An International Journal for Community Skin Health

Community Dermatology

EDITORIAL: COMMUNITY DERMATOLOGY – WHAT IS IT?

In most of the world, health care is provided not by doctors but by other health care workers, such as nurses, the majority of them in small rural communities. Usually, there is very limited access to hospitals and medical specialists for their patients.

Knowledge and Training

In addition to medical supplies, health care workers also need opportunities for training and further education. This is already provided for Dermatology in some centres, such as the Regional Dermatology Training Centre at Moshi in Tanzania. The vast majority of these health workers, however, do not have access to such centres and an alternative is to send information to the workers in their own situations. This is already being done by *Community Eye Health*, published by the International Centre for Eye Health for the last 16 years. Currently, 16,000 copies are distributed to 178 countries, four times a year (free to developing countries). The founder of this Journal, Dr Murray McGavin, has said; "Every day, in communities throughout the world, individuals urgently require health care, yet all too often a health worker simply does not know what to do. Sadly, patients may even be harmed – simply through lack of knowledge".

Village young people in Tanzania

Photo: Paul Buxton

Editorial Board; ICTHES World Care

This Journal is intended to provide guidance for health care workers in the area of skin diseases and conditions such as leprosy, that manifest themselves by skin changes. There is an Editorial Board that plans the policy, content and management of the Journal. Publishing and distribution is being carried out by ICTHES World Care – a charity that also publishes similar journals in three other specialties. It is funded by voluntary contributions.

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J Comm Dermatol 2004; 1: 1–16 Issue No. 1

Editorial

Objectives

The objectives of the Journal are to bring up to date, relevant information on the diagnosis and treatment of skin disease to health workers in rural areas, using the resources available to them. It also sets out to provide information that can be used to educate health workers and the populations they serve. TALC cards will also be introduced and used in conjunction with the Journal. TALC (Teaching Aids at Low Cost) cards will be described in the next issue. There will also be a place for contributions from health care workers themselves with their views and experience.

In a wider context, the Journal also aims to increase awareness of the need for Dermatology services in developing countries and draw attention to the opportunities for providing them.

The Journal

Community Dermatology is not just a means of communicating knowledge but is a forum for all of us who are concerned to bring health and healing to those parts of the world that need it most. Your comments and ideas are very welcome.

This first issue covers the very important topics of Emollients and the Skin, AIDS and Leprosy. There are also abstracts from other journals. In future issues there will also be a quiz and case studies, as well as contributions on other common problems of the skin, such as scabies, and also traditional treatments. Regular features on treatment and compounding skin medications are also planned. The Editorial Board hope that you will enjoy reading the Journal and find it both instructive and a help in managing your patients.

Paul K Buxton

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Dr Paul K Buxton was born in Ethiopia and followed undergraduate studies at Cambridge University (Trinity College) and St Thomas's Hospital, London. In 1961–1962 he carried out general medical work with the Grenfell Mission in Newfoundland. Specialist Dermatology training followed in the UK and then between 1971 and 1981 he practised in British Columbia, Canada. Thereafter, Dr Buxton was consultant dermatologist in Fife and the Royal Infirmary, Edinburgh (1981–2002). Dr Buxton was one of three founding editors of 'Ethics and Medicine' and has written the 'ABC of Dermatology', published by the BMJ Publishing Group. He is now retired and living in Hampshire, UK. Other interest include art, christian ethics, country pursuits and seafaring.

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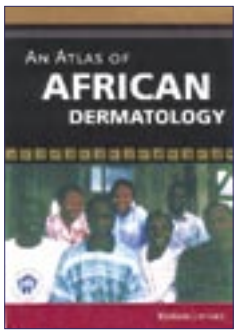
Book Review

AN ATLAS OF AFRICAN DERMATOLOGY



Barbara Leppard
Radcliffe Medical Press, 2002

This is a terrific book. Textbooks with good clinical photographs of skin conditions in African populations are few and far between. Of those I have seen, this is by far the most comprehensive. Years of practice in a local, regional and tertiary referral Centre for Dermatology in Tanzania have allowed Dr Leppard to build up an extremely wide range of first-rate photographs that includes all dermatoses likely to be encountered in a lifetime of medical practice. One or two of the photographs are slightly underexposed but overall the quality is excellent and the relevant physical signs are demonstrated with beautiful clarity.

Diseases covered range from common, everyday dermatoses, such as dermatitis and tinea, to rare conditions like epidermolysis bullosa and incontinentia pigmenti. This book is therefore a helpful aid to the important task of diagnosing more obscure and difficult skin conditions but Dr Leppard has not fallen into the trap of over-emphasising the rare and wonderful. Common treatable skin conditions are dealt with at greater length with plenty of practical advice about how to really help patients presenting with these diseases. For instance all varieties of eczema/dermatitis are covered in 14 pages with very helpful tips on management.

This book is therefore not just a large collection of excellent clinical photographs. It would also serve well as a basic textbook of Dermatology because for each condition depicted there is a concise and very practical explanation about aetiology, diagnosis and treatment.

Sam Gibbs

AN ATLAS OF AFRICAN DERMATOLOGY
BARBARA LEPPARD, 2002

Authorative guide to dermatological conditions for black populations in Africa. Contains over 600 clear colour pictures of dermatological cases to assist the recognition, diagnosis and treatment of both common and rare skin conditions.

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The First Commandment: Oil It!

An appreciation of the science underlying water and emollients for skin care

Terence J Ryan

Professor Terence J Ryan is Emeritus Professor of Dermatology, at Oxford University. His first Consultant Appointment, in 1970, was at the Royal Postgraduate Medical School, when he was also a Senior Lecturer at the Institute of Dermatology, London. Professor Ryan has had a lifelong interest in the blood supply and lymphatic drainage of the skin. From the time of Registrar appointment in Dermatology, in Oxford, 1962, he has collaborated with that Department's interest in leprosy. In 1987, he was appointed as Secretary to the new International Foundation for Dermatology of the International League of Dermatology Societies and became its Chairman in 1997. He was President of the International Society of Dermatology at that time. Professor Ryan is a Director of the Oxford based organisations, 'The Global Initiatives For Traditional Systems (GIFTS) of Health' and 'The Oxford International Biomedical Centre'.

Professor Terence J Ryan DM FRCP
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In the ancient traditions of the use of goose fat, valuing vernix caseosa and the slippery surface of the new-born, the activities of the cosmetic industry, or the much used coconut oil to shine the African skin, therein is a miraculous first principle. The skin benefits from washing and emollients. Both water, or animal urine, and grease of some kind are nearly always available, sustainable and at low cost; except when refined and perfumed and marketed as a gold standard beauty cream for the wealthy.

This miracle, an emollient applied as a first principle for the maintenance of the health of the skin and often to cure it of its sickness, has a rational scientific basis for such claims – and justifies its promotion as the subject of the First Commandment in Dermatology.

The Functions of the Skin

In pursuit of 'Health for All', the skin must not fail.

The functions of the skin are display (also known as 'the look good feel good factor'), thermoregulation, perception and barrier function. These are most obvious when the skin is absent, as in burns, abrasions and ulcers. The barrier function is measured by a very sensitive technology known as transepidermal water loss. It increases in many skin diseases.

The Structure of the Skin Barrier in Health

The skin is the largest organ of the body. It is not just a passive 'sleeve' keeping our insides in, but it is pliable and elastic, watertight, and an immunosurveillance system keeping out irritants, allergens and infections. Its surface barrier is manufactured by a keratin and lipid factory which is mostly anaerobic from the mid-epidermis to the surface. The components are tightly knit. The factory is policed by immunosurveillance cells ready to detect any foreign penetration of the barrier. There are also the pigment cells, melanocytes, secreting a free radical quenching agent, melanin, dampening down any potential harmful agents. In health, it all works very well and requires very little fuel. Only the occasional migration from, or division of a cell within the basal layers of the epidermis requires oxygen. The provision of tissue fluid and other minimal nutritional requirements, and the balance of tension between fibres and cells by tissue fluid and pressure is controlled, in health, by an effective blood supply in the upper dermis.

While the epidermal factory behaves in this way, little other than a barrier is manufactured and there are no demands for an increase in blood supply. It has even been argued that the richness of the blood supply exceeds the metabolic needs of the skin and that it is necessary for thermoregulation. "Not so!", says this author, for hardly a minute goes by without injury from a constantly threatening mechanical and chemical environment placing the skin constantly on a 'war footing' (expecting attack and defence) demanding an instant increase in fuel supply.

