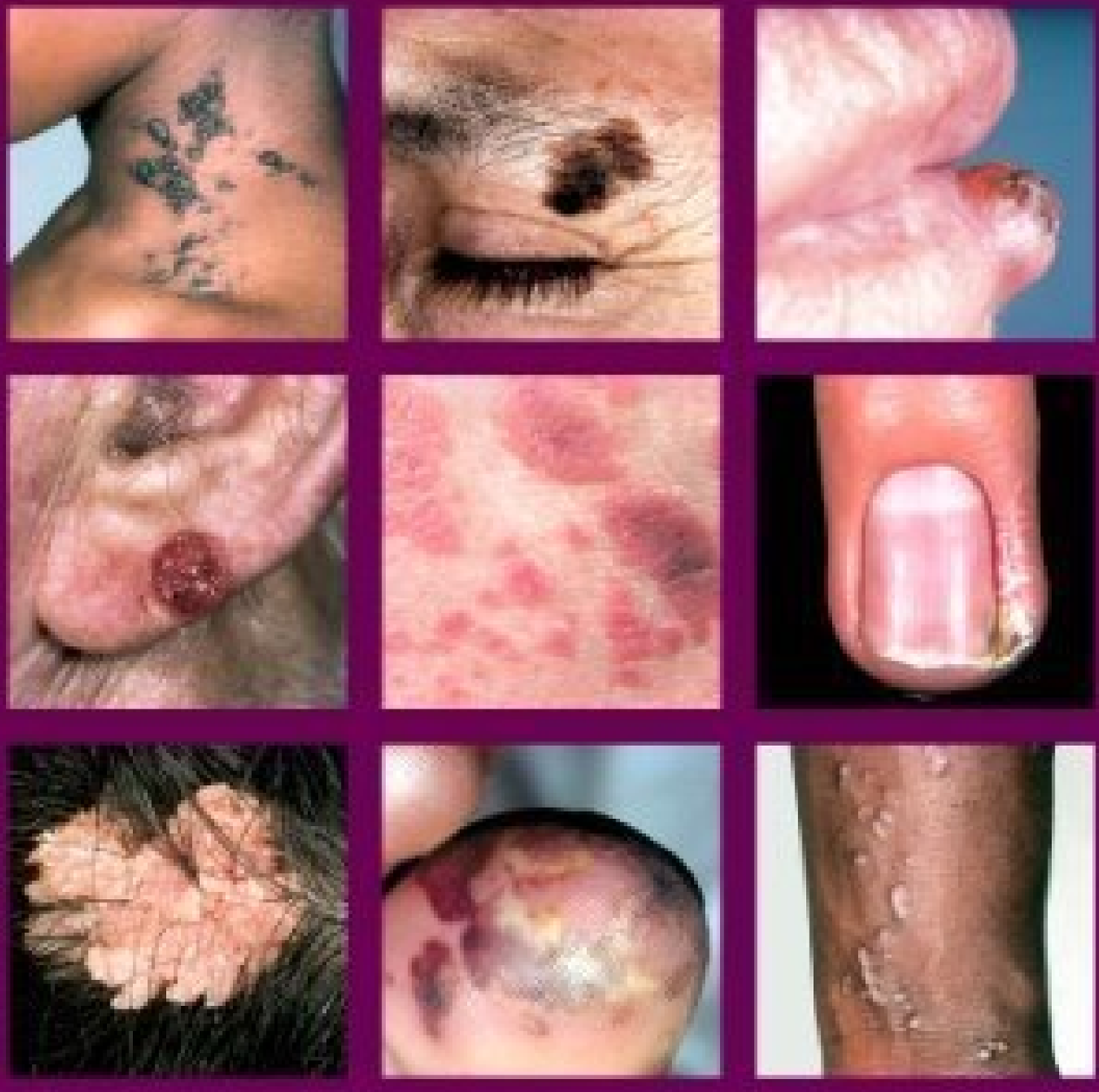
Anthony du Vivier

ATLAS OF CLINICAL DERMATOLOGY

FOURTH EDITION



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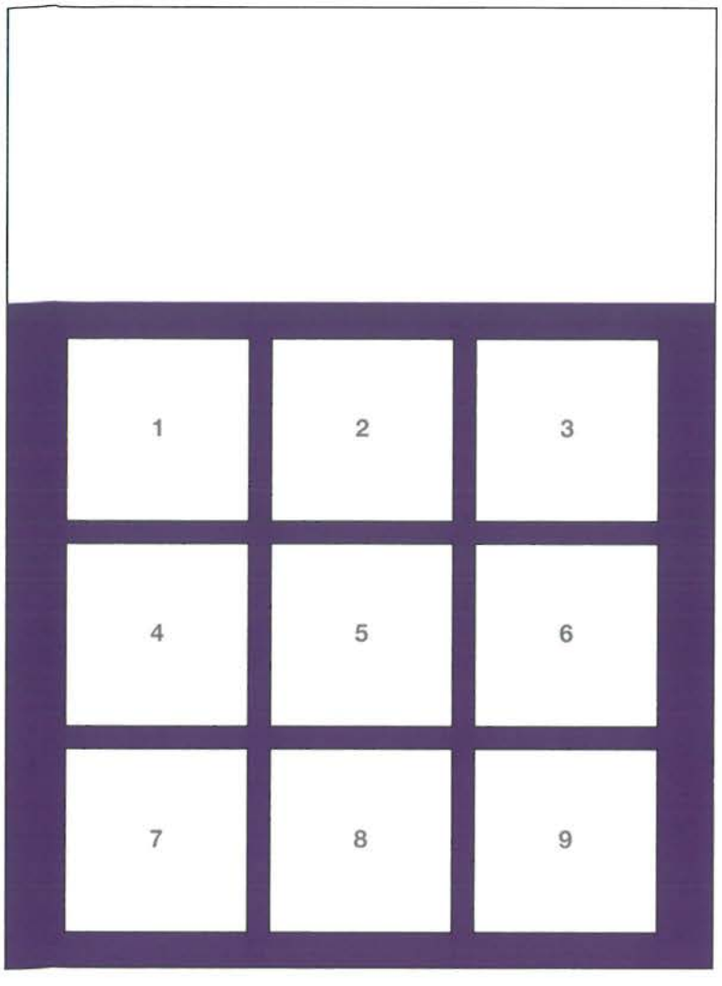
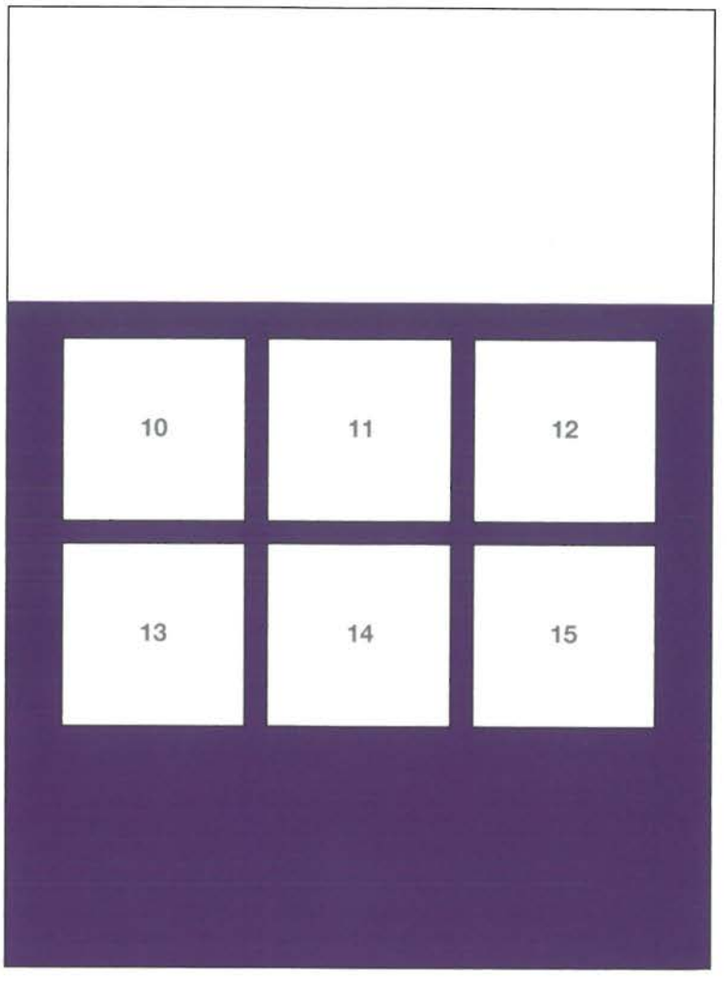
ATLAS OF CLINICAL DERMATOLOGY

Front Cover



1 Epidermal naevus
2 Superficial spreading malignant melanoma
3 Squamous cell carcinoma of the lip
4 Squamous cell carcinoma of the ear lobe
5 Psoriasis
6 Chronic paronychia
7 Naevus sebaceous
8 Subacute bacterial endocarditis
9 Lichen planus

Back Cover



10 Keratoacanthoma
11 Purpura
12 Psoriatic nails
13 Bullous pemphigoid
14 Candidiasis
15 Psoriasis

Endpapers Normal sebaceous glands

Commissioning Editors: Sue Hodgson/Russell Gabbedy
Development Editor: Sharon Nash
Project Managers: Lucy Boon/Caroline Jones
Design: Alan Studholme/Charles Gray/Christian Bilbow
Illustration Manager: Jennifer Rose
Illustrator: Antbits
Page Layout: Marion Stockton
Marketing Manager(s) (UK/USA): Gaynor Jones/Helena Mutak

ATLAS OF CLINICAL DERMATOLOGY

FOURTH EDITION

Anthony du Vivier MD FRCP
Consultant Dermatologist
London, UK

Advisor on Pathology, Fourth Edition
Jon Salisbury BSc MD FRCPath
Consultant Histopathologist and Head of Clinical Service
Department of Histopathology
King's College Hospital
London, UK

Advisor on Pathology, Previous Edition
Phillip H McKee MD FRCPath
Associate Professor of Pathology
Director, Dermatopathology Division
Brigham and Women's Hospital
Boston MA, USA

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This book is dedicated to a special lady, Judith Brett, who is both my lovely wife and favourite doctor.

"Age cannot wither her, nor custom stale
Her infinite variety"

ANTONY AND CLEOPATRA, ACT II, SCENE 2, 243–4.

Preface

The first edition of this Atlas of Clinical Dermatology was a collection of over 1500 colour illustrations of common skin disorders accompanied by a series of essays on their clinical features. It attracted a wider audience than I had anticipated in that it appeared to be of interest to both generalists and dermatologists. I therefore completely revised the text of the second edition with both in mind. Each condition was defined and its aetiology, clinical features and pathology discussed. The management of the common disorders was described in considerably more detail so that the book could be of practical as well as diagnostic value to those in family practice. Rarer disorders were added and illustrated for the benefit of the dermatologist in training. In the third edition, the chapters on cutaneous manifestations of reactive, developmental and systemic processes were greatly expanded to broaden the appeal for the internist and dermatologist, and those on naevi, malignant melanoma and other skin tumours were increased in size for the surgeon. In this edition my publisher, Sue Hodgson, requested that I put the book on a diet and reduce its size. I would not have done it for anyone else but have given in despite one charming New York reviewer comparing the 3rd edition to a 1/3-lb extra lean pastrami sandwich on rye. The number of illustrations however remain at approximately 2400 and many new ones have been added. Where possible, I have tried to compare conditions in both black and white skins.

Acknowledgements for the fourth edition

I have acknowledged in the third edition my mentors and colleagues who have meant so much to me in dermatology. In the final chapters of my career, more house physicians, registrars and consultants have come my way whom I respect and who are now or about to become great dermatologists. They are Genevieve Osborne, Deirdre Buckley, Kate Short, Nuala O'Donaghue, Karen Watson, Jonathan White, Sarah Macfarlane, Claire Martyn Simmonds, Sasha Dhoat, Aileen and Saqib Bashir, Ferina Ishmail, Rishika Sinha, Emma Craythorne, Emma Benton, Sacha Goolamali, Victoria Hogarth, John Ferguson and Sarah Walsh.

Many dermatologists have the privilege of collaborating with physicians from other specialties. My pleasure has been to work with the department of haemato-oncology at King's under the leadership of Professor Ghulam Mufti, the finest scientist and physician I have ever met. Also, Dr Jon Salisbury is our first class pathologist at King's and he has updated some of Dr Philip McKee's excellent work on pathology in Chapters 2 and 9.

Elsevier has been good to me. Sue Hodgson, who commissioned the fourth edition, remains a good friend. Rus Gabbedy has been utterly charming throughout despite my impossibly demanding nature. Sven Pinczewski, Ruth Noble, Lucy Boon, Caroline Jones, Christian Bilbow and Marion Stockton have been wonderful to work with and Sharon Nash who orchestrated the finer details of the project has done her native Manhattan proud.

Finally, I belong to a generation, which cannot survive without an efficient and sympathetic secretary. Annette Norey continues to manage me and my Wimpole Street practice in central London with great aplomb. Monica Braithwaite has taken special care of me and my needs at King's College Hospital. I salute and thank them both.

Acknowledgements from the third edition

I have in the first edition acknowledged the deep gratitude I have to the physicians who taught me dermatology. In particular, without the encouragement and example of Dr Dowling Munro and the late Dr Peter Borrie at St Bartholomew's Hospital, London, I might have missed dermatology and been an unhappy man. Dr Richard Stoughton introduced me to the laboratory side of the subject at the Scripps Clinic and Research Foundation in La Jolla, California, and was generous in opening up the exciting world of American dermatology to me. Since my consultant appointment at King's College Hospital, London, I have been greatly helped by my colleagues Drs Andrew Pembroke, Elisabeth Higgins, Claire Fuller and Daniel Creamer, and I am particularly grateful to them for their forbearance and support whilst I have been writing these books. I have also been particularly fortunate in having a series of excellent registrars to work with at King's who are all now Consultants, but I would like to acknowledge how good they have been to me, and they include Drs Barry Monk, Michèle Clement, Sallie Neill, Jenny Hughes, Olivia Schofield, Stephanie Munn, Pamela Todd, Noreen Cowley, Lindsay Whittam, Karen Harman, Fiona Child, Fiona Keane and Professor Hywel Williams.

