatology'. Pruritus as a symptom may have its primary cause in the skin (eczema, dermatitis, scabies, urticaria, dermatitis herpetiformis, lichen planus, psoriasis). However, generalised itch and shiny nails suggest it is genuinely systemic and may be due to internal diseases including chronic renal failure, obstructive liver disease, lymphoma, polycythaemia rubra vera (in which the itch is often worse after bathing), drug sensitivity, thyroid disease, diabetes mellitus and psychiatric diseases.

Hair and nail clues
In man, hair and nails are relatively vestigial differentiated epidermal structures, yet can provide a plethora of clues to the keen observer.

(a) The yellow nail syndrome, characterised by slow growth, over-curvature, yellow colour, onycholysis and shedding, may have associations with bronchiectasis, serous cavity effusions, lymphoedema, sinusitis, rheumatoid arthritis, thyroid disease and neoplasia.

(b) Beau's lines, which are transverse single depressed lines across the nail, may occur with any acute illness including psychiatric disorders. The equivalent may also be seen in the hair as a narrowing of the hair shaft.

(c) Silent haematomas from bleeding diatheses may manifest as splinter haemorrhages. Trauma is the most likely aetiological factor if they occur at the separation of the nail plate and fold, but if occurring more proximally, arterial emboli, vasculitis, septicaemia and scurvy should be considered.

(d) Onycholysis may be associated with trauma, dermatological (psoriasis, onychomycosis) or systemic disease, particularly thyroid dysregulation.

(e) Hairloss can prove devastating to the patient. In telogen effluvium the hair is lost in the resting phase and represents exaggeration of normal shedding. This forms the basis of most causes of diffuse alopecia. It may be associated with iron

*A list of speakers and the titles of their papers presented at this symposium is recorded in *Proceedings* Vol 24, p 475.

and zinc deficiency, hypopituitarism, thyroid dysregulation, post partum and post menopausal hairloss. Cytotoxic drugs act in the growing phase of the hair cycle to cause hairloss (anagen effluvium). A more specific pattern of hairloss is seen in alopecia areata with exclamation mark hairs in the active phase. Auto-immune disorders especially hypothyroidism, should be considered. Hair excess can be equally as distressing as hairloss and hyperandrogenism should be excluded. Hypertrichosis should prompt a search for occult malignancy, particularly lymphoma and upper GI neoplasia because if the tumour can be treated, the hair will regress.

Skin and the psyche
See pp 29–33.

Drug eruptions: their mechanisms and manifestations
Adverse drug reactions are an 'inevitable' consequence of the benefits of modern drug therapy. Despite extensive *in vitro* and animal testing, serious reactions of low incidence may not be suspected until a large number of patients have been treated with a new drug. Drug eruptions represent 2·2 per cent of cases of drug sensitivity. Frequently implicated drugs include antimicrobial agents (42 per cent), antipyretic/anti-inflammatory analgesics (27 per cent), with drugs acting on the CNS accounting for 10 per cent of reactions. Increasing polypharmacy, particularly in the elderly, compounds the problem. Drug reactions may arise as a result of immunological allergy or more frequently by non-immunological mechanisms. They may be predictable (Type A), usually dose related, or unpredictable (Type B), dose dependent. The skin has a limited number of responses to a wide variety of insults and it is therefore impossible to identify an offending drug or the mechanism involved on purely clinical appearances alone. Pharmacogenetics may underlie tolerance and idiosyncrasy, eg aspirin sensitivity is linked with HLA-DQw2; oxidative metabolism of sulphonamides by cytochrome P450 enzymes yields reactive metabolites, which are toxic to lymphocytes. Acetylator status is important—the lupus-like syndrome due to procainamide occurs more often in fast acetylators implying that a conjugate and not the parent compound is responsible. Slow acetylators are more liable to develop adverse reactions to isoniazid (pellegra-like syndrome) and dapsone (haemolysis).