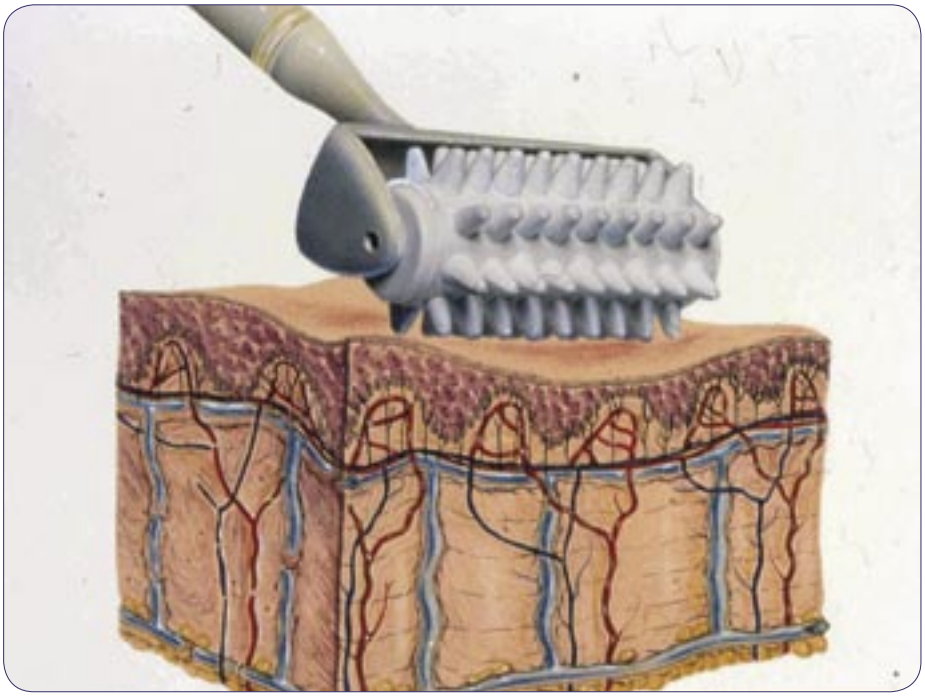

Fig. 1: Diagram epidermis under mechanical stress. Should disruption of the integrity of the epidermal barrier occur, repair mechanisms will be switched on. The integrity is most likely to be retained if the skin surface is well 'moisturised' by emollients

The Skin at War

The slightest breaching (damage) of the epidermal barrier places the epidermal factory in a repair mode. Experimentally, this may be a range of insults such as stripping the surface with a few applications of sticky tape, the application of an irritant or allergen, depriving the factory of essential nutrition (as in vitamin or protein deficient experimental animals), pricking the skin with a pin or scratching its surface. Within seconds of a scratch, the axons of the sensory nervous system stimulate the release of agents such as acetyl choline and histamine to increase the blood supply 200 times above the normal non-stimulated level. This is necessary for the fuel requirement of the epidermis as its cells divide, migrate, or manufacture the bricks for the barrier to be repaired, as well as the increased production of a range of agents needed to inflame.

The *Inflammatory Reaction* is necessary to remove the insult and hasten repair by the recruitment of cells from the blood stream. When the damage is great, a new vascular organ of repair, granulation tissue has to be formed, and eventually removed, to provide enough fuel for the dividing and migrating epidermal cells and the recruited white cells, all of which are demanding oxygen and other nutrients.

It is only during the last decade that the full picture of the activated epidermis has been seen to be more than just the release of histamine and the production of prostaglandins. It now includes interleukins, interferons, tissue necrosis factors, as well as growth factors such as Vascular Endothelial Growth Factor (VEGF). These are rapidly produced in the epidermis to flood the dermis and activate the blood vascular endothelium – to bring into play the white cells and macrophages for elimination of the undesirable and for repair.

A Correlation Between an Intact Barrier and the Switching On or Off of the 'War Footing' Status: Emollients Can Do It!

Contemporary technology can breed mice without essential components for the epidermal factory. This has confirmed that when the barrier is broken, the cytokine production is switched on, and when repair is complete they are no longer manufactured. Probably the epidermis first knows that its barrier is breached when transepidermal water flows faster and is lost from the surface. Such flow and loss can be instantly slowed down by placing an artificial barrier on the skin surface, such as an emollient petrolatum, honey or a covering of polythene or hydrocolloid dressing.

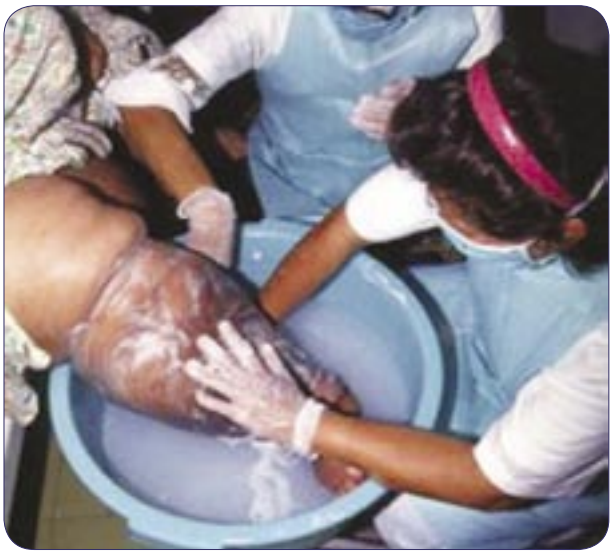

Fig. 2: Washing rituals are global and traditional. They also have an evidence based medical rationale

Photo: Terence Ryan

Bacteria and Other Potential or Actual Enemies on the Skin

There are always bacteria on the skin and often viruses, fungi and parasites. These thrive on unhealthy skin, macerated, eczematous, cracked with deep wet crevasses, or moistened under a dried out scale. More than fifty years ago Dermatologists applied large amounts of bacteria and fungi to the surface of intact skin, under a plaster, and there was no inflammation or evidence of harm. When they first, quite gently damaged the surface of the skin by irritants or scratches, there was rapid infection and increased pathology. Loss of barrier function and easy penetration allows infective organisms, not only to invade but to find an environment in which they can thrive and cause trouble. Today, we know that many epidermal products manufactured when on a 'war footing' are used by infectious organisms, while some known as 'defensins' dampen their capacity to do harm. Penetration of the barrier may allow organisms to go where there are no such 'defensins', nor white cells to deal with them.

The clinical picture, for example, is that of maceration between the toes – nearly always a potential 'entry point' for bacteria. Other organisms, such as fungi, may break up the epidermis and add to the barrier breakdown, as may irritants from soil or soaps, or excess moisture unable to evaporate in a humid environment.

A recent Cochrane review of *Impetigo* emphasised that antibiotic creams can speed resolution, but given a little time, ointment bases do as well. In the field of *Lymphology*, treatment of the recurrent inflammatory episodes in lymphoedema, attributed to bacteria gaining easy access through a broken skin barrier, has given strong emphasis to skin care with washing and emollients, rather than hastening to prescribe antibiotics. This self-help, low cost approach explains that washing clears excess organisms and skin surface debris and emollients provide barrier function. Any emollient, traditionally used, is probably safe and an agent such as coconut oil works well. Some cleansing agents from herbals have additional

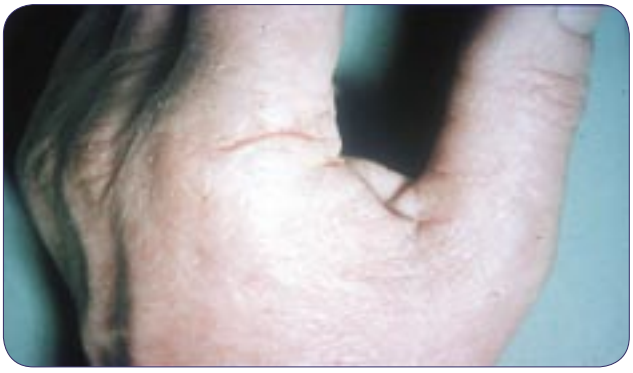

Fig. 3: Dry and cracked skin due to wear and tear. The cracks could be prevented by emollients, frequently and liberally applied

Photo: Terence Ryan

attributes. Tea is antiseptic, antihistaminic and antioxidant and, like diluted honey, can be added to water and emollient to make a highly recommendable application. There are more than 300 traditional plant based soaps with a low level of irritancy and allergenicity.

The best emollients are natural to the species on which they are found. In man, sebum and sweat are natural emollients and salty tears and the lipid of the Meibomian glands are exquisitely effective, protecting the epithelial surface of the cornea. Natural emollients, such as wool fats from sheep have long been the basis of the lanolins used as hand and face creams, with a wealth of supportive evidence as to why they are so effective. Together with the evolution of the emollient, Man has evolved ritual washing, written into ancient religious texts, such as in Leviticus (Bible), the Koran or in Sanskrit. Further, the mucosal surfaces in relation to sexual practices have never been the focus of so much attention to the concept of 'Barrier', as they are now in the face of the AIDS epidemic.