The majority of illustrations are, unless otherwise stated, of patients under the care of myself or members of the Dermatology Department at King's College Hospital, London. The photographs have largely been taken by the Medical Illustration Department of King's College Hospital. The rest have come from photographic departments of the hospitals where I trained, viz St Bartholomew's, St Mary's and St John's, London, and my own collection. I particularly wish to thank, therefore, Mr E. Blewitt, Mr D. Tredinnnick, Dr P. Cardew, Mr B. Pyke, Mr E. Sparkes, Mr S. Robertson, Yvonne Bartlett, David Langdon, Lucy Wallace, Margaret Delaney and Alex Dionysiou for the help they have given me over the years.

I am once again indebted to Dr Phillip McKee for his help in providing me with illustrations of the pathology. He is an exceptionally delightful, generous and special man. Other pathologists who have been particularly helpful to me during the course of my work at King's are Dr Jon Salisbury and Dr Debbie Hopster. They have taught me that it is impossible to practise medicine without the backing of the pathologists. Elsevier Health Sciences have provided a team that I have really enjoyed working with. Sue Hodgson has been an outstanding editor. It is not easy to completely revise a textbook and combine this with a busy medical practice. Most editors would have been exasperated as each deadline passed, but her charm, patience and tact cajoled me ultimately into delivery. Quite probably my salvation lay in her happiness at becoming engaged and I can vouch for the fact that her fiancé is a lucky man. Louise Cook stage-managed the early part of the project and Scott Millar was a pleasure to work with as he went through each proof with me late into the evening and orchestrated the production of the final product.

Finally, I would not have been able to write these books in the first place were it not for the skills of two superlative secretaries: Mrs Annette Norey who has run the London practice of my friend Dr Jeremy Gilkes and myself with masterly efficiency and understanding for 30 years and probably knows more about dermatology than most doctors; and Miss Pauline Johnson who ran the skin department at King's for 3 years before she returned to her native Scotland and subsequently found a charming dermatology registrar to marry, Dr Colin Morton, now a Consultant at Falkirk Royal Infirmary. Pauline has retyped the whole of the 3rd Edition at home in Scotland with her mother helping out by babysitting for Penny, Jack and Zoe.

Contents

The dermatological diagnosis 1

The 'spot diagnosis' is a delusion, a belief strongly adhered to by medical students and others despite its obvious deficiencies. It is always impressive to see an experienced dermatologist arrive seemingly instantly at a diagnosis but, just as Sherlock Holmes astounded Dr Watson with observations regarding his life history at their first encounter in the pathology department at St Bartholomew's Hospital, it is a matter of a series of careful observations that result in correct deduction. Just as no one would approach a cardiologist, proffer a radial pulse and expect a diagnosis, so it is not enough to 'glance' at an eruption and expect to make a diagnosis. Surprisingly, this is a common request from colleagues, often in dimly lit corridors – a practice aptly referred to as 'kerbside dermatology'. Like any other branch of medicine, dermatology is a science and diagnosis results from detailed history taking, thorough examination and accurate observation. It is essential that this should take place in consulting rooms with plenty of natural sunlight affording good illumination. A British winter's evening is an anathema for examining the skin.

The teaching of dermatology varies. Some medical schools have excellent training programmes, but others do not teach undergraduates dermatology at all; sometimes the course is optional. This is regrettable, because approximately 20% of consultations in family practice relate to the skin. Even in good centres, medical students will not have enough exposure to the diagnosis and management of skin disorders, for understandably their time is limited. It is, therefore, an excellent idea to spend time as a postgraduate with the local dermatologist. The problem with dermatology is its visibility and the fact that the conditions were codified many years ago in ancient languages that are no longer learnt at school. This nomenclature gives rise to confusion. Nonetheless, with the gradual elucidation of the aetiology of each skin disorder and the major advances being made in therapy, the field is rapidly expanding and has already given rise to specialists within the speciality.

The history

Taking the history of a skin disorder is usually a less prolonged affair than in general medicine. Indeed, sometimes it is more productive to examine the skin early in the consultation to get some idea of the problem and then to proceed to ask the relevant questions.

General Questions

How long has it been present? Some eruptions begin acutely (e.g. drug eruptions), whereas others are more insidious (e.g. pityriasis versicolor).

How does it behave? Some disorders are liable to relapse and remit. For example, a diagnosis of urticaria, which may not be visible on the day of the consultation, may be made from a characteristic history of red, itchy swellings that appear anywhere on the skin and disappear without trace within 24 hours. Factors relating to the relapses may be important (e.g. exposure to bright sunlight may precipitate recurrent attacks of herpes simplex). Some disorders evolve through different stages, classically chickenpox. Others result in crops of lesions (e.g. lymphomatoid papulosis, where the initial red-brown papules become haemorrhagic, crusted, necrotic and heal as scars).

How did it start? In pityriasis rosea, a solitary patch appears before a large number of smaller patches appear all over the torso several days later. The single lesion is known as the herald patch and is an important clue to diagnosis.

What did it look like initially? Some patients are very observant and can record the progress of their condition accurately. For example, in impetigo, they note that it starts as blisters, which quickly break and form crusts. Their descriptions may be very helpful if the rash has temporarily disappeared, as happens with urticaria and herpes simplex. Often, however, if it has disappeared, it is wise to suggest that the patient returns immediately when it recurs and has access to an 'SOS appointment' so that the eruption can be seen at its height.

Is it anywhere else? Patients may complain about something they consider important but disregard a long-standing skin condition elsewhere. Thus, an acute weeping eczema on the face may clearly be the prime problem to the patient, but the cause of it may be autosensitization following an allergic contact sensitivity to a cream used to treat chronic varicose eczema on the leg, which is thought to be totally unrelated by the patient. Therefore, all the skin should be examined. Alternatively, the patient may be unaware of a skin eruption elsewhere on the body or be too embarrassed to mention it because of its location.

What affects it? The patient's view is sometimes invaluable. The patient may suspect the sun, the cat, something at work, 'nerves', her menstrual periods or some tablet or food as the cause of the complaint. Often the patient is correct.

Where do you come from? A knowledge of diseases endemic in various parts of the world is useful: a Vietnamese may have erythema nodosum secondary to tuberculosis; a Philippino leprosy and an African onchocerciasis. Certain racial groups are more prone to disease processes, e.g. a West Indian may have sarcoidosis and a Caucasian skin cancer.

Have you been abroad recently and if so where? Foreign travel exposes the patient to diseases that are uncommon in their country of origin. For example, the insect that bit the patient may have been infected with the protozoan *Leishmania* sp. and Baghdad boil (cutaneous leishmaniasis) can occur in less exotic places such as the Mediterranean.

Symptoms

Does it itch? Some disorders always itch (e.g. scabies), and the very intensity of the complaint may suggest the diagnosis. The rash of secondary syphilis virtually never itches. Psoriasis and pityriasis rosea are quixotic and may or may not itch.

Is it painful? Few dermatological disorders are acutely painful, but the classic example is that of herpes zoster. Pain dominates the history (you do not need to ask) and the suspicion is confirmed when the unilateral vesicular eruption is revealed on examination.

Is it sore? An eruption such as eczema or psoriasis may become sore when it dries out and cracks, particularly in cold climatic conditions.

Does it burn? Few skin disorders burn; patients thus afflicted volunteer the symptom. The rash of erythropoietic protoporphyria burns and the localization of the symptoms to the light-exposed skin might suggest the diagnosis. If burning affects the mouth, genitalia or face without any visible physical signs, it is often a psychosomatic symptom.

Associated Symptoms

The skin disorder may follow a prior illness. For example, a streptococcal infection frequently precedes guttate psoriasis, erythema nodosum and Henoch–Schönlein purpura. The rash, however, may result from a drug given to treat an illness (e.g. the unfortunate prescription of ampicillin for a sore throat in a young adult with unsuspected infectious mononucleosis) or the skin rash may be one of the presenting features of a systemic disease (e.g. sarcoidosis or lupus erythematosus). It is important, therefore, to enquire about symptoms relating to other systems.

Past History of Skin and Related Disorders

A past history of skin and related disorders is often relevant. This is particularly relevant to young women who have had eczema in childhood; they may develop it again on the hands, either as a result of an occupation such as hairdressing or when looking after small children. Alternatively, a patient may develop late-onset eczema, with the clue to its cause being other symptoms of atopy such as hay fever or asthma in childhood.

Family History

Many common skin diseases are inherited, including psoriasis, ichthyosis and eczema. Sometimes the patient denies a family history at the initial consultation, but this is usually simply because no one has ever mentioned a skin disorder to them previously. Subsequent questioning at a family reunion may provide information of which the patient was unaware.

Past Medical History

A previous illness may help to explain the present complaint. Thus, a difficult and protracted labour may be responsible for a diffuse loss of hair 3 months later (telogen effluvium). A chronic illness (e.g. diabetes mellitus) might make the patient more prone to a chronic candidal paronychia.

Previous and Current Drug Therapy

Clearly, a systemic agent may be the cause of a skin disorder, such as a phenothiazine, a diuretic or a tetracycline for a phototoxic eruption or an antibiotic for a morbilliform rash. The patient may recall a previous allergic reaction but be unaware that the present medication is a related product. Drugs such as systemic steroids are immunosuppressive and may make a patient more prone to infections with commensals (e.g. pityriasis versicolor). Family practitioners are very good at listing the oral agents in their letters of referral to the specialist, but sometimes topical remedies get forgotten. These are important because the therapy prescribed may have been correct and yet ineffective in a particular patient and there is no point in represcribing the drug.

Conversely, the prescribed therapy may be making the disease worse and patients will often note this. This may be because the condition has been misdiagnosed (e.g. when tinea or rosacea is diagnosed as eczema and treated with steroids). Moreover, a complication may have occurred in a steroid-responsive dermatosis so that the steroid is no longer appropriate, as when molluscum contagiosum is superimposed upon eczema. Equally, the patient may have become sensitized to the prescribed agent; this occurs particularly when varicose eczema is treated with agents containing topical antibiotics (e.g. neomycin). Another possibility is that the patient may have developed an irritant reaction to a drug such as dithranol, which is used in the treatment of psoriasis. Some races like to lighten their skin colour or bleach pigmented marks on their skin; noxious chemicals including steroids can be obtained from the local market for this purpose. Finally, alcohol is a drug that is often forgotten. In sufficient quantity it tends to exacerbate psoriasis, discoid eczema and rosacea.

Occupation

Just as coal miners are prone to pneumoconiosis, so certain occupations predispose to skin disease. Percival Pott established the link between carcinoma of the scrotal skin and previous exposure to soot in men who had cleaned chimneys as children. Dubreuilh pinpointed the relationship of malignant melanoma on the face of the workers in the vineyards of Bordeaux to exposure to ultraviolet light. Exposure to contact allergens at work is often suspected because the patient gets better at the weekend or when away on holiday. This might be so with dermatitis secondary to chromate exposure, which is common in builders who work with cement. Probably the most common occupational skin diseases seen in dermatology are primary irritant dermatitis and chronic candidal paronychia in housewives, nurses and barworkers, caused by the frequent exposure of the hands to water.

Social History

The situation at home is of great importance. Other members of the household may be itching, which may suggest a diagnosis of scabies. The family cat and its cohabitants may be the source of the insect bites. Psychological factors are as critical in skin disorders as they are in other branches of medicine. The unhappiness of a marriage or relationship, guilt (religious or otherwise), success or failure at work or difficult children, all take their toll and it is difficult to manage any chronic skin disease without a knowledge of these problems. It may be that the whole eruption is caused by the patient (dermatitis artefacta) or that the failure to recover is because the patient does not wish to get better, preferring instead to evoke sympathy and attention because of the condition. Sometimes patients simply overreact to a minor skin condition, the presence of which is the final straw in a lifestyle that has got completely out of control.

Effect of the Disease on the Patient

The patient is likely to be anxious regarding the nature of the disorder. Infectivity and malignant disease are the commonest fears so that informed reassurance to the contrary, if appropriate, may be of enormous help to the patient. Sometimes the concern may amount to a phobia, for example regarding herpes simplex in the 1970s and now acquired immunodeficiency syndrome (AIDS); consequently, further psychological help will be required.

The functional effects are important. An eruption on the feet may make it difficult to walk, and one on the hands difficult to work. The appearance of the condition on exposed parts may make the patient feel leprous and ostracized. Extraordinary variations in patients' reactions occur. Some will put up with a considerable degree of psoriasis and yet others will be disgusted and aggrieved by minimal disease. All these factors have to be considered when deciding how aggressively to treat a disorder.

The examination

Ideally the whole patient should be examined. This is not usually necessary for warts, but it is for most conditions. A basal cell carcinoma of the face may be accompanied by other solar-induced malignancies elsewhere, such as a malignant melanoma on the back, an area that the patient cannot easily see; an eruption on one hand may be explained by the spread of tinea from the toenails and feet, and a condition of both hands that is unresponsive to therapy may be due to the psoriasis revealed by examining the elbows and scalp.

It is sensible to examine the skin in an orderly manner, starting at the hands, so as not to miss the burrows of scabies, and then proceeding up the arms, face, trunk and so on. The hair, nails and mouth should not be forgotten.