Xanthematic (maculo-papular) reactions are the most frequent cutaneous reactions to drugs. They occur with almost any drug but most commonly after ampicillin and penicillin, phenylbutazone, sulphonamides, phenytoin, carbamazepine, gold and gentamicin. The eruption may develop at any time but usually two weeks after administration and may be associated with fever, pruritus and eosinophilia. The features are variable—scarlatinaform, rubelliform, morbiliform or less commonly, large macules, polycyclic and gyrate erythema. The eruption is generally symmetrical affecting the trunk and extremities. Drug eruptions usually fade with desquamation but, if the offending drug is continued, an exfoliative dermatitis may develop.

Urticarial eruptions may be a cutaneous manifestion of a Type I reaction mediated by IgE antibodies (penicillins, cephalosporins, hydralazine, phenylbutazone, radiographic contrast media), or a Type III reaction in which the antigen forms a complement-fixing-complex with the antibody (aspirin, penicillins, thiouracils). Some drugs, for example, aspirin and morphine are capable of

inducing urticaria by an allergic or pharmacological mechanism. ACE inhibitors may have a direct action on kinins.

Drug induced vasculitis may involve the skin and internal organs and can be associated with allopurinol, captopril, cimetidine, hydralazine, penicillin, thiazides and sulphonamides.

The mechanism of drug-induced lichenoid eruptions is unknown. These may closely resemble idiopathic lichen planus and tend to be extensive sometimes developing into an exfoliative dermatitis. The agents implicated include beta-blockers, captopril, thiazides, gold, chlorpropamide, frusemide, methyldopa and phenytoin.

Fixed drug eruptions tend to occur at the same site each time the drug is administered. Acute lesions are well-defined, round or oval plaques with erythema, oedema and blistering appearing within 30 minutes to eight hours of drug administration. The limbs, hands and feet, genitalia and perianal areas are favoured more than the trunk. Tetracyclines, sulphonamides, barbiturates, ibuprofen, dapsone, paracetamol and benzodiazpines have all been implicated.

Drugs are the aetiological factor in ten per cent of cases of erythema multiforme. Macular, papular or urticarial as well as the classical iris or target lesions may be seen. Deposits of IgM and C3 may be found in the walls of superficial blood vessels and circulating immune complexes have been reported. Sulphonamides, cotrimaoxazole, barbiturates, rifampicin, penicillins, phenothiazines, chlorpropamide and thiazides have all been indicted. Emphasis was placed on the importance of appreciating that re-exposure to drugs suspected of causing a reaction has resulted in fatality and should not be carried out for diagnostic purposes. Toxic epidermal necrolysis (TEN) is one of the real dermatological emergencies with a mortality rate between 20 and 30 per cent. The commonest triggers include sulphonamides, barbiturates, carbamazepine, phenylbutazine, allopurinol, phenytoin, ampicillin and amoxycillin. It presents clinically with an initial 'burning' morbiliform eruption, accompanied by flu-like symptoms and rapid progression to areas of confluent erythema, followed by blistering and widespread exfoliation. Patients can become critically ill requiring management in a burns unit. Treatment requires dedicated nursing, and accurate fluid and electrolyte balance. The use of steroids, however, remains controversial.

The diagnosis of a drug eruption demands a full drug history with close attention to laxative, vaccines, homeopathic and over the counter medication. It is imperative to establish when each drug was first taken relative to the onset of the reaction and whether the same or related drug has been administered previously. Resolution of a drug eruption on withdrawal of a drug is supportive incriminatory evidence but not diagnostic. Rationalisation and possible substitution of medication, particularly with polypharmacy, should be practised if a drug eruption is suspected. Skin testing, including prick and intradermal testing have only a limited role to play. Patch testing, however, may be useful in fixed drug eruptions.

Genes and the skin
See pp 25–28.