This author promotes *Bee products* as perhaps the most successful of agents that protect the bee from a range of infections and should be used for human disease with more confidence. The latest studies show that honey immobilises at least 20 bacterial genes controlling their reproduction. (www.abc.net.au/programsales/programs/s989015.htm)

Dermatology`s greatest service to 'Health for All' may be its promotion of the maxim, 'cleanliness is next to godliness'. This is supported by evidence and should receive greater emphasis. It is an extension of the bathing used for common skin diseases, of wet wrapping, and the emphasis on emollients in atopic eczema so as to leave a lipid film on the surface of the skin as a barrier contributing to an intact skin. It is found in best *Dermatology Nursing* practice for the moisturising of dry skin, as well as for the 'drying' of the incontinent, whether the infant or the bedridden elderly.

It is the nearest thing to a 'cure-all' or the miracle that deserves to be the subject of the *First Commandment of Dermatology*. It should be regularly prescribed. Even in health, the feel of the presence of an emollient after applying a hand cream soon wears off. For the barrier effect to be prolonged on diseased skin, the application must be very frequent and well spread. The large organ may need to be treated as a whole. The need is for locally available, sustainable low cost provision. It should not sit on a shelf or become rancid in the sun. When prescribed generously, effective use is *for now*. This has to be understood by the patient and, therefore, good advice and good advocacy are essential.

If this presentation is regarded as too basic, some kind of witchcraft or unscientific, then the following '*Further Reading*' may provide the necessary *Evidence Based Medicine* to convince the sceptic.

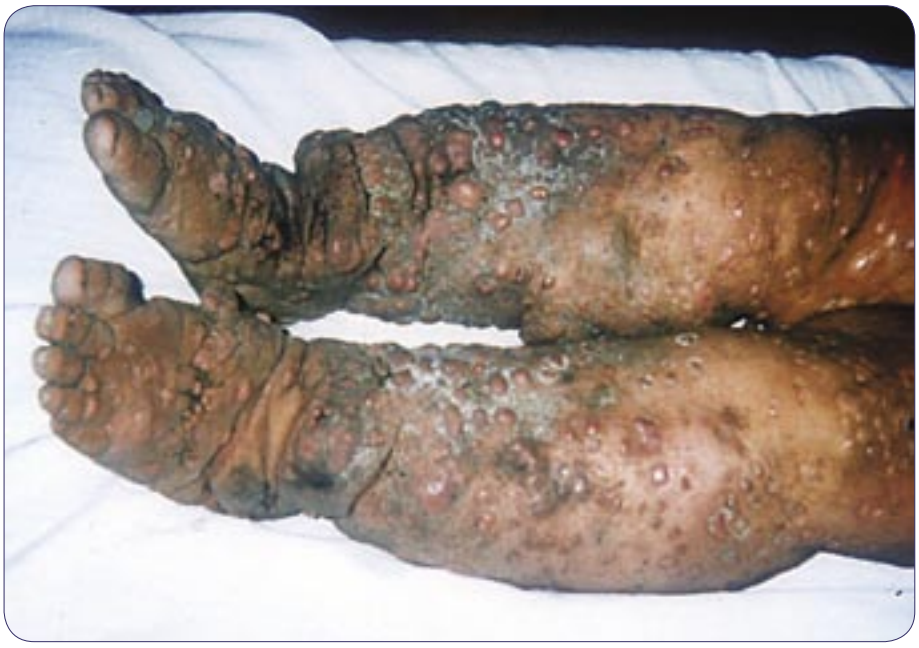

Fig. 4: The skin changes in lymphoedema, appropriately described as 'elephantiasis'. There are many cracks and crevasses. Regular washing and emollients prevent the loss of barrier function becoming a regular entry point for bacteria

Photo: Terence Ryan

Further Reading

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Skin Signs of HIV/AIDS

Barbara Leppard

In countries where HIV testing is expensive the diagnosis of AIDS is made on clinical grounds (see World Health Organization criteria in box). Most patients with HIV/AIDS have skin disease at some point in their illness. The most important ones to recognise are:

- Herpes zoster
- Other viral infections which are extensive or have an unusual distribution
- Kaposi's sarcoma
- Oral candidiasis
- Extensive fungal infections
- Any combination of infections (bacterial, viral, fungal)
- Drug rashes
- Eczema
- Photosensitivity +/- actinic cheilitis
- Blue nails.

Dr Barbara Leppard qualified at St George's Hospital in London in 1967. She trained in Dermatology at St George's Hospital with Stephen Gold and Ken Sanderson, and at St John's Hospital for Diseases of the Skin. During her training she spent 16 months setting up a Dermatology department in Shiraz, Iran.

Dr Leppard was appointed as Senior Lecturer in Dermatology at the University of Southampton in 1977. From 1992 to 2001 she worked as a Consultant Dermatologist at the Regional Dermatology Training Centre, KCMC, Moshi, Tanzania, where she was also the Academic Officer. When KCMC became a medical school she was appointed Associate Professor. Her main interests are teaching and clinical medicine. She has written 3 popular textbooks of Dermatology, one of which is reviewed in this Journal.

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WHO Criteria for the Clinical Diagnosis of AIDS

Major signs
- Weight loss of > 10% body weight
- Chronic diarrhoea for > 1 month
- Prolonged fever for > 1 month (intermittent or constant).

Minor signs
- Persistent cough for > 1 month
- Herpes zoster
- Chronic progressive or disseminated herpes simplex
- Oropharyngeal candidiasis
- Generalised itchy rash.

In an adult the diagnosis of AIDS is made if there are at least 2 major signs and at least 1 minor sign.

Herpes Zoster

Herpes zoster (Figure 1) is often called 'the soldier's belt' in Africa. This is an infection with the *herpes varicella zoster virus* which has been lying dormant in the body after an episode of chicken pox in childhood. It begins with pain for a day or two before the rash appears. Then groups of blisters appear along the course of a dermatome, stopping abruptly at the midline. The rash occurs in a band around the trunk, down a limb or on one side of the face. The blisters then break, ooze fluid, crust over and heal in 3–4 weeks. The rash is painful until it heals and sometimes afterwards.

Treatment:

1. Regular analgesics for the pain:
   - Aspirin 600mg every 4 hours
   - Paracetamol 1g every 4 hours.
2. Topical Gentian Violet paint (0.5%) once the vesicles have burst to prevent secondary bacterial infection.
3. If the patient is seen within 48 hours of the onset of blisters, give oral aciclovir 800mg five times daily for 7 days (but this is very expensive).

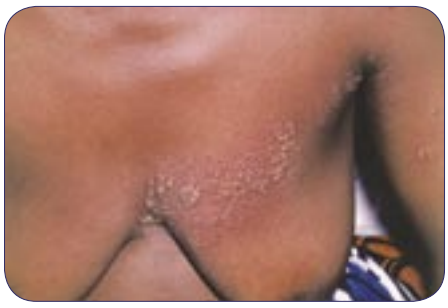


Fig. 1: Herpes zoster around the left side of the chest
Photo: Barbara Leppard

Other Viral Infections

Chicken pox (Figure 2). This is a common viral illness in children, but when seen in adults should make you think of HIV infection. It is a very itchy rash on the face and trunk. The lesions come in crops, so that there are always lesions at different stages of development present at the same time – macules, papules, vesicles, pustules and crusts. The most characteristic lesion is the vesicle looking like a clear drop of water on the skin.

Treatment:
Applying calamine lotion will help the itching.

Herpes simplex (Figures 3 & 4). Grouped blisters, around the mouth (type 1) or on the genitalia (type 2), which crust and

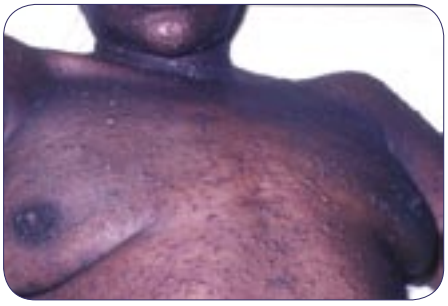


Fig. 2: Chicken pox in an adult
Photo: Barbara Leppard

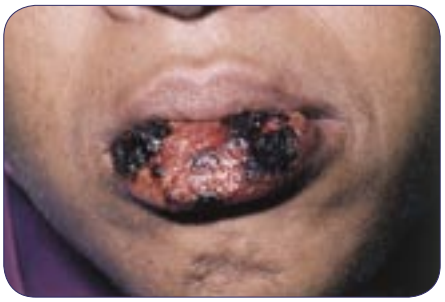


Fig. 3: Chronic herpes simplex type 1 infection on lower lip. This ulceration and crusting has been present for 2 months (HIV)

Photo: Barbara Leppard

heal in 7–10 days, are commonly associated with fever and keep recurring at the same site. In patients with HIV infection the blisters may ulcerate and last for weeks or months rather than just a few days.