It is important also to palpate the lesions. Disorders involving primarily the dermis (e.g. sarcoidosis or lymphoma) can be distinguished from those affecting the epidermis (e.g. eczema) because they are palpable. Patients are reassured by a thorough general medical examination. They are relieved that the doctor is prepared to feel their skin because it helps to dispel the fear that their condition makes them 'untouchable'. Finally, the

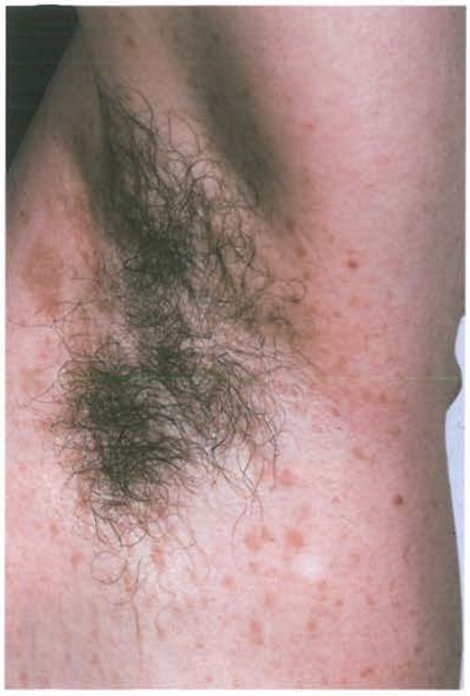

Fig. 1.1 Macules. A macule is a flat lesion less than 1 cm in diameter. Many freckle-like macules are present in the axilla. A larger café-au-lait patch gives away the diagnosis of *neurofibromatosis*.

skin is a part of the whole, and a full general medical examination may be necessary if the patient appears ill or if the cutaneous signs give rise to suspicions of a systemic process.

Vocabulary

Just as râles, rhonchi and bronchial breathing are physical signs that, once elicited, contribute to a pulmonary diagnosis, so there is a dermatological vocabulary that must be grasped to ascertain the nature of a skin disorder. The main types of lesion may be defined as follows.

Primary Lesions

Macule A macule is a circumscribed flat alteration in the colour of the skin which is less than 1 cm in diameter (Figs 1.1 and 1.2). It may be variously coloured, for example pink or red owing to vasodilatation and mild inflammatory changes; purple (Fig. 1.3) or yellow–brown from blood or haemosiderin; or brown, black, pale or white owing to a disturbance in melanin synthesis.

Papule A papule is a circumscribed palpable elevation of the skin less than 1 cm in diameter (others define it as less than 0.5 cm in diameter). A papule may be epidermal (Fig. 1.4), dermal or both in origin. For example, the papules of warts or mollusca contagiosa (Fig. 1.5) are caused by viral parasitosis of epithelial cells, leading to epidermal hyperplasia, whereas

Fig. 1.2 Macules. These hypopigmented macules vary in size and have enlarged by confluence. They are due to a superficial yeast infection, *pityriasis versicolor*.

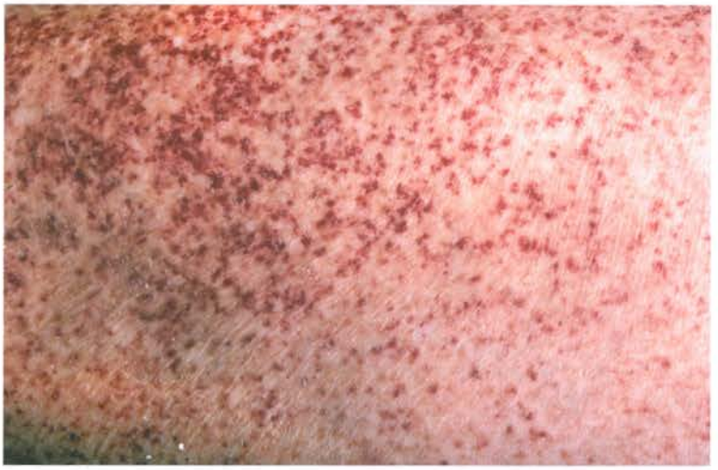

Fig. 1.3 Macules. These tiny macules are purple in colour and do not blanch with pressure. They are known as petechiae. Thrombocytopenia and *vasculitis* are common causes.

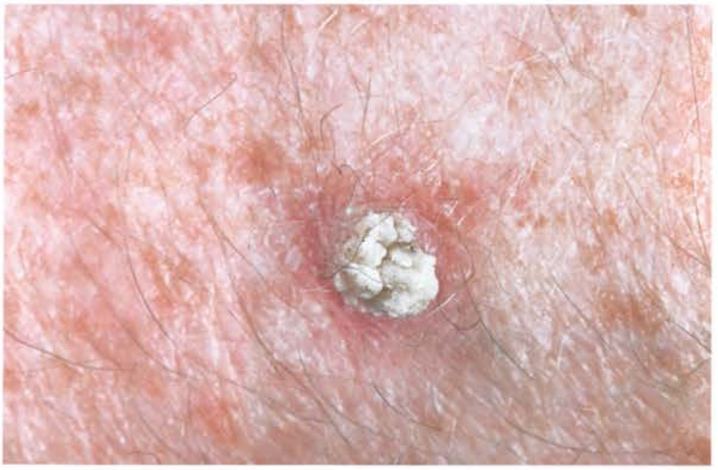

Fig. 1.4 Papule. A papule is a circumscribed palpable elevation of the skin less than 1 cm in diameter. This red papule with an adherent scale is a *solar keratosis*.

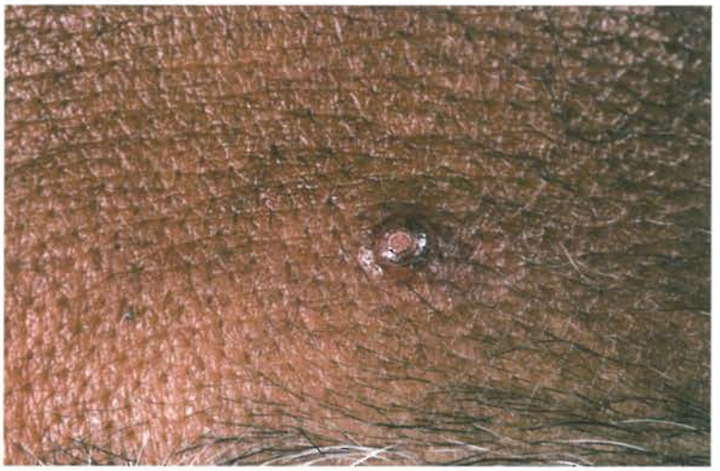

Fig. 1.5 Papule. This more or less flesh coloured papule with central dimpling of the surface is caused by *molluscum contagiosum*. Lesions on the face in adults are suggestive of immunodeficiency.

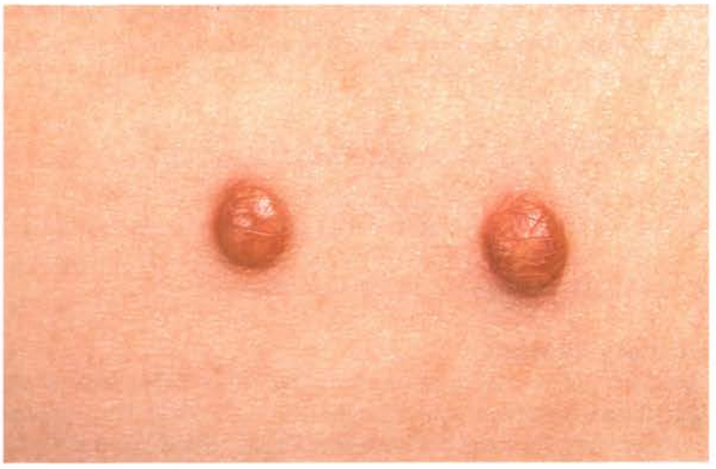

Fig. 1.6 Papules. These lesions are yellow in colour, which suggests the diagnosis of hyperlipidaemia. They are called *xanthomas*.

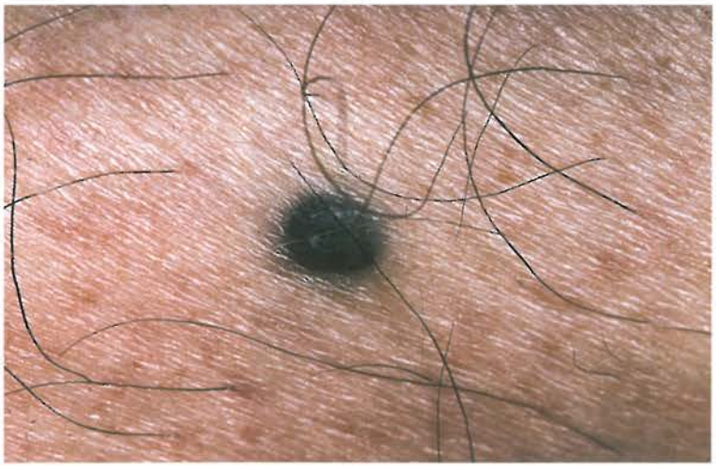

Fig. 1.7 Papule. The bluish hue of this solitary lesion is striking and serves to distinguish it from a malignant melanoma. It is a benign *blue naevus*, often found on the face or back of the hands.

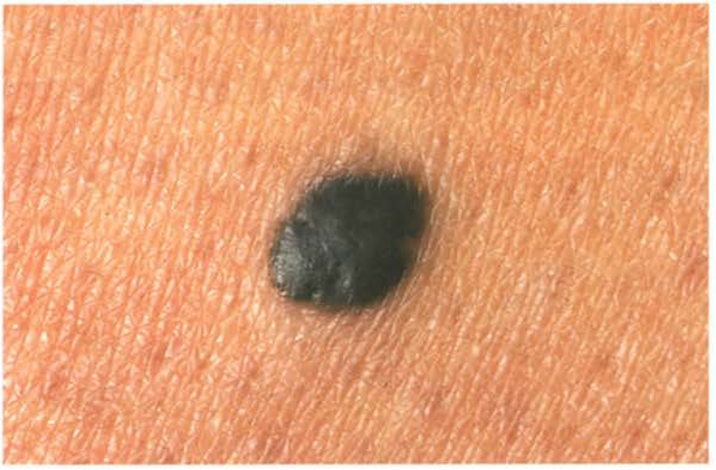

Fig. 1.8 Papule. The history of gradual increase in size and the black colour of this solitary papule suggested the diagnosis of a vertical growth phase invasive *malignant melanoma*.

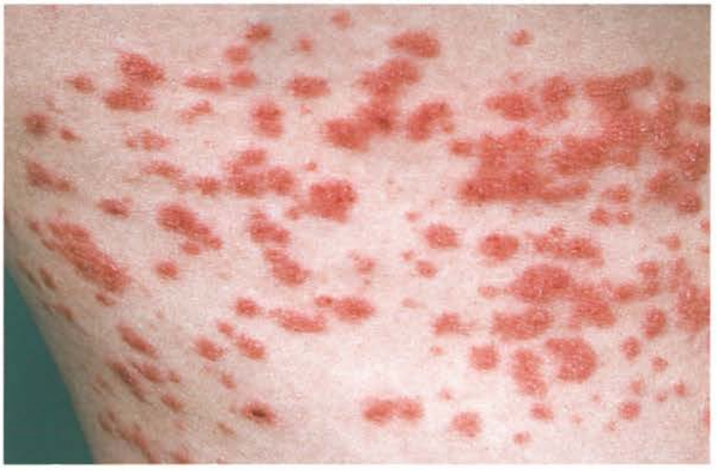

Fig. 1.9 Papules. The deep red colour of these papules is distinctive. Some of the papules have become confluent and formed plaques. This is *psoriasis*.

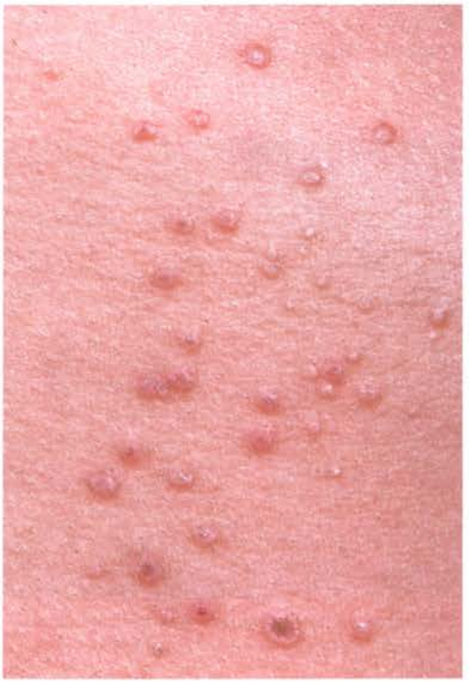

Fig. 1.10 Papules. The purple colour of these papules is distinctive. Their surface is also flat-topped and shiny, permitting a diagnosis of *lichen planus*.

Fig. 1.11 Papules. These papules are flat surfaced and somewhat shiny. Older lesions show hyperpimentation following damage to the basal cell layer of the epidermis by *lichen planus*. The purple colour may be difficult to discern in a black skin.

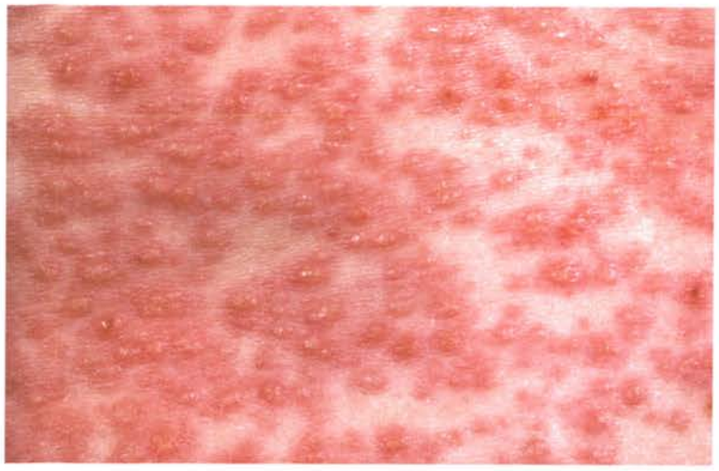

Fig. 1.12 Papules. These papules are red and oedematous and have become confluent with erythematous macules (hence 'maculopapular'). It began ten days after starting a course of ampicillin.