Skin malignancy
As a result of increased attention, both in the media and medical press, the

TABLE 1
Breslow thickness and effect of public education

	Pre-campaign 1979–1984		*Subsequent 1986–1990*	
	Males (%)	*Females* (%)	*Males* (%)	*Females* (%)
Thin	33·3	41·5	47·8	58·5
Intermediate	28·5	30·1	25·6	22·1
Thick	38·2	28·4	26·6	19·4

significance and importance of skin cancer is gradually being made aware to all. Malignant melanoma is the most rapidly increasing cancer in Scotland, with the incidence of other skin cancers (basal cell and squamous cell carcinoma) also rising. The Health of the Nation document in 1992 set out to 'halt the year on year increase in skin cancer by the year 2005'.

Basal cell carcinomas represent the commonest type of skin cancer making a significant contribution to the morbidity rather than mortality of cutaneous neoplasms. Squamous cell carcinomas tend to arise on a background of solar damage, seen in the elderly, particularly on the face and hands but also in the immunosuppressed. Chemical carcinogens, ionising radiation, human papillomavirus and ultraviolet light, are all aetiological contributors. Definitive treatment is by excision with skin grafting if necessary. Localised radiotherapy is a therapeutic possibility in the elderly or infirm, and the oral retinoids may provide some benefit to the immunosuppressed who often have disseminated squamous cell carcinomas.

Malignant melanoma continues to be 'the black spot' of dermatology. World wide epidemiological studies show an overall increase of seven per cent per annum of invasive malignant melanoma with somewhat worryingly, a more rapid increase in the last five years. The Scottish Melanoma Group is one of the largest geographically based databases with information on over 5,000 people with melanoma. Data collection started in 1979 from five regions in Scotland—Highland, Grampian, Tayside, West of Scotland and the South-East. The Group has witnessed a rise of invasive melanoma over the last ten years of eight per cent per annum, particularly on the legs of females and on the back and trunk of males. In the UK superficial spreading malignant melanoma represents 60 to 70 per cent of all cases. Nodular melanoma and lentigo malanoma each account for approximately 15 per cent and the acral lentiginous variant less than 10 per cent. This last type is eight times more common on the feet than on the hands and to the unaware may be overlooked, particularly if it is an amelanotic variant.

Risk factors for malignant melanoma include a large number of naevi, a freckling tendency, dysplastic naevus syndrome (greater than two clinically atypical naevi), history of severe sunburn (greater than three episodes of blistering sunburn, particularly in early childhood), previous malignant melanoma, family history of melanoma and a Celtic skin. Artificial ultraviolet light and sunbeds are associated with an increased risk of developing melanoma, particularly if more than 10 courses of UVB are undertaken in a year.

Malignancy should be considered if a mole changes in size regularity, shape or colour, or if there is oozing, crusting, bleeding or inflammation

The single most useful prognostic factor in melanoma is the Breslow thickness. This is measured from the granular layer of the epidermis to the deepest invasive cell; thin melanomas (0·1–1·49 mm), intermediate (1·5–3·49 mm), thick (>3·5 mm). The five year disease-free survival for thin malignant melanomas is 92·5 per cent, intermediate 72·6 per cent, thick 48 per cent. Other poor prognostic factors include the site of the lesion, the worst being the trunk, posterior scalp and posterior arm. Male gender and the presence of ulceration (even microscopic) are poor prognostic factors. Why females do better than males is yet to be fully explained (five year disease-free survival for males is 58·7 per cent and for females 77·6 per cent).

In the Health of the Nation document, the prime prevention of melanoma aims to increase public awareness and particularly to target children. The wearing of sunscreens and clothing echoing the 'slip slap slop' educational campaign in Australia are advised, with the avoidance of sun exposure—'between 11 and 3 get under a tree'.

In 1985 a public and professional education campaign was launched by Professor R. MacKie (University Department of Dermatology at Glasgow), to promote awareness of melanoma. The success of this campaign has resulted in an increase in thin melanomas with a concomitant fall in intermediate and thick melanomas (Table 1).

AIDS and the skin

See pp 40–44.