Treatment:
Treatment with oral aciclovir is only useful if it can be started within 48 hours of the onset of vesicles. The dose is 200mg five times daily. It will not help chronic ulceration. Prophylactic treatment with aciclovir 400 mg bd is helpful to prevent recurrences in patients who are getting them frequently, but this is very expensive.

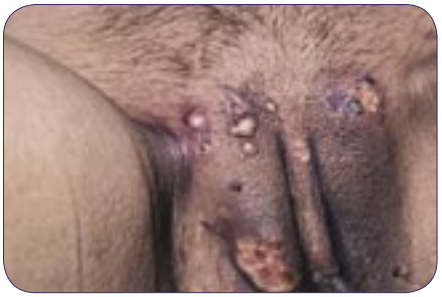


Fig. 4: Herpes simplex type 2. Grouped vesicles and ulceration

Photo: Barbara Leppard

Molluscum contagiosum (Figure 5). Small umbilicated papules in children are common. When seen in adults think of HIV infection.

Treatment:
Apply 1% hydrogen peroxide cream bd for 3 weeks.

Oral hairy leukoplakia (Figure 6). Viral infection due to the *Epstein Barr virus*. Looks like fine white hairs along the sides of the tongue. It does not scrape off like *Candida*. It is asymptomatic. Its importance is that it is a reliable marker of HIV infection.

Warts (Figures 7 & 8) are an infection of the epidermis with one of the human papilloma viruses. All types (common warts on the hands, plantar warts of the soles of the feet, plane warts on the hands and face, warts in the mouth and genital warts) are more common in people with HIV. Be concerned if the warts are extensive as in these pictures.

Treatment:
Most warts can be left alone. Extensive genital warts can be treated with 15%

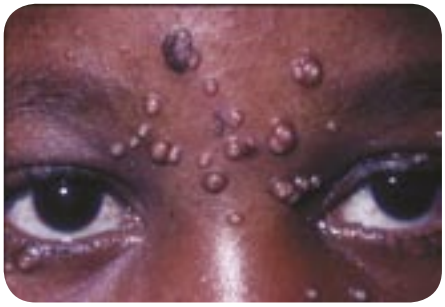


Fig. 5: Molluscum contagiosum

Photo: Barbara Leppard

podophylline paint, applied once a week only. It is painted carefully onto the warts and washed off with soap and water after 6 hours.

Kaposi's sarcoma (Figures 9, 10, 11 & 12) is an infection with *human herpes virus 8*. It often begins with papules, nodules and plaques on the lower legs which are reddish purple in colour, although they can occur anywhere on the skin. Individual tumours may ulcerate and the leg(s) may be swollen and feel woody hard. Lesions on the palms and soles show the purple colour better than the rest of the skin. It is common to get lesions in the mouth, especially on the hard palate; these may be asymptomatic as in the picture on the next page (bottom left), but large

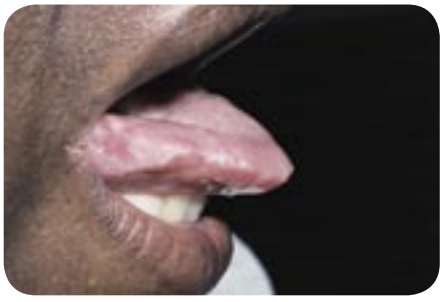


Fig. 6: Oral hairy leukoplakia

Photo: Barbara Leppard

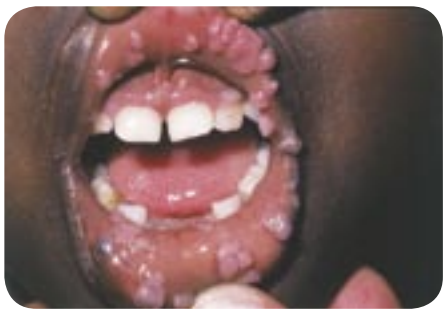


Fig. 7: Warts in the mouth

Photo: Barbara Leppard

tumours can bleed and make it difficult for the patient to speak and eat.

Treatment:
Treatment is only needed if tumours are painful or unsightly. Lesions on the arms and legs can be treated with radiotherapy. Widespread lesions can be treated with chemotherapy (vincristine, doxorubicin and bleomycin), but this is very expensive.

Fungal Infections

Oral candidiasis (Figure 13). Candida in the mouth causes white plaques on the tongue and buccal mucosa. They come off easily if you scrape them with a

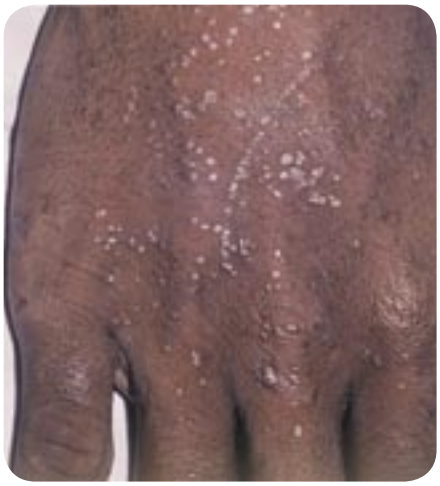
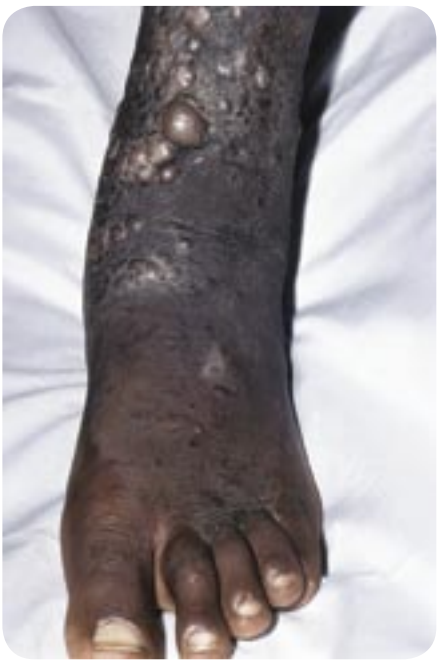
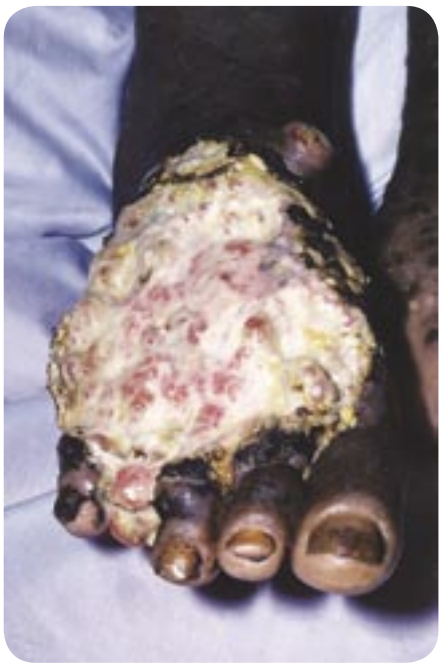
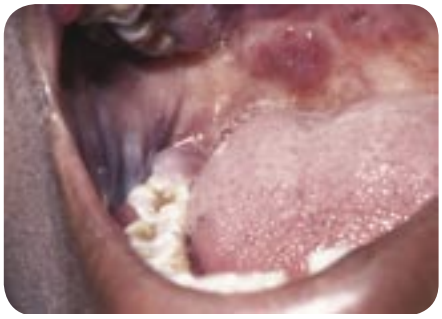
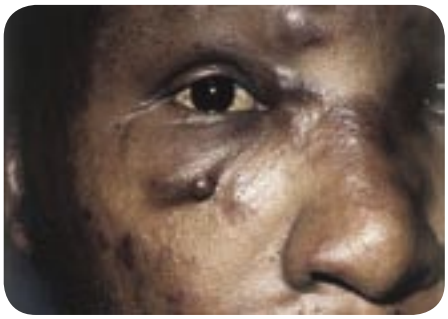


Fig. 8: Plane (flat) warts on the back of a hand

Photo: Barbara Leppard

spatula. Patients do not like this because it causes a burning sensation and spoils the taste of the food.

Treatment:
Gentian Violet 0.5% paint tds, used as a mouth wash. It is cheap and effective, working in 3–4 days. It tastes foul and is unsightly because it stains the teeth, tongue and lips a purple colour. Alternatives are:

Figs. 9, 10, 11 and 12: Examples of Kaposi's sarcoma

Photos: Barbara Leppard

- Nystatin oral suspension (100,000 units/ml). 1ml washed around mouth and then swallowed qds until clear
- Ketaconazole tablets 200mg/day for 2 weeks
- Itraconazole tablets 100mg/day for 2 weeks
- Fluconazole tablets 100mg/day for 2 weeks.

Extensive fungal infections (ringworm) (Figures 14 & 15). Ringworm is a scaly rash in the shape of a ring on the body or a semi-circle in the groin. There can be one or several lesions, but it is usually unilateral. If it is extensive or bilateral and symmetrical, think of HIV infection.