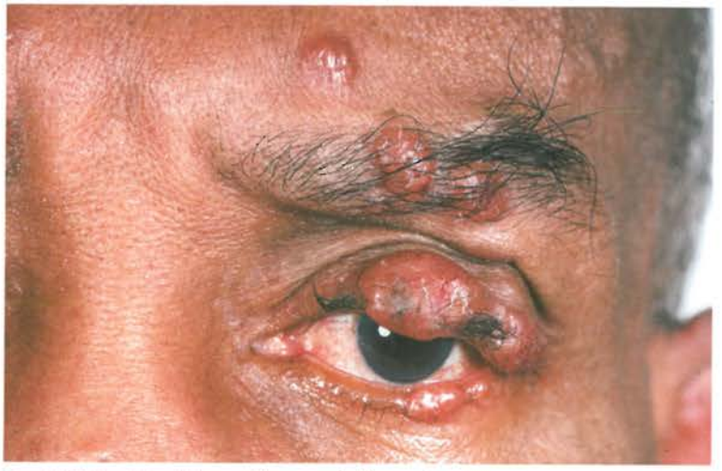

Fig. 1.13 Nodules. The nodules are of different sizes and shapes. The involvement of the eyelids is very characterstic of sarcoidosis. Many skin disorders have a predilection for certain sites on the body.

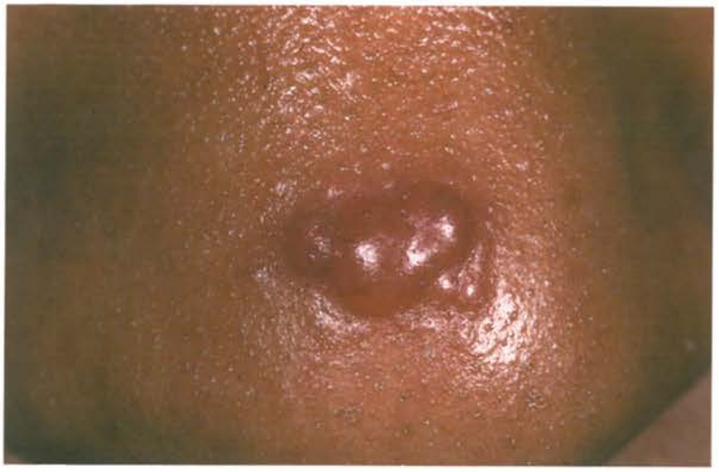

Fig. 1.14 Nodule. This nodule also on the nose of a West Indian is lobulated and red brown in colour, but has an entirely different explanation from the nodule depicted in Fig. 1.13. It was due to *sarcoidosis*.

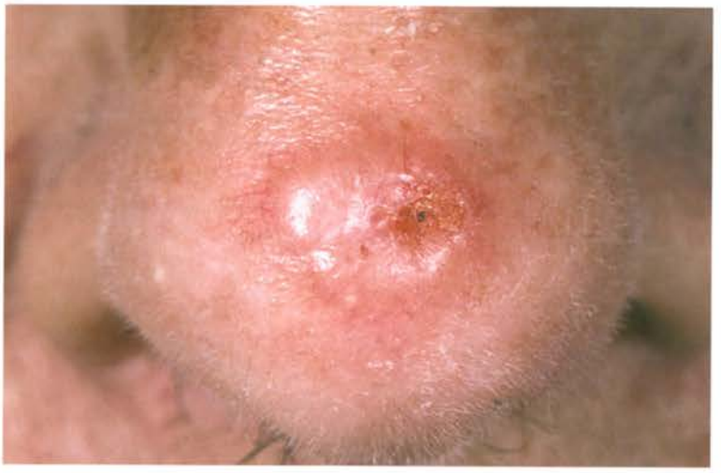

Fig. 1.15 Nodule. This raised circumscribed palpable mass on the nose was greater than 1 cm in diameter and pearly in colour. It bled occasionally and was a *basal cell carcinoma*.

those in which the dermis is involved may be due to deposits of lipid (Fig. 1.6) or collections of benign (Fig. 1.7) or malignant cells (Fig. 1.8). The papules of skin eruptions such as eczema are caused by epidermal oedema (spongiosis), of psoriasis (Fig. 1.9) by increased epidermal cell turnover, and of lichen planus (Figs 1.10 and 1.11) by a lymphocytic infiltrate which involves the upper dermis and epidermal–dermal junction. In a morbilliform drug eruption, there is a mild dermal perivascular infiltrate which gives rise to oedematous lesions clinically (Fig. 1.12).

Nodule A circumscribed palpable mass larger than 1 cm in diameter. The epidermis plus dermis, dermis plus subcutis or subcutis alone may be involved. The lesion may clearly evolve from a papule. The causes are legion but include an infiltrate of granulomas (e.g. sarcoid; Figs 1.13 and 1.14), benign (e.g. a dermatofibroma) or neoplastic cells (e.g. a basal cell carcinoma; Fig. 1.15, keratoacanthoma or squamous cell carcinoma, Fig. 1.16),

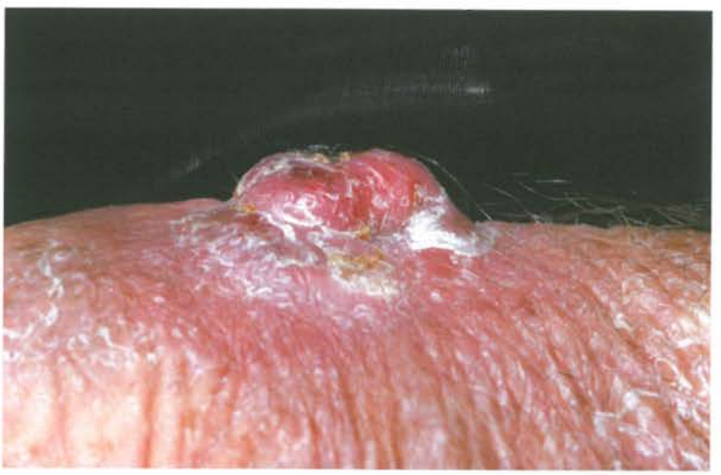

Fig. 1.16 Nodule. There is a red lobular mass arising from a plaque on sundamaged atrophic skin. This is a *squamous cell carcinoma*.

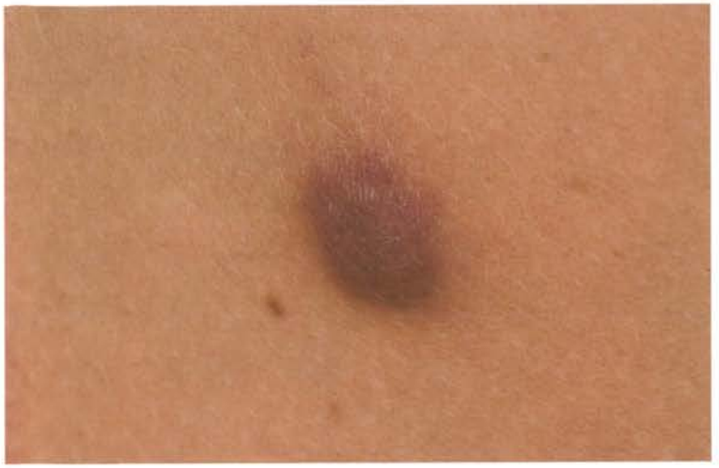

Fig. 1.17 Nodule. This nodule (or tumour) is purple in colour, which is very suggestive of *Kaposi's sarcoma*. Biopsy establishes the diagnosis. Colour is an important physical sign in dermatology.

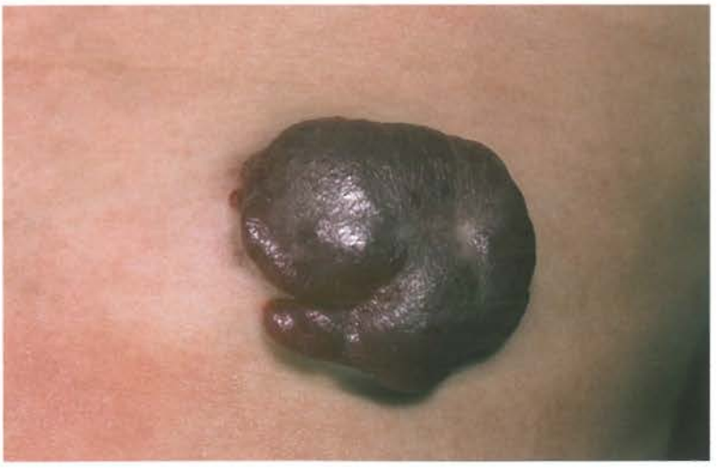

Fig. 1.18 Nodule. This large red and purple lobulated mass grew rapidly shortly after birth and was a *haemangioma*.

inflammation (e.g. erythema nodosum), Kaposi's sarcoma (Fig. 1.17), haemangioma (Fig. 1.18) or lymphoma.

Patch A flat lesion greater than 1 cm in diameter (i.e. a large macule) is known as a patch (Fig. 1.19).

Plaque A slightly raised lesion greater than 1 cm in diameter (Fig. 1.20). Colour (Figs 1.21, 1.22, 1.23 and 1.24), the presence or absence of scaling (Fig. 1.25), changes on the surface (Fig. 1.26) and the shape (Fig. 1.27) may assist in diagnosis. It may be formed by an extension or coalescence of papules, as in psoriasis or granuloma annulare.

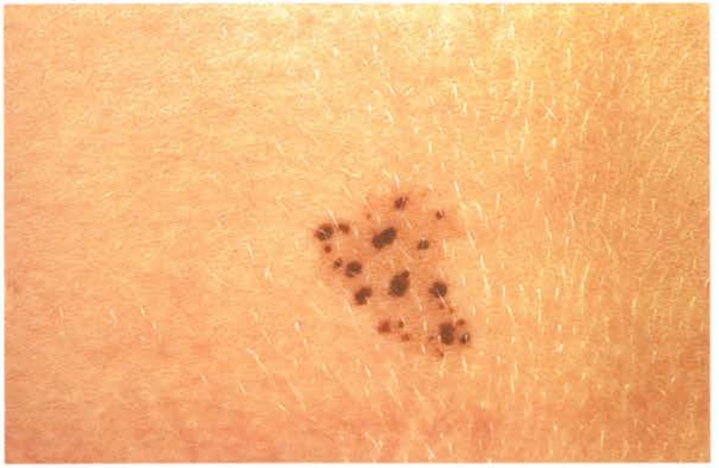

Fig. 1.19 Patch. A patch is a flat lesion greater than 1 cm in diameter. There are small pigmented macules within a light-brown (café-au-lait) patch. This congenital melanocytic lesion is known as *naevus spilus*.

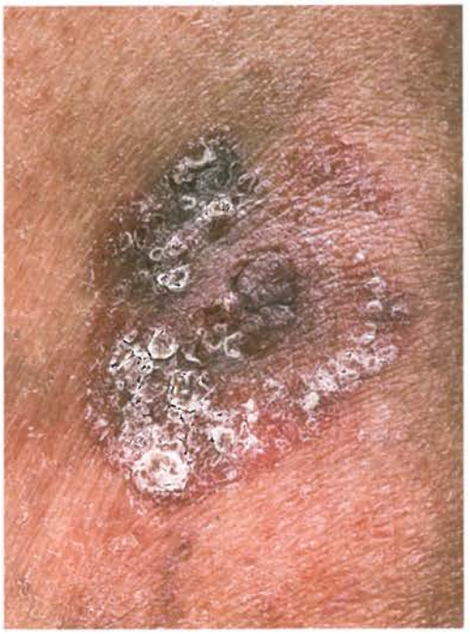

Fig. 1.20 Plaque. Although psoriasiform, this well-defined red plaque was solitary. It was an *intraepidermal carcinoma*.

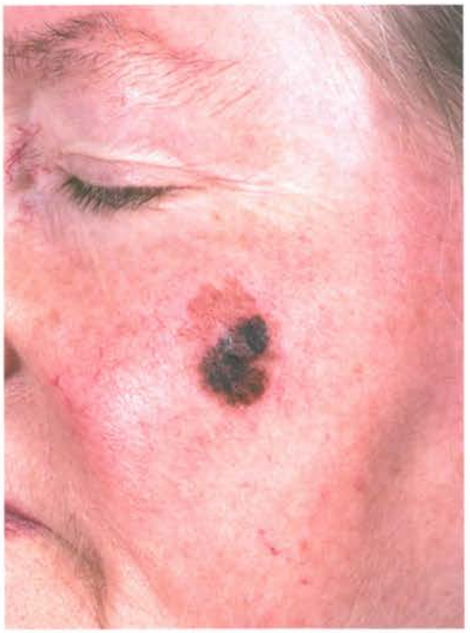

Fig. 1.21 Plaque. The multiplicity of pigmented colours in this *lentigo maligna melanoma* help to clinch the diagnosis. It has evolved from a patch (tan colour) to a plaque (blacker area).