Treatment:
- Whitfield's ointment (Benzoic acid compound ointment) applied bd until the rash is gone and for a further 2 weeks. In the groin this stings, so alternatives are:
- Castellani's paint bd (but this stains the skin and clothes a magenta colour)

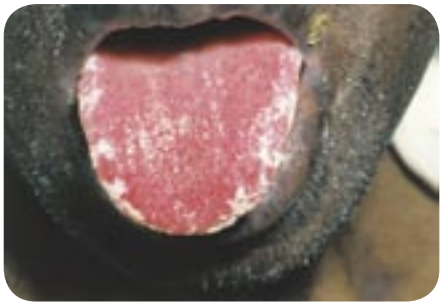


Fig. 13: Oral candidiasis

Photo: Barbara Leppard

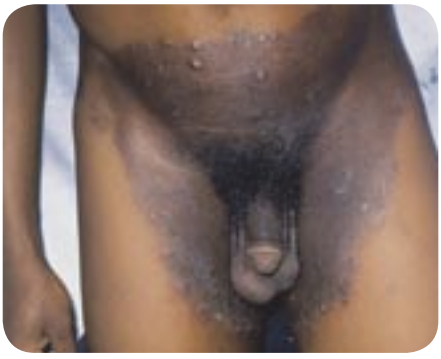


Fig. 14: Ringworm in the groin in patient with HIV. The rash is bilateral and symmetrical

Photo: Barbara Leppard

- An imidazole cream bd for 2 weeks (e.g., clotrimazole, ketaconazole or miconazole).

Scalp ringworm. Scaly bald patches with short broken off hairs on the scalp is ringworm. If you see it in an adult, think of HIV infection because it normally gets better spontaneously at puberty.

Treatment:
- Oral griseofulvin 500mg day for 6 weeks.

Assume that any patient with a combination of bacterial, viral or fungal infections, or eczema which is secondarily infected has HIV until proved otherwise.

Drug Rashes

All kinds of drug rashes are more common in patients with HIV infection. The most important ones are:

- *Fixed drug eruption* (Figure 16). One or more perfectly round, oedematous,

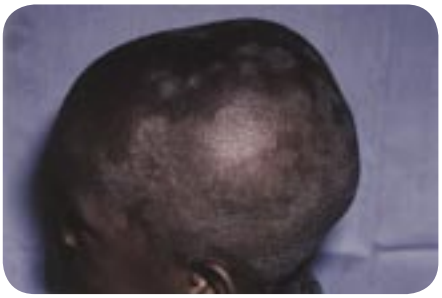


Fig. 15: Scalp ringworm

Photo: Barbara Leppard

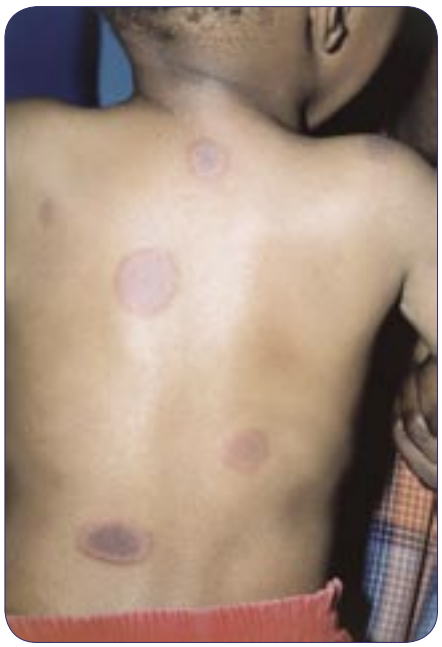


Fig. 16: Fixed drug eruption

Photo: Barbara Leppard

red plaques (+/- blisters) which keep recurring at the same site on the body every time the drug is taken. Between episodes there is a round brown patch where the rash has been. Septrin is the commonest drug to cause this.

- ***Toxic epidermal necrolysis*** (Figure 17). This is a very serious drug rash where the skin dies and peels off. It can be fatal due to fluid loss or secondary infection. The most likely drugs to cause it are sulphonamides or one of the anti-tuberculous drugs.

Eczema

Eczema is a poorly defined scaly rash which is usually very itchy. Think of HIV infection if:

- The patient had eczema as a child and it recurred as an adult
- There is sudden worsening of atopic eczema in an adult
- The eczema is follicular (around hair follicles) or very papular (Figure 18)
- The eczema is on sun exposed sites (face, 'v' of neck, back of neck and dorsum of hands and forearms) (Figure 19)
- The eczema is confined to the lower lip (actinic cheilitis) (Figure 20).

Treatment:
1% hydrocortisone ointment applied bd.

Blue fingernails and toenails in patients who are not taking HAART are a reliable marker of HIV infection (Figure 21).

Many other skin diseases can also occur in patients with HIV infection.
Whenever you see a rash, whatever its nature, keep the possibility of HIV in mind.
Although HIV infection is most common in the 20–40 age group, it can affect children and older adults, even in the 60s and 70s. ❒

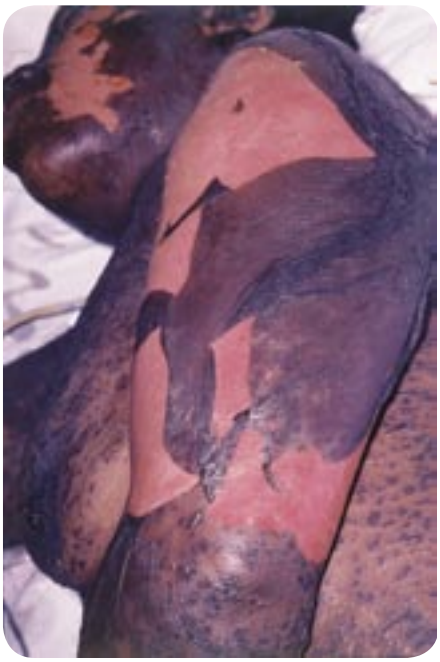


Fig. 17: Toxic epidermal necrolysis
Photo: Barbara Leppard

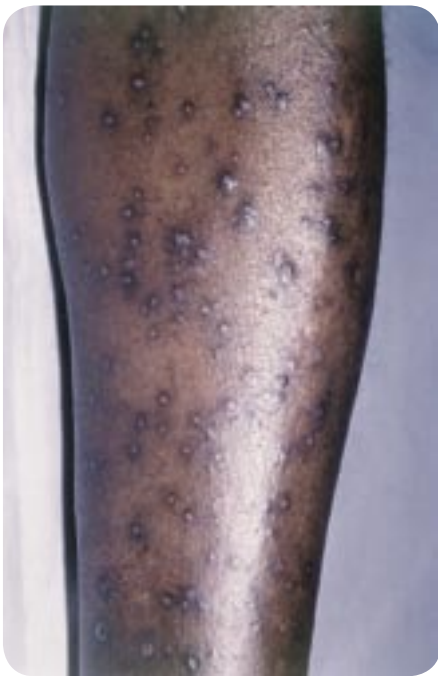


Fig. 18: Follicular/papular eczema, also known as a papular pruritic eruption (PPE)
Photo: Barbara Leppard

Fig. 19: Photosensitive eczema
Photo: Barbara Leppard

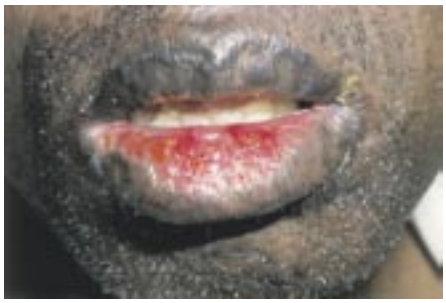


Fig. 20: Actinic cheilitis
Photo: Barbara Leppard

Fig. 21: Blue fingernails and toenails in patients who are not taking HAART are a reliable marker of HIV infection
Photo: Barbara Leppard

Skin Changes in Leprosy

Antoon J M Baar
Ben Naafs

Introduction

Leprosy is an infectious disease caused by an intracellular acid-fast bacterium: *Mycobacterium leprae*. In 1873, Armauer Hansen was the first to describe the bacterium as the cause of leprosy. However, it has not yet been possible to infect someone deliberately with *M. leprae,* although anecdotal reports show infection after tattooing, and following the skinning and cleaning of infected armadillos for the cooking pot. Though leprosy, in general, is considered to be a skin disease, it has acquired the stigma due to nerve damage, feared over centuries, making its sufferers social outcasts. When diagnosed and treated in time, no permanent damage will be done. However, the diagnosis of leprosy is often missed or delayed, with permanent disability as a consequence.