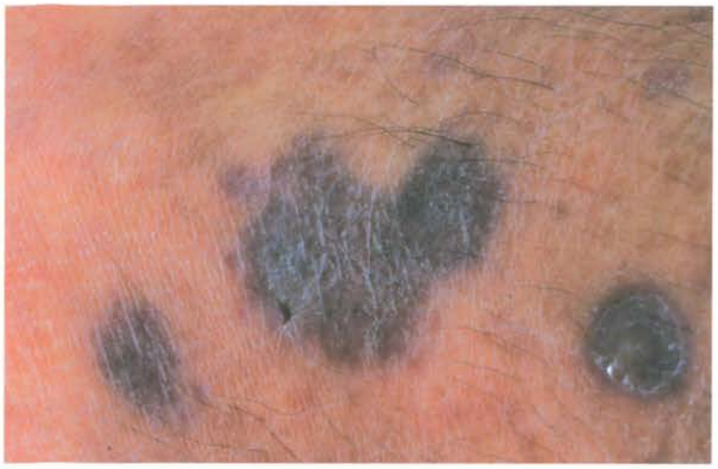

Fig. 1.22 Plaque. These purple raised lesions have evolved from confluence of papules of *lichen planus*. This colour is the characteristic appearance in Asian skin.

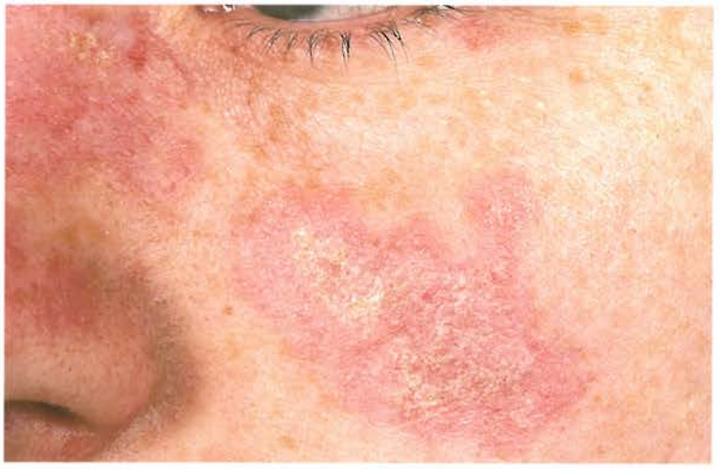

Fig. 1.23 Plaque. The mauve colour and tenacious scale are characteristic of *lupus erythematosus*. Light-exposed areas such as the cheeks are particularly affected.

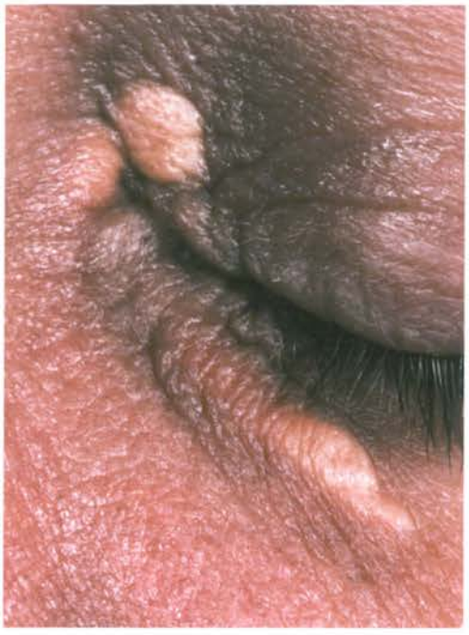

Fig. 1.24 Plaque. This palpable lesion has no surface change, which would suggest that the pathology is in the dermis. The yellow colour and position around the eye is characteristic of *xanthelasma*.

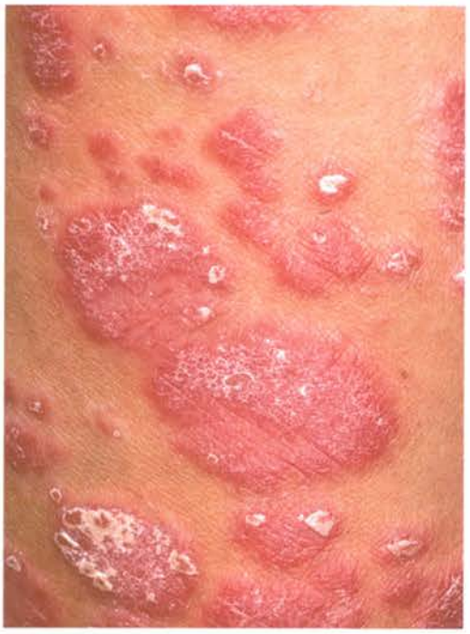

Fig. 1.25 Plaque. Small papules are present but most have enlarged to form well-defined plaques. The deep red colour and thick white scale are typical of *psoriasis*.

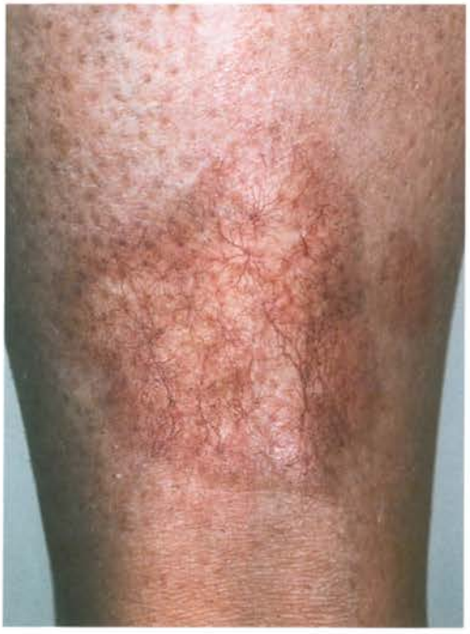

Fig. 1.26 Plaque. A plaque is a raised lesion greater than 1 cm in diameter. The yellow-orange centre and telangiectasia with a mauve edge is typical of *necrobiosis lipoidica*.

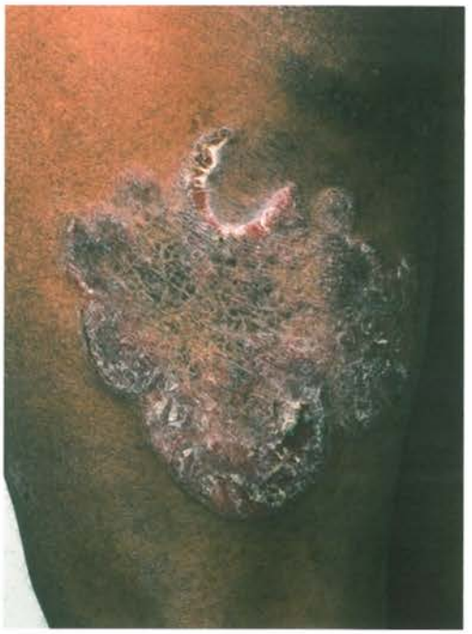

Fig. 1.27 Plaque. This has a well-defined and in some areas indented margin. It is flat in some parts and thickened in others. Biopsy showed it to be *mycosis fungoides*.

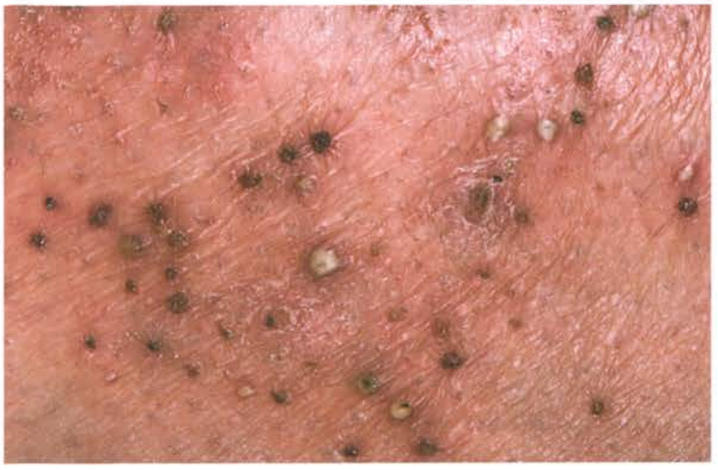

Fig. 1.28 Pustule. A pustule is a raised lesion less than 0.5 cm in diameter containing yellow fluid, which may be infected or sterile. Culture of the pustule in the centre of the picture grew *S. aureus*. This is *folliculitis*.

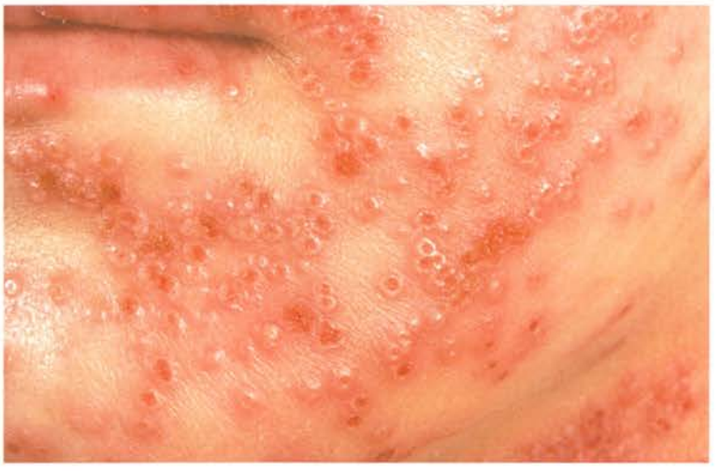

Fig. 1.29 Vesicle. A vesicle is a raised lesion less than 0.5 cm in diameter containing clear fluid. These are umbilicated on the surface. The patient had extensive herpes simplex and eczema (*eczema herpeticum*).

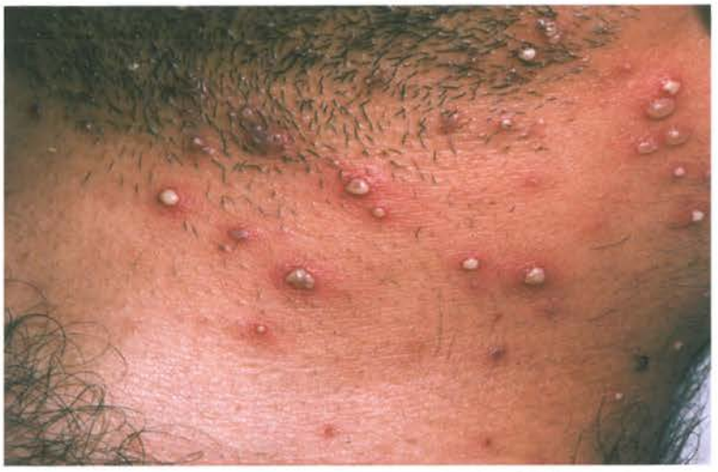

Fig. 1.30 Vesiculopustules. Vesicles initially contain clear fluid but may become turbid or purulent, as in this patient with *chickenpox*. Note the characteristic erythema around the lesions.

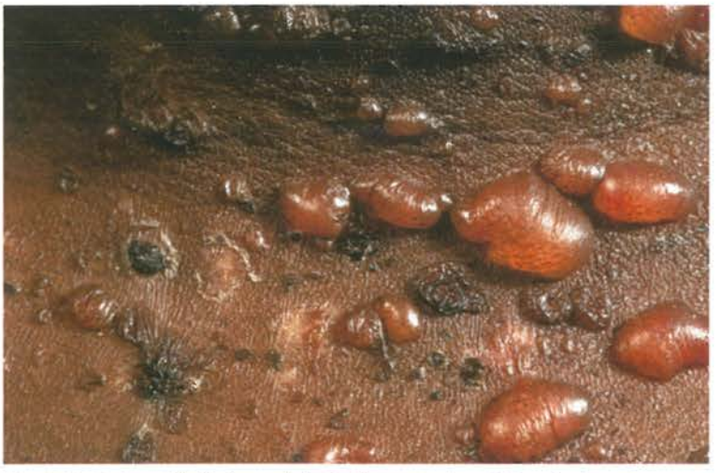

Fig. 1.31 Bullae. A bulla is a vesicle that is greater than 0.5 cm in diameter. Multiple tense fluid-containing blisters are present. Immunofluorescence showed that this child had *bullous lupus erythematosus*.

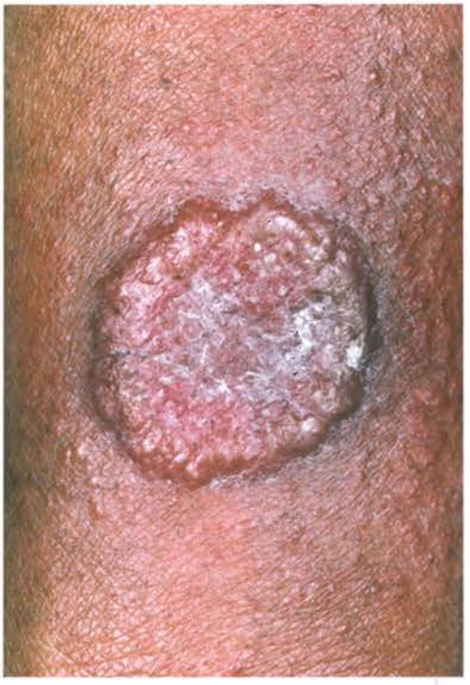

Fig. 1.32 Blistering. Only the edge of this lesion is bullous. The shape is annular and the central area is raised, red and scaling. This was bullous *tinea corporis* caused by *Trichophyton tonsurans*.

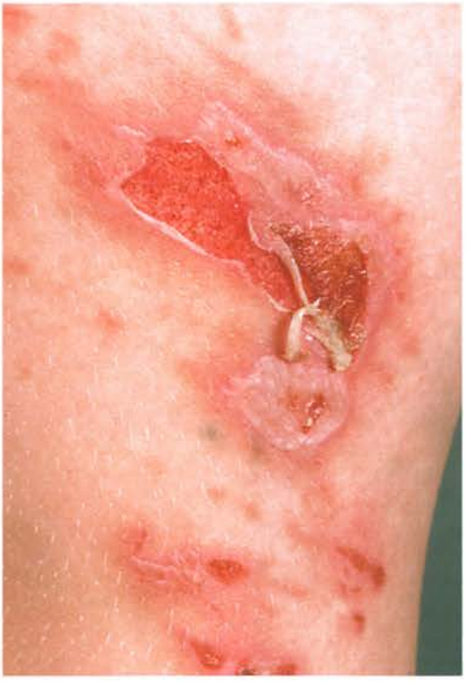

Fig. 1.33 Blistering. The roof of the blisters has been broken in places, resulting in raw, denuded, eroded skin. This linear streaky arrangement was the result of an interaction between a photosensitizer (a psoralen in a plant) and sunlight (*phytophotodermatitis*).