The changing attitude of the World Health Organization (WHO), governments and donor agencies towards leprosy control has resulted in rearrangement of 'vertical' leprosy services. These services are, for practical reasons, either combined with tuberculosis control or integrated into the general health services. As a result, in many instances, the care for the individual patient has decreased. As the number of health workers responsible for leprosy treatment increased, it was hoped that patients would be diagnosed in a very early stage of the disease, provided the health workers had sufficient knowledge of – and experience in – the diagnosis and the treatment of leprosy. However, the skills that were present in the vertical leprosy programmes disappeared due to the decreased prevalence of leprosy and the increased attention that had to be given to diseases such as HIV /AIDS and, consequently, tuberculosis. Early manifestations of leprosy are skin manifestations. Therefore, health staff trained to inspect skin (Dermatologists, Dermatological Officers and Nurses and, in the past, leprosy officers) are appropriate persons to diagnose leprosy and teach these skills to general health workers.

Fig. 1: The leprosy spectrum

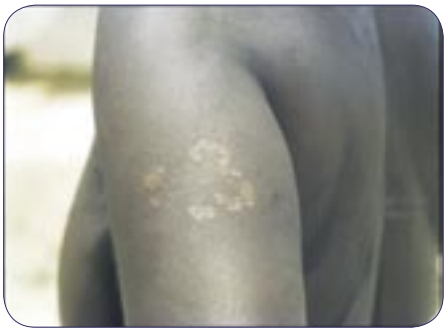


Fig. 2: Tuberculoid leprosy; well defined, loss of sensation

Dr Antoon JM Baar was trained as a Dermatovenereologist by Professor A H Klokke at the University Hospital Groningen in The Netherlands. Before this, he worked for 5 years in Tanga, Tanzania, the last 3-years as Regional Leprosy Officer. Formal training in leprosy was with Dr Leiker in Amsterdam and ALERT in Addis Abeba, Ethiopia. He is recently retired from his post as Consultant Dermatovenereologist at the Medical Centre Leeuwarden and at the Hospital Talma Sionsberg in Dokkum, where he practised general Dermatology in all its aspects, including venereology, flebology and dermatosurgery. At present, he is invited Senior Lecturer at the Regional Dermatology Training Centre (RDTC), in Moshi, Tanzania for three months a year.
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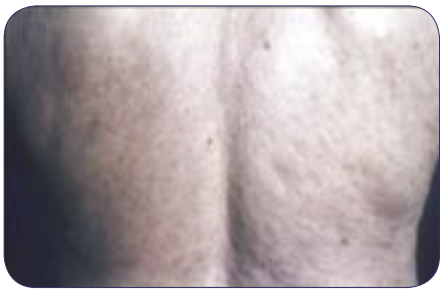

Fig. 3: Lepromatous leprosy; diffuse infiltration

Clinical Spectrum

The manifestations of leprosy are various, but it has been possible to classify the patients according to a clinical spectrum. This was effectively done coincidentally, but independently by Ridley and Jopling in the United Kingdom and by Leiker in The Netherlands, in 1966.

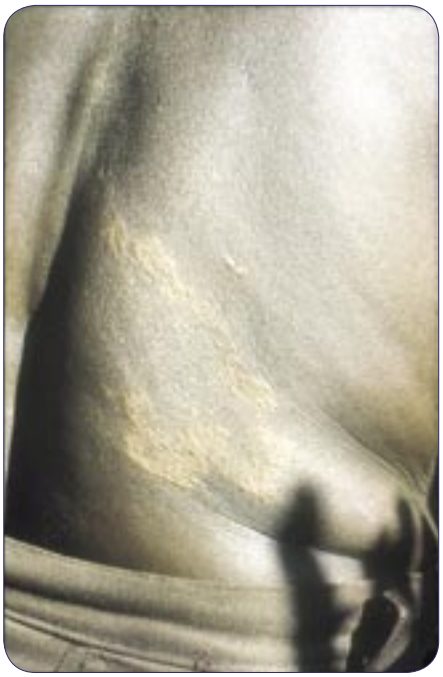

Fig. 4: Borderline tuberculoid leprosy; asymmetrical patches, loss of sensation, central healing

These classifications are based on the cell mediated immune response (CMI) of the patients against *M. leprae* (Figure 1). At one end of the spectrum, the tuberculoid (TT) leprosy patients have a relatively high CMI response towards *M. leprae*, with one or a few well defined hypopigmented or erythematous patches, usually with central healing and loss of sensation in the patch (Figure 2), and/or with an enlarged peripheral nerve. *M. leprae* is not detectable. At the other side of the spectrum, the lepromatous (LL) leprosy patients present with a complete tolerance to *M. leprae* and without a detectable CMI response against the organism. These patients harbour very many bacteria; they present the perfect culture medium. The bacteria may be present anywhere in the body, with the central nervous system (CNS) as a possible

Fig. 5: Borderline lepromatous leprosy: nodular infiltration in the colder areas

exception. The lepromatous patients may show minimal hypopigmented or erythematous patches, which are poorly defined and with sensation still present. However, they may show glove and stocking anaesthesia with symmetrically enlarged peripheral nerves. They may also have nodules and plaques, skin which is coloured or hyperpigmented or show a diffuse infiltration (Figure 3). There may be loss of eyebrows (madarosis) and a more or less generalised diminished sweating. Between these two ends of the spectrum, the borderline leprosy group is found, including most of the patients. The clinical picture of borderline tuberculoid (BT) leprosy is characterised by a few asymmetrically distributed, well-defined tuberculoid patches with slightly indefinite edges and occasional satellite lesions (Figure 4). Frequently, there are a few enlarged nerves. In borderline lep-

Fig. 6: Mid borderline leprosy: dome-shaped and punched-out lesions, the centre is not involved and has no sensory loss

Fig. 7: Indeterminate leprosy: hypopigmented or coppery lesions

romatous (BL) leprosy, symmetrically distributed, hypopigmented or erythematous macules and/or papules and nodules may be seen. The nodules are mainly located on the colder parts of the body (Figure 5). In the middle of the spectrum is a very unstable group, mid-borderline (BB) leprosy, showing lesions with an immune area (the centre of the lesion is not involved) and typical dome-shaped elevated small, urticaria-like plaques (Figure 6).

In the borderline range, patients may show variation in the clinical features, up-

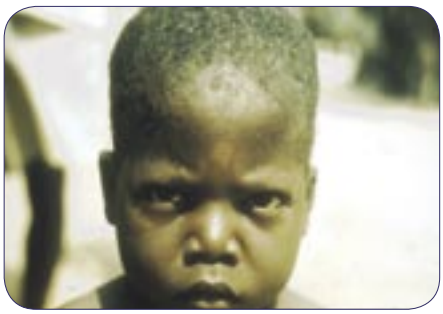

Fig. 8: Leprosy in children

or downgrading (e.g., change their classification within the spectrum). Upgrading indicates that the patient develops more tuberculoid features, downgrading more lepromatous. In upgrading leprosy, the bacterial load diminishes and in downgrading, the bacteria multiply. In a downgraded patient, a few patches may show loss of sensation, whereas the new lesions do not. In an upgrading patient, new tuberculoid lesions may appear or lesions may heal with atrophy.

Up- and downgrading occurs either unnoticed or is accompanied by a reactional phenomenon called Type-I or reversal reaction (RR), in which an enhanced CMI response towards *M. leprae* antigenic determinants may cause irreversible nerve damage.

Indeterminate leprosy comprises a special group of leprosy patients having

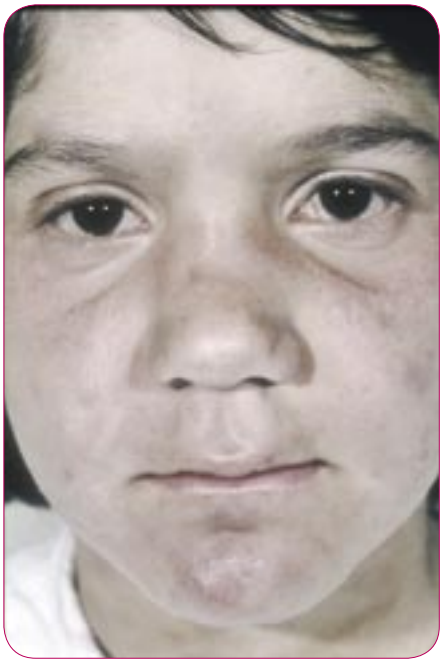

Fig. 9: Lepromatous leprosy in children, particularly in the colder skin areas (face)

one or two slightly hypopigmented or erythematous macules (Figure 7), with or without detectable loss of sensation or loss of sweating. Biopsy may show a single bacterium or minimal lymphocytic infiltration in a dermal nerve. The diagnosis is difficult to establish and some leprologists consider it to be an early, transient form which may either heal (over 80%) or become frank leprosy of one of the above mentioned types.