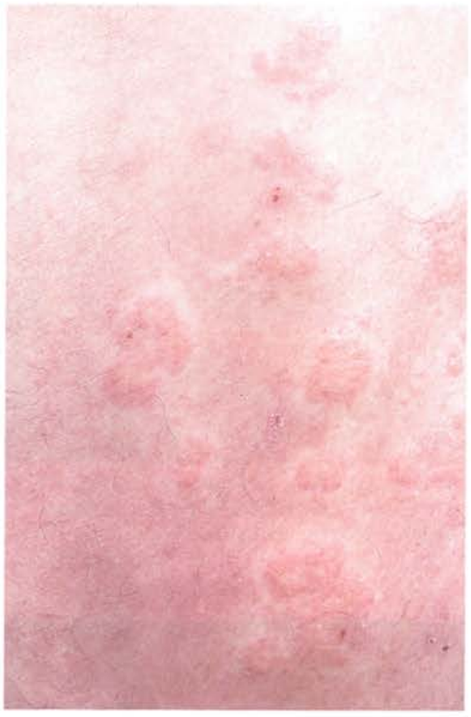

Fig. 1.34 Wheals. A wheal is a transient, itchy, pink swelling that disappears without trace. Wheals often have central swelling of varying size and shape. This patient has *urticaria*.

Fig. 1.35 Wheals. The oedema and varying sizes and shapes of *urticaria* are well shown but the pinkness may be difficult to see in a black skin.

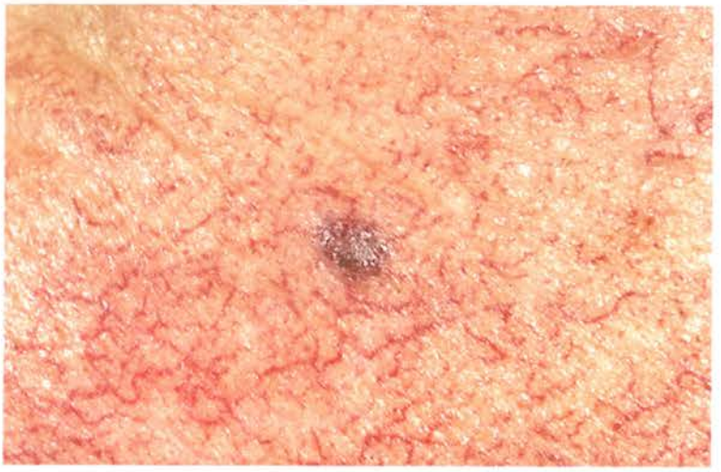

Fig. 1.36 Telangiectasia. A term used to describe finely dilated capillaries. There is also a central red papule (haemangioma). These changes were on the cheeks and were secondary to weathering and *alcohol misuse*.

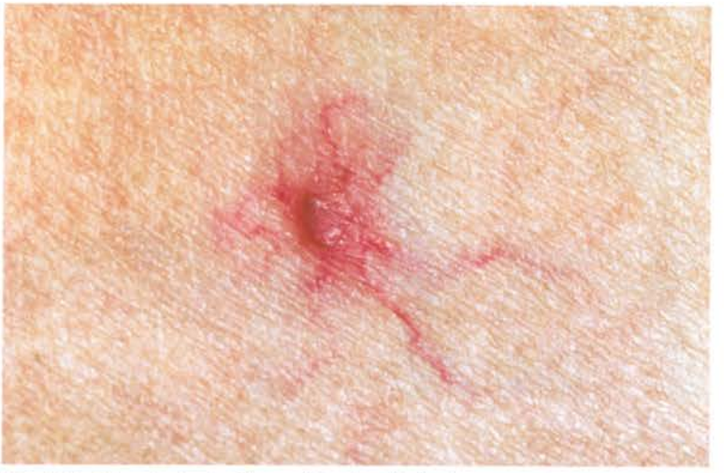

Fig. 1.37 Telangiectasia. These dilated capillaries have spread out from a central red papule, simulating the configuration of a spider, hence its name, *spider naevus*.

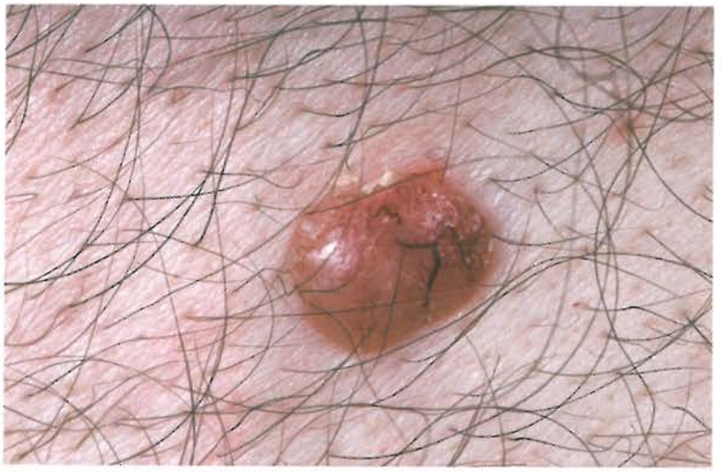

Fig. 1.38 Telangiectasia. This nodule has a pearly colour and a characteristic telangiectatic surface. It is a *cystic basal cell carcinoma*.

Pustule A pustule is a raised lesion less than 0.5 cm in diameter containing yellow fluid, which may be sterile as in acne or pustular psoriasis, or infected (Fig. 1.28).

Vesicle A vesicle is a raised lesion less than 0.5 cm in diameter containing clear fluid (Figs 1.29 and 1.30).

Bulla (blister) A vesicle that is greater than 0.5 cm in diameter is known as a bulla (Fig. 1.31). Insect bites are one of the most common causes of blisters but they also occur in bullous pemphigoid, impetigo, tinea (Fig. 1.32) and phytophotodermatitis (Fig. 1.33).

Wheal A wheal is a transient, itchy, pink or red swelling of the skin, often with central pallor (Fig. 1.34). Wheals can be of various shapes and sizes (Fig. 1.35).

Telangiectasia Visible dilated capillaries (Figs 1.36, 1.37 and 1.38).

Secondary Lesions

Crust A dried exudate, which may have been serous, purulent (Fig. 1.39) or haemorrhagic.

Excoriation A haemorrhagic excavation of the skin resulting from scratching. It may be shallow or deep, linear (Fig. 1.40) or discrete (punctate) (Fig. 1.41).

Lichenification Thickening of the skin with exaggeration of the skin creases (Fig. 1.42).

Lichenoid This term describes lesions that have been scratched and rubbed, giving rise to flat-topped papules similar to those in lichen planus. The appearance is common in eczema in black skins (Fig. 1.43).

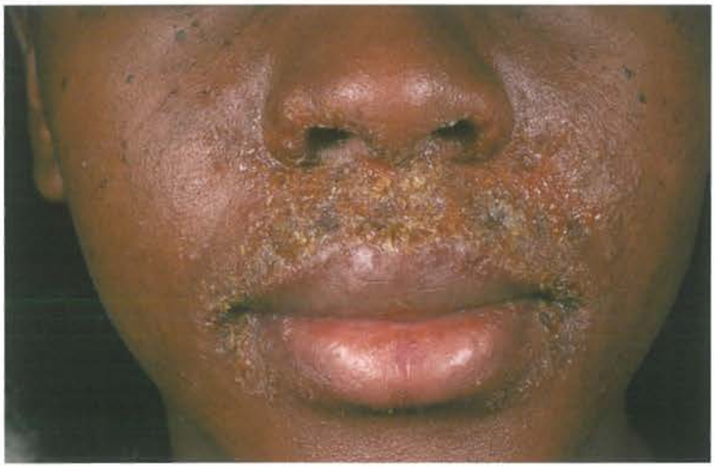

Fig. 1.39 Crust. A crust is a dried exudate, which may have been serous, purulent or haemorrhagic initially. The yellow purulent material visible below the nose has dried in other areas becoming crusted. This is *impetigo*.

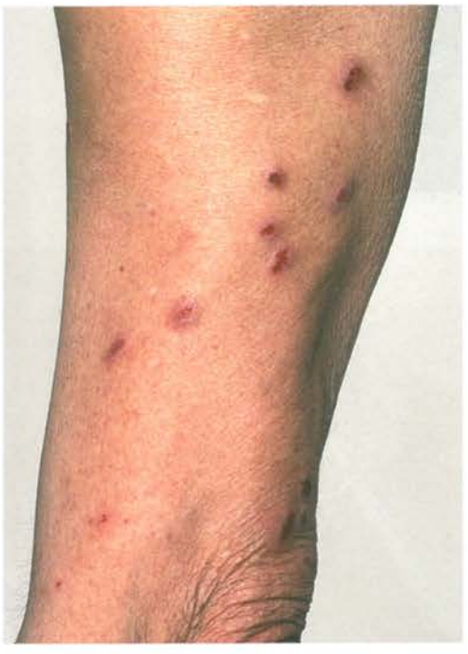

Fig. 1.40 Excoriation. An excoriation is a haemorrhagic excavation resulting from scratching. These discrete scratch marks occurred in a patient who was itching from *primary biliary cirrhosis*.

Fig. 1.41 Excoriation. These haemorrhagic excavations have resulted from interference with the skin. The linear configuration of the lesions is suggestive of *artefactual* disease.

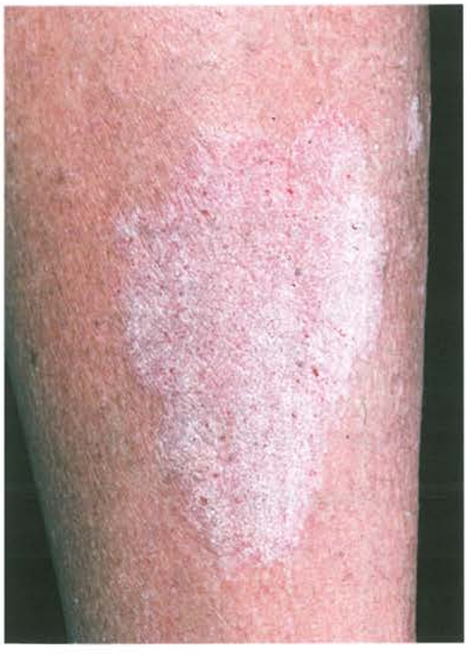

Fig. 1.42 Lichenification. This is a thickening of the skin with exaggeration of the skin creases in a criss-cross manner; it results from continual rubbing and scratching. This is *lichen simplex*.

Fig. 1.43 Lichenoid papules. In black skin in particular, rubbing and scratching of *eczematous skin* may result in flat-topped itchy papules that simulate lichen planus, hence the term lichenoid.

Necrosis Death, or necrosis, of skin tissue is usually black in colour (Fig. 1.44). Gangrene (Fig. 1.45) is a form of necrosis. Necrolysis is a superficial necrosis with shedding of the skin (Fig. 1.46).

Scar The final stage of healing of a destructive process (disease or injury) that has involved the deeper dermis results in a white, smooth, firm, shiny lesion (Fig. 1.47). There are various forms (Fig. 1.48).

Scaling A scale is a flat plate (lamella) or flake of stratum corneum.

Exfoliation Splitting off of the stratum corneum in fine scales or sheets.

Fissure This is a linear split or gap in the skin surface.

Keratoderma A horny thickening of the keratin layer of the skin. It may occur in a congenital abnormality of keratin formation or as a result of simple mechanical stimulation.

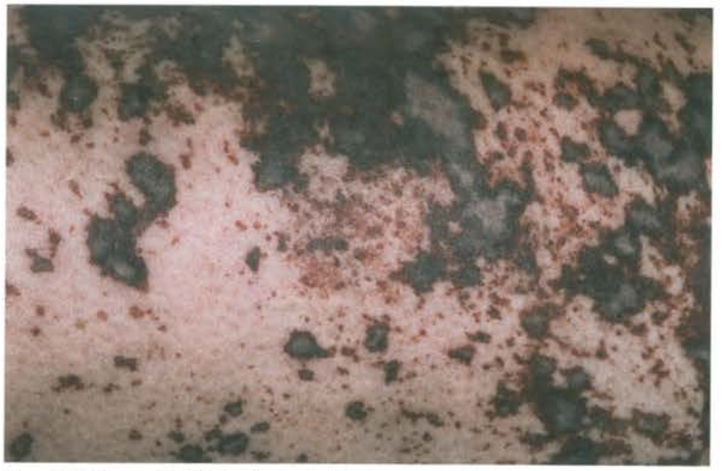

Fig. 1.44 Necrosis. This is death of skin tissue and is usually black in colour. These haemorrhagic purpuric lesions are necrotic and are caused by disseminated intravascular coagulation, due to *meningococcal septicaemia*.

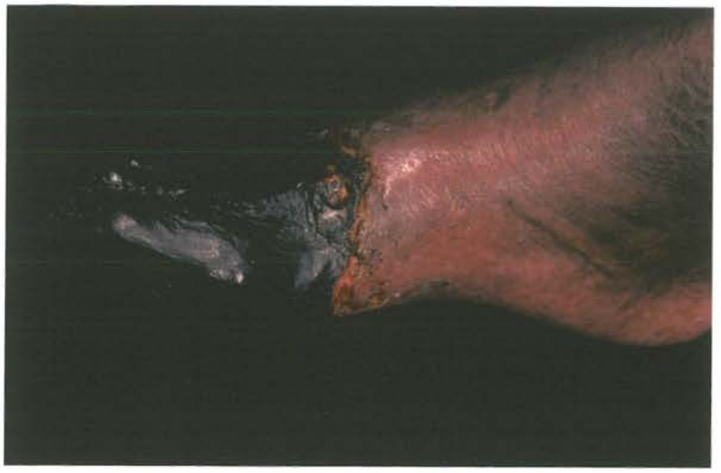

Fig. 1.45 Gangrene. This is death of skin and other tissue secondary to loss of the blood supply. There is profound necrosis. This patient had *arteriosclerosis* secondary to diabetes mellitus.

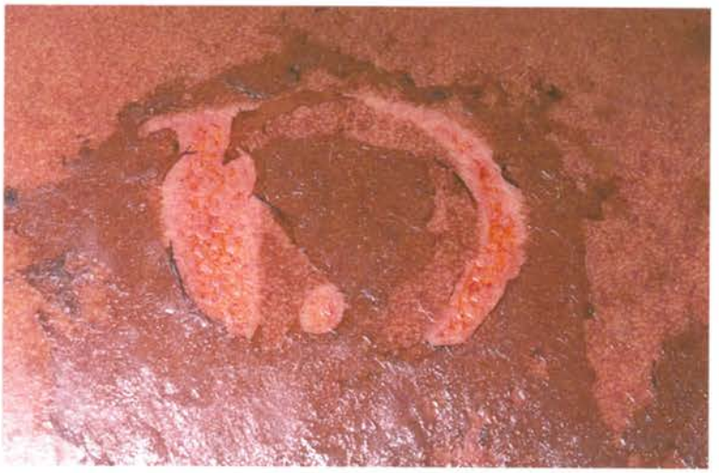

Fig. 1.46 Necrolysis. There is superficial necrosis and sheeting away of the skin. In this patient with *toxic epidermal necrolysis*, it was particularly marked where the electrocardiograph leads had been applied.

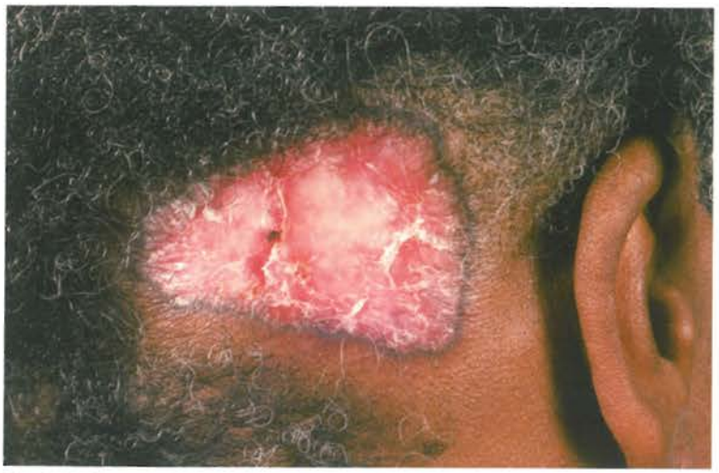

Fig. 1.47 A scar. A scar is the final stage of a destructive process that has involved the deeper dermis and resulted in permanent damage to the skin. In this patient with *lupus erythematosus*, hair follicles have been destroyed.

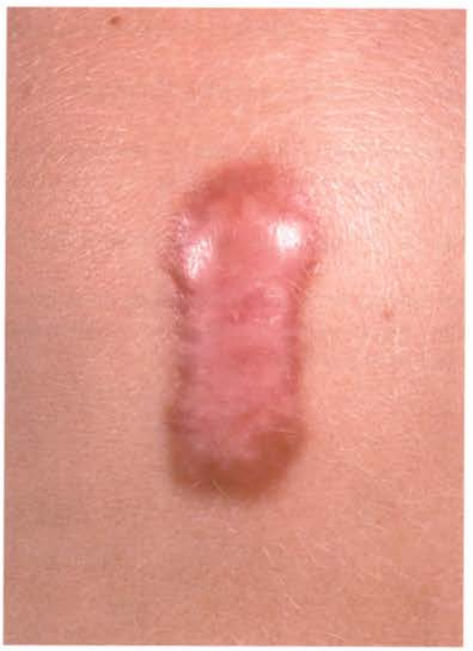

Fig. 1.48 Keloid scar. A scar is the replacement of the skin by fibrous tissue. It may be atrophic (thin and wrinkled), hypertrophic or *keloid* (elevated with excess growth of fibrous tissue), or cribriform (perforated with small pits).

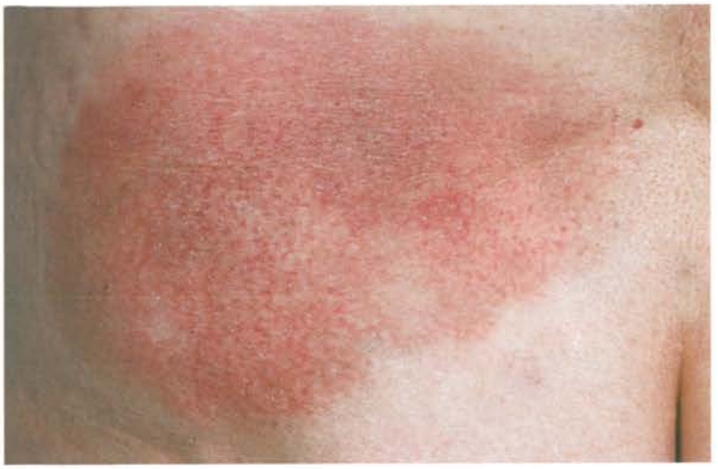

Fig. 1.49 Poikiloderma. This refers to skin that is atrophic, pigmented and telangiectatic. This well-defined patch on the buttock (a characteristic site) was caused by *mycosis fungoides*.

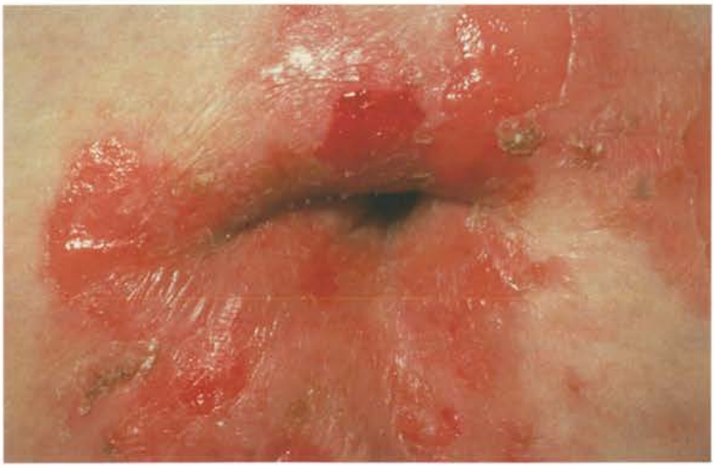

Fig. 1.50 Erosion. An erosion is a partial loss of epithelium, in this case due to intraepidermal blistering, which is visible around the edge of the raw glistening base of the erosions. This is *pemphigus vulgaris* around the umbilicus.

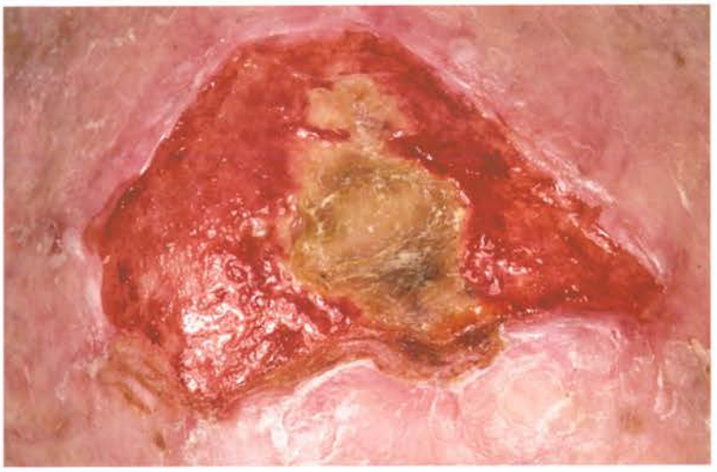

Fig. 1.51 Ulcer. An ulcer is full-thickness destruction of the epidermis. This is *radionecrosis* following irradiation of scalp ringworm.

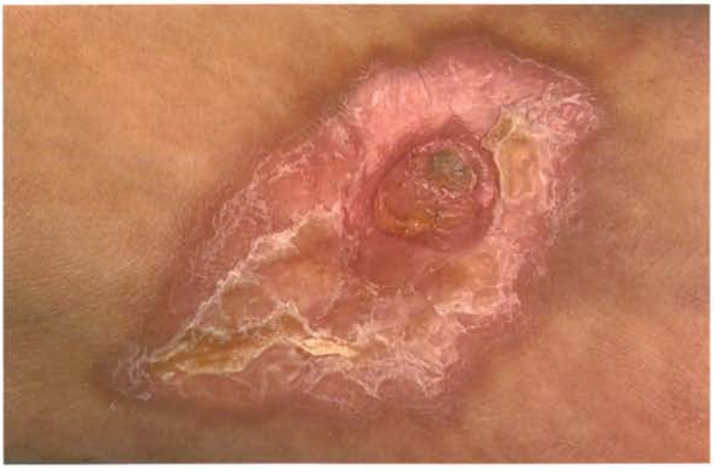

Fig. 1.52 Ulcer. There is an ulcer within a very well-defined, waxy, yellow-red plaque with a mauve edge. This is *necrobiosis lipoidica diabeticorum*.

Poikiloderma This refers to an appearance of pigmentation, atrophy and telangiectasia (Fig. 1.49). The term is derived from the Greek word ποικιλος meaning dappled.

Vegetation A growth of pathological tissue consisting of multiple, close-set, papillomatous masses.

Erosion A partial break in the epidermis is known as an erosion; it heals without scarring unless secondary infection occurs (Fig. 1.50). It commonly follows a blister.

Ulcer An ulcer is a full-thickness loss of the epidermis that heals with scarring (Figs 1.51 and 1.52).

Atrophy Thinning and transparency of the skin is caused by diminution of the epidermis, the dermis, or both (Fig. 1.53). There is wrinkling and translucency of the skin with loss of skin markings.

Sclerosis A circumscribed or diffuse hardening or induration of the skin (Fig. 1.54) can occur as a result of dermal or subcutaneous oedema, cellular infiltration or collagen proliferation.

An eruption may be either essentially monomorphic (e.g. molluscum contagiosum) or polymorphic (consisting of various forms; Fig. 1.55). For example, comedones, papules, pustules, cysts and scars may be found in acne. The lesion may evolve through various stages, such as from macules, to vesicles, pustules and crusts, and sometimes to postinflammatory pigmentation, as in herpes simplex and scarring in chickenpox. Certain other characteristics of the lesions must be observed as described below.

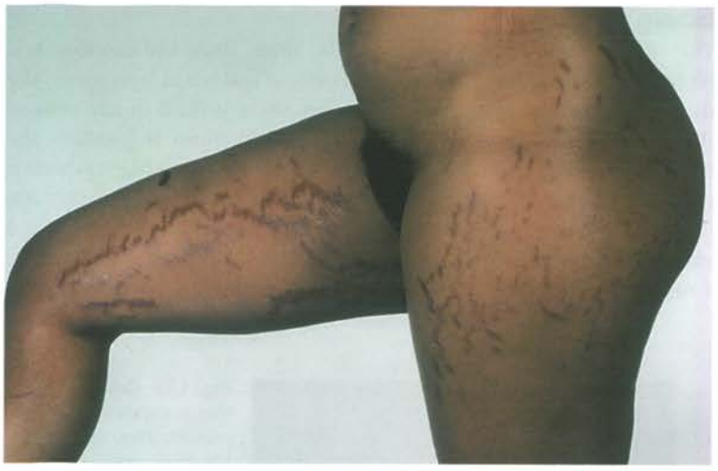

Fig. 1.53 Striae. There is atrophy of both the epidermis and dermis to be seen here on the inner and outer aspects of the thighs secondary to the use of skin lightening creams containing, inter alia, *superpotent steroids*.

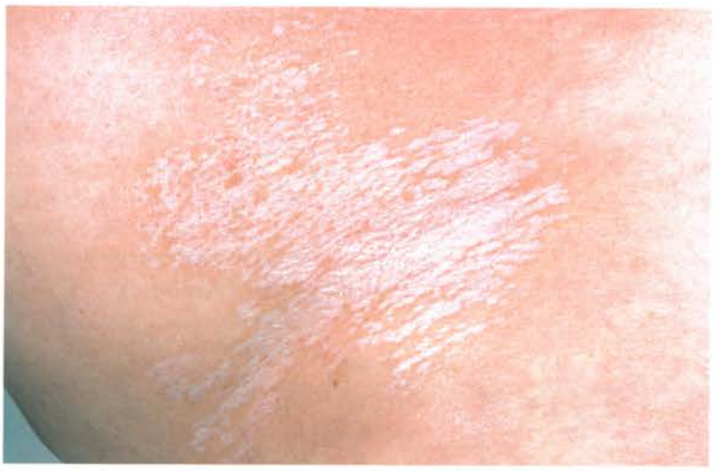

Fig. 1.54 Sclerosis. There is hardening of the skin. Here in *lichen sclerosus et atrophicus*, the ivory-white areas are sclerotic and will become wrinkled and atrophic.