In 1982, the WHO proposed a classification based on the bacterial load of the patient. For operational purposes, the leprosy spectrum was simplified to paucibacillary (PB) and multibacillary (MB) leprosy. PB leprosy patients are Indeterminate, TT and BT leprosy, without *M. leprae* in the skin smear or biopsy. MB patients are BT, BB, BL and LL patients with *M. leprae* in the smear or biopsy. Because in many control programmes smear services are unreliable or unavailable, these programmes have to resort to very simple clinical criteria. Some classify all patients with 5 or less lesions as paucibacillary, others those with 3 or fewer lesions. The remaining patients are then classified as MB leprosy.

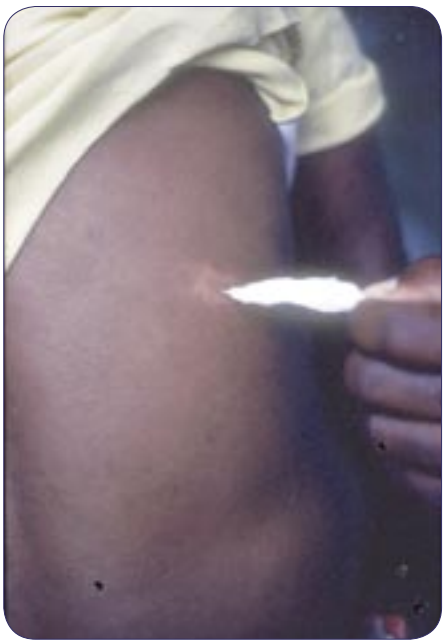

Fig. 10: Loss of sensation

Although leprosy may be present as early as 3 months after birth, it usually becomes manifest after the age of 6, depending on the endemicity and socio-economic circumstances. An important reason for this is that the incubation time, in general, appears to be between 2 and 5 years. Most children, when diagnosed early, show indeterminate leprosy or leprosy with borderline features, mostly tuberculoid (Figure 8). Since PB leprosy needs only a few bacteria to show clinical symptoms, the clinical manifestations of tuberculoid leprosy develop earlier in life than those of lepromatous leprosy. Lepromatous leprosy, when it develops in children, seems especially confined to the head and the extremities, i.e., the colder parts of the body (Figure 9).

Diagnosis

Awareness is the most important factor in the diagnosis of leprosy. When someone lives or has lived in a leprosy endemic country, leprosy must always be considered in the differential diagnosis of a hypopigmented or erythematous patch or a papular or nodular eruption. The same certainly applies for each condition accompanied by peripheral nerve function impairment.

Hypopigmented or erythematous patches are frequently seen. An important feature of a leprosy lesion is that it does not itch but shows loss of sensation to fine touch. This can be tested by using a piece of cottonwool made to a fine thread (Figure 10). The skin should not be stroked but touched with the cotton tip. The sensation in the patch is then compared with the sensation of the surrounding normal skin by asking the patient to point to where he or she was touched. For children, this can be done as a game in which the child keeps his eyes closed. It is remarkable how sensitive this investigation is. Established loss of sensation makes the diagnosis of 'leprosy' very likely, especially when an enlarged nerve can be palpated. It may not be possible to test the sensation in very young children. The absence of sweat in a skin lesion after exercise, sun bathing or exposure to heat may then be a helpful diagnostic tool.

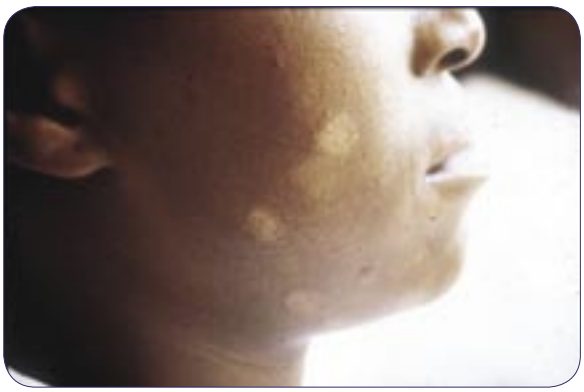

Fig. 11: Pityriasis alba

It is important to recognise that, in the face, hypopigmentation frequently occurs due to other skin problems, such as pityriasis alba (Figure 11), pityriasis versicolor, seborrhoeic dermatitis and mild forms of tinea. Differentiation of these common skin diseases on the face from early leprosy is not easy because loss of sensation in a leprosy lesion on

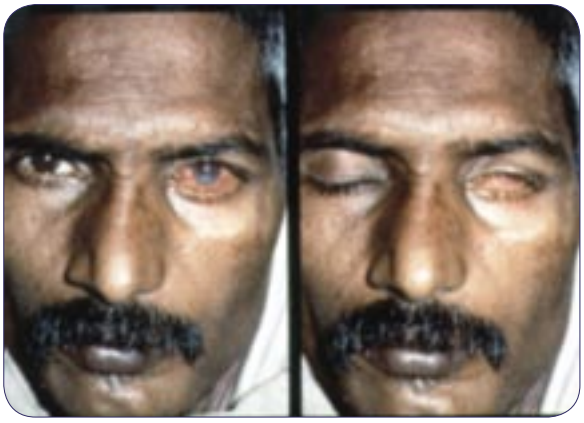

Fig. 12: Lagophthalmos

Photos: Margreet Hogeweg

the face occurs late, if it occurs at all. The diagnosis can be made by carefully describing the size and site of each lesion, and requesting that the child be brought back in 3 months. By then, a pityriasis alba spot has most likely disappeared or changed place. A leprosy patch will remain in exactly the original situation and may have enlarged. However, the evolution of leprosy at this stage is usually slow. Be alert for a hypopigmented patch that suddenly becomes inflamed. This is a sign of danger: a reversal reaction may be imminent! A reversal reaction in the face often leads to lagophthalmos (Figure 12) and loss of corneal sensation. This can result in blindness.

Nerves which can be inspected and palpated are the cutaneous nerves in the immediate vicinity of a patch: the great auricular, the ulnar, the median, the radiocutaneous, the lateral popliteal and the posterior tibial nerves. Enlarged nerves

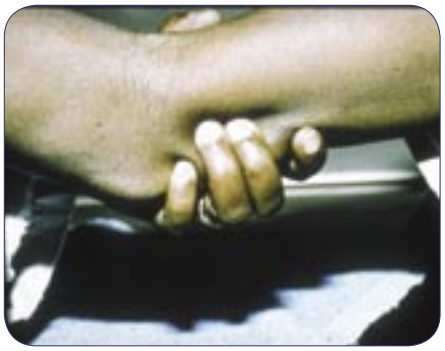

Fig. 13: Palpation of the ulnar nerve

indicate leprosy. Tender nerves may be a sign of reaction and require immediate action. Inexperienced examiners should learn to palpate at least the ulnar (Figure 13), the radiocutaneous (Figure 14) and the great auricular nerves.

When there are nodules or papules, which are skin coloured and firm on palpation, leprosy should be suspected, especially when these are symmetrically distributed on the colder parts of the body, such as the ears, nose, cheeks, elbows, buttocks and knees. A skin smear or biopsy should be positive for *M. leprae* (Figure 15). The presence of enlarged nerves may be helpful. Incomplete closure of an eyelid and dry spots on the skin of the palms or soles of the feet may also indicate leprosy and require further investigation. The same applies in the case of lagophthalmos, claw hands, drop feet, painless blisters and ulcers, but then severe damage has already occurred.

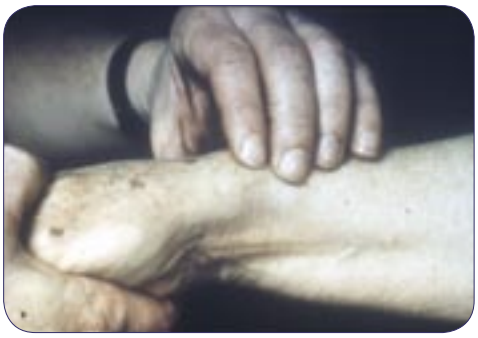

Fig. 14: Palpation of the radiocutaneous nerve

Classification may be difficult for the average health worker:

- Paucibacillary features are: loss of sensation in a well-defined patch with central healing. The patches are few (less than 3 or 5 depending on the definition used in the local leprosy programme) and asymmetrically distributed.
- Multibacillary features are: papules and nodules and/or ill-defined patches with a symmetrical distribution. In particular, small papules may be present on the ears; the earlobes may be swollen and sometimes a lateral madarosis (loss of eyebrows) is present; skin smears are positive. Nerves are symmetrically involved and enlarged.

Laboratory Tests

There are no laboratory tests sensitive enough to replace an expert field worker. Serology, especially against phenolic glycolipid I, can be used for the follow-up of an individual multibacillary patient during treatment, in the same way as the bacteriological index (logarithmic representation of the count of acid fast bacteria in a skin smear) is used. Serology may be positive in contacts and negative in PB patients. Modern techniques like PCR and Nasba will detect *M. leprae* DNA and RNA in all untreated MB patients. They are very sensitive and often do detect *M. leprae* also in PB patients and sometimes even healthy individuals.

Skin tests, in particular the well known lepromin or Mitsuda test, may be of assistance in the classification, being positive in tuberculoid and negative in lepromatous patients. However, it can be positive in leprosy contacts and even in leprosy non-contacts. The same applies for laboratory tests such as the Lymphocyte Transformation Test.

Histopathology is an important and sensitive diagnostic tool, but still the experienced physician remains the 'golden standard' for diagnosis.

Summary

Leprosy is a disease of the peripheral nerves, which become damaged when the disease is diagnosed late. The skin is the signalling organ. Well-trained staff, with great experience in the investigation of the skin are needed to diagnose the differences within the clinical spectrum of leprosy. Thus, early detection of nerves in danger is possible. Due to the change of most leprosy control schemes from vertical to integrated programmes, skilled staff are becoming rare, leading to late diagnosis of nerve pathology and increased chance of irreparable nerve damage and disability. Therefore, great efforts should be made to train primary health workers in recognising the early signs of nerve involvement in leprosy patients. This paper may assist in this training.

Acknowledgement

The photographs (apart from Figure 12) are from the archives of the late Professor Dr D L Leiker and those of the authors. Dr Margreet Hogeweg kindly provided the photographs for Figure 12.

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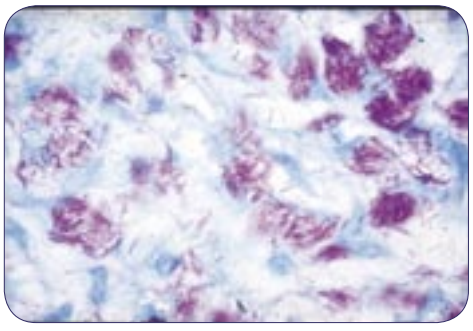

Fig. 15: Histopathology of borderline lepromatous leprosy

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Journal Extracts

This section offers some brief summaries of reviews and research from Dermatology and other medical journals. Space does not allow a detailed discussion of the contents of each article but this will be summarised and may be accompanied by an editorial comment or suggestion. We hope that this will be useful. If readers have read articles that they feel could be usefully brought to the attention of colleagues, we would be happy to receive any suggestions and details.

Neil H Cox
Editorial Board Member

Dr Neil H Cox is a 'general' Dermatologist, covering all areas of the discipline. His areas of interest and resulting publications over a 20-year period are varied, and have included diabetic skin, dermatitis herpetiformis, dermatomyositis, melanoma and non-melanoma skin cancer, photobiology and phototherapy, antimalarial drugs and lower leg cellulitis. Dr Cox has been involved with various clinical guidelines (Bowen's disease, melanoma and lichen sclerosus) and has chaired the Therapy Guidelines committee of the British Association of Dermatologists.
In recent years, Dr Cox has been involved with various 'process' issues in Dermatology, such as Nurse Prescribing, the UK National Cancer Dataset Project, and the National Health Service Modernisation of Medicines Manufacturing Committee.

He is co-author of four textbooks, has been a co-editor of the *British Journal of Dermatology*, and is currently co-editor of Rook's Textbook of Dermatology. Dr Cox is on the executive committee of the UK Dermatology Clinical Trials Network and is President-Elect of the Dermatology section of the Royal Society of Medicine.

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Xerosis and pruritus in the elderly: recognition and management

Norman RA. *Dermatol Ther* 2003; 16: 254–259

This USA journal devotes each issue to a single topic, in this case the topic is skin disease in older patients. This article reminds the reader that elderly patients have reduced sebaceous and sweat gland activity, combined with a higher chance of having additional disease and of being on medications. Relevant systemic diseases that cause itch and that apply world-wide include chronic renal disease, nutritional deficiency (especially of zinc and fatty acids), thyroid disease, malignancies and HIV infection.

Editorial comment: When considering causes of itch, it is important to think differently about patients with rash and those without. Keep in mind the fact that some rash may simply be secondary to scratching (in which case, the middle of the back that cannot easily be reached is usually spared) but also that scratching may hide the features of scabies. Conversely, remember that some patients without obvious rash may have subtle features of dryness, or may have urticaria that is only apparent when the skin is scratched to produce a weal (and that may be particularly difficult to detect in coloured skin).

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Chloroquine-induced, vitiligo-like depigmentation

Martín-García RF, et al. *J Am Acad Dermatol* 2003; 48: 981–983

This case report describes a rare pattern of pigmentary disturbance due to chloroquine. Diagnosis of depigmented patches is important in the tropics – pityriasis alba and other forms of post-inflammatory depigmentation are an important differential of leprosy. This pattern of pigment change related to chloroquine seems to occur only in the tropics. Early follicular repigmentation in the case reported suggests that it may be reversible if chloroquine treatment is stopped.

❐

Value of diagnostic techniques for cutaneous leishmaniasis

Faber WR, et al. *J Am Acad Dermatol* **2003; 49: 70–74**

The aim of this study was to evaluate the diagnostic role of polymerase chain rection technology in leishmaniasis – this has high sensitivity of 96% but is not widely available. However, it is of use that the authors also looked at more established diagnostic techniques. All had 100% specificity, but the sensitivity of each varied: 54% for smear, 69% for histopathology, 70% for culture and 89% for Montenegro skin test. Doing more than one test may improve the diagnostic rate. ❐

Dermatological disorders in Johannesburg, South Africa

Hartshorne ST. *Clin Exp Dermatol* **2003; 28: 661–665**

This is an analysis of 7029 patients from 5 academic units. There is no particularly complicated outcome, the importance is that the survey proves what we should all have suspected – across all races, the commonest problem (in a third of cases) was eczema. Of these, a third were of seborrhoeic type (an increasing prevalence, probably due to HIV infection) and 20% were atopic type. Acne was rather more common in black skin than white, and superficial fungal infections were most common in Indian skin. It is useful to remember that, despite the large number of dermatological conditions, common disorders still account for the majority of patients. ❐

A study of 124 Indian patients with lichen planus pigmentosa

Kanwar AJ, et al. *Clin Exp Dermatol* **2003; 28: 481–485**

This review is of interest to UK readers as we rarely see this condition, but even in India it represented patients accumulated over 12 years. The face and neck are the commonest sites for this grey-black pigmentation disorder, which is diffuse in 77% but may be reticular, blotchy or perifollicular. This disorder belongs in the spectrum of lichen planus, possibly sunlight-triggered or sunlight-aggravated; a minority, a bit over 10%, have associated typical lichen planus. ❐

Fatal Lucio's phenomenon in 2 patients with previously undiagnosed leprosy

Ang P, et al. *J Am Acad Dermatol* **2003; 48: 958–961**

This report from Singapore describes patients with progressive ulceration, blisters or bruises of lower legs for 2 years, in whom biopsies showed vasculitis and microthrombotic vascular occlusion. Both were anaemic with low serum proteins, impaired renal and/or liver function and high erythrocyte sedimentation rate, and both died despite treatment. Lucio leprosy or 'la lepra bonito' is a rare but aggressive necrotizing form of non-nodular leprosy. ❐

MEETINGS / CONFERENCES / COURSES

MAY 2004

IX International Congress of Dermatology

Venue:	Beijing, China
Date:	19th–22nd May 2004
Contact:	Cindy Froehlich
Tel:	001 847/240-1421
Fax:	001 847/330-1135
Email:	cfroehlich@aad.org

JUNE 2004

2nd World Congress on "Quality in Clinical Practice"

Venue:	Thessaloniki, Greece
Date:	3rd–6th June 2004
Contact:	Congress Secretariat
Tel:	+30 2310 256194
Fax:	+30 2310 256196
Email:	salonica@triaenatours.gr

JULY 2004

British Association of Dermatologists 84th Annual Meeting

Venue:	Belfast, Northern Ireland
Date:	6th–9th July 2004
Contact:	Emma Clayton
Tel:	020 7383 0266
Email:	conference@bad.org.uk

SEPTEMBER 2004

Paediatric Dermatology Course

Venue:	Liverpool, UK
Date:	9th–10th September 2004
Contact:	Dr G Sharpe
Tel:	0151 706 4030
Fax:	0151 706 5842
Email:	lmf@liverpool.ac.uk

SEPTEMBER 2004

13th Congress of the European Academy of Dermatology and Venereology

Venue:	Florence, Italy
Date:	17th–21st November 2004
Contact:	Torello Lotti
Email:	president@eadv2004.org
Web:	www.eadv2004.org/

An International Journal for Community Skin Health

Community Dermatology

Promoting Skin Health Around the World

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An older man in China

A child in Cambodia

A woman in Africa

Photos: Murray McGavin

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