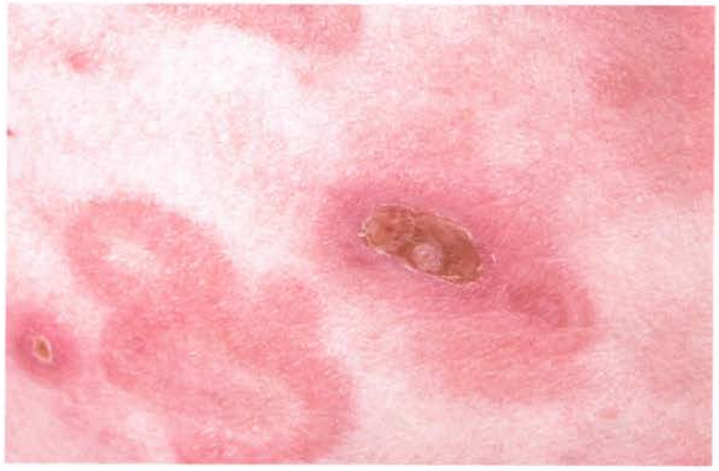

Fig. 1.55 Polymorphic eruption. There are various forms in this patient with *mycosis fungoides*. There is an annular plaque (bottom left), a diffuse pink patch (top right), a necrotic tumour (centre) and a crusted papule (left).

Morphology of the lesions

Once the type or types of lesion have been discerned, they must be defined further in terms of colour, consistency, nature, texture, scaliness or otherwise of the surface, pattern, shape, margin and arrangement.

Colour

The colours found in the skin are derived from melanin (brown), phaeomelanin (as in red hair), carotenoids (yellow), oxyhaemoglobin (bright red) and reduced haemoglobin (bluish red). Their presence, absence, diminution or excess produce a wealth of colours, which are critical for diagnosis of skin disorders. For example, eczema is pink, psoriasis is red and pityriasis versicolor is frequently brown; the papules of lichen planus are purple (Fig. 1.56), of scabies red, of xanthomas yellow (Fig. 1.57) or orange, and of a blue naevus blue. The pigments and colours of a

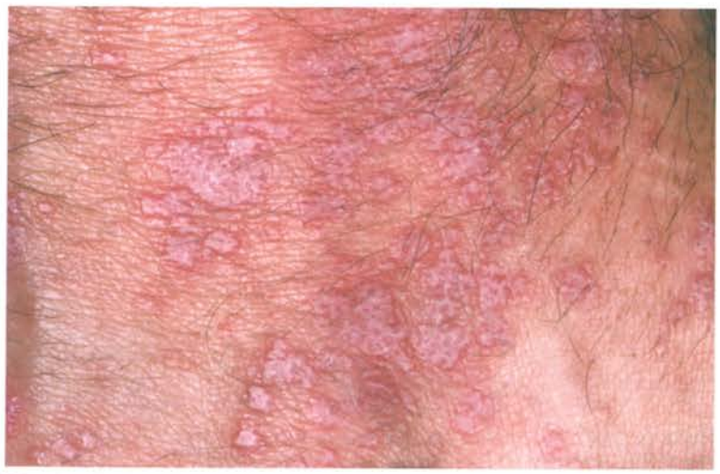

Fig. 1.56 Colour. These papules have merged into each other to form plaques. They have a distinctive purple or violaceous colour with a flat, shiny surface covered with white striae. This is *lichen planus*.

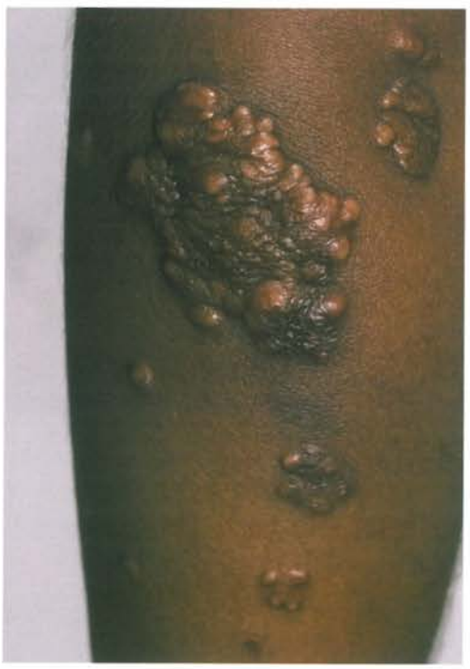

Fig. 1.57 Colour. This may be more difficult to detect in black skin, but even with these papules and nodules a yellow or orange colour is visible amongst the pigment. These *xanthomas* are secondary to biliary hypoplasia.

superficial spreading malignant melanoma are varied (Fig. 1.58), whereas those of a solar lentigo are quite uniform. Purpura (Fig. 1.59) is distinguished from erythema (see Fig. 1.12) because the latter is red and blanches with pressure, whereas the former is purple and does not. Changes in melanin pigmentation following common inflammatory disorders such as acne and eczema are usual in pigmented races but occur barely, if at all, in white races. This may make diagnosis confusing for the inexperienced as well as bedevil treatment. Certain skin disorders always produce postinflammatory pigmentary changes whatever the patient's basic skin colour (e.g. lichen planus and pityriasis versicolor).

Scale

The skin is shed imperceptibly all the time, since the epidermis is replaced every 28 days. This becomes visible as scales if at least part of the disorder affects the epidermis. In eczema, there is fluid in and around the epidermal cells, producing a disordered epithelium. In psoriasis, the basal cells are mitotically active and a hyperproliferative epidermis results. These disorders are scaly and because they are also red, they are known as erythematosquamous diseases. The scale is fine in eczema and exfoliative dermatitis (Fig. 1.60), and thick and silvery in psoriasis (Fig. 1.61). After sunburn or an infection producing an erythemogenic toxin,

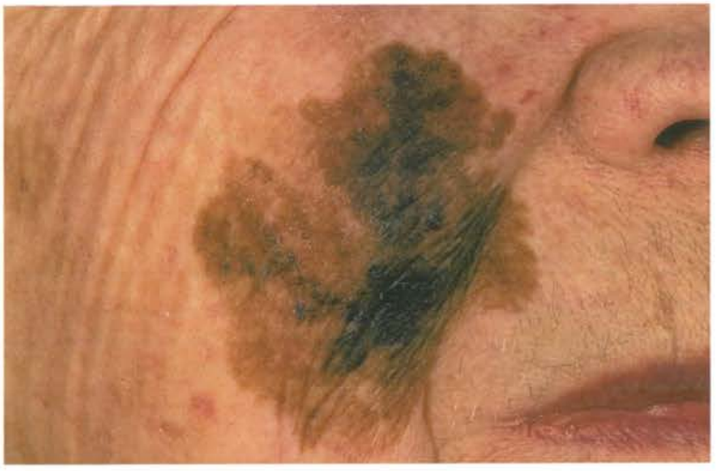

Fig. 1.58 Colour. There is a multiplicity of shades of melanin pigment ranging from light brown to black and an irregular scalloped outline. This is *Hutchinson's lentigo maligna melanoma* on the face.

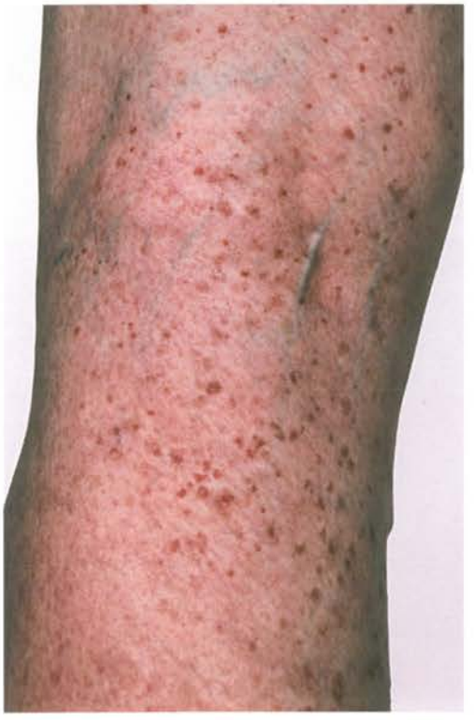

Fig. 1.59 Colour. Purple papules are present. They did not blanch on pressure and this purpuric eruption was confirmed on biopsy to be a *vasculitis*.

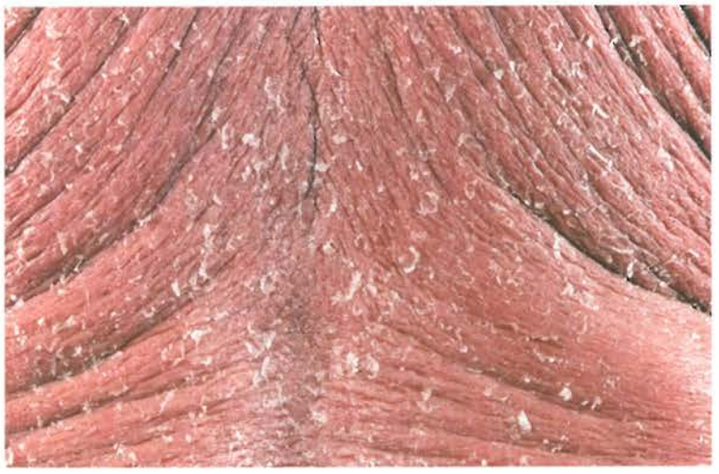

Fig. 1.60 Scale. The scale may be fine as in eczema or as in this case of *exfoliative dermatitis* secondary to the Sézary syndrome.

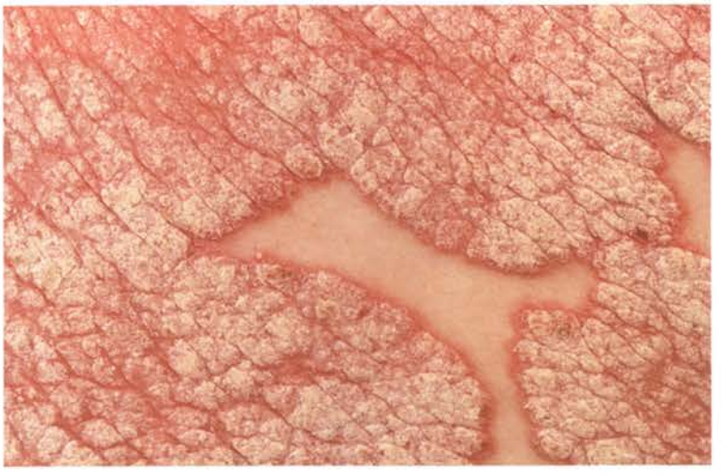

Fig. 1.61 Scale. In *psoriasis*, the scale is usually thick and a silvery white colour. The lesions are slightly raised plaques and very well defined.

a very superficial peeling of the stratum corneum (desquamation) occurs. In pityriasis versicolor, where there is colonization of the outermost layer of the epidermis by a fungus, the scale may be barely imperceptible until the macules are scraped with a blunt scalpel. The scale may be tenacious, as in lupus erythematosus, or thick and easily scraped off to reveal minute bleeding points, as in psoriasis. The scale may be uniformly spread across the lesion, as in eczema, or more marked peripherally, as in pityriasis rosea. In certain ichthyoses, on the one hand, where there is a failure to desquamate owing to retention of horn cells, scaling occurs without inflammation (Fig. 1.62). On the other hand, if the primary pathology is in the dermis, as in lichen planus or granuloma annulare (Fig. 1.63), no scaling is produced at all.

Shape

Lesions may be round or discoid as in nummular eczema (Fig. 1.64), oval as in pityriasis rosea or all manner of shapes and sizes as in psoriasis. Annular lesions are not synonymous with round ones (Fig. 1.65). The

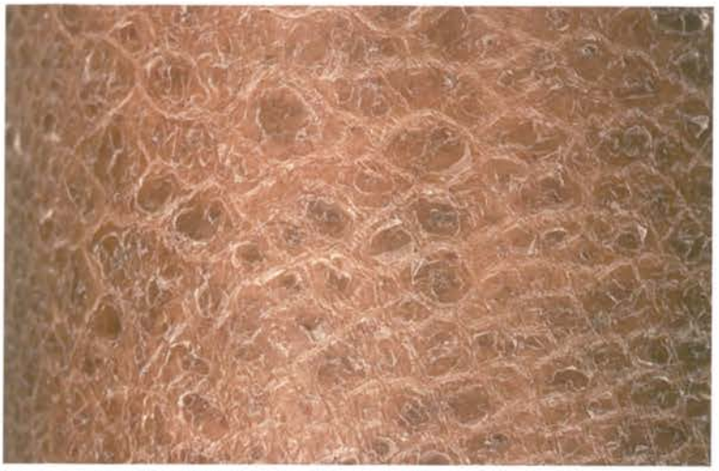

Fig. 1.62 Scale. In *X-linked ichthyosis vulgaris*, the scale is a flat plate (lamella) and results from steroid sulphatase deficiency such that the corneocytes adhere together incorrectly.

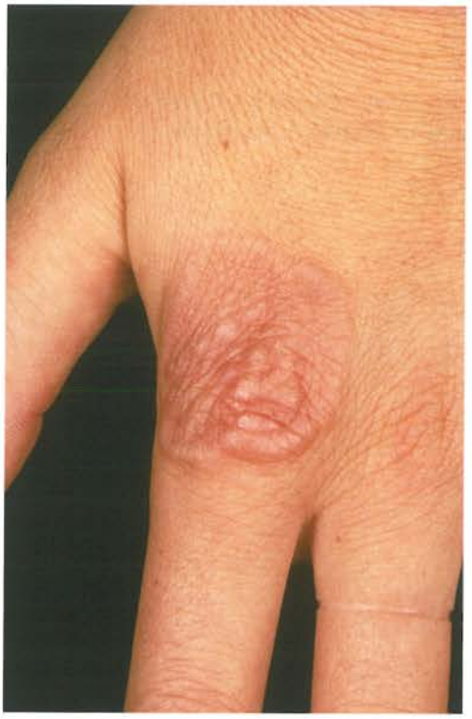

Fig. 1.63 Shape. In *granuloma annulare*, the lesion is annular with a raised margin, often composed of small papules with a contrasting flatter area within. There is no scaling. The back of the hand is a common site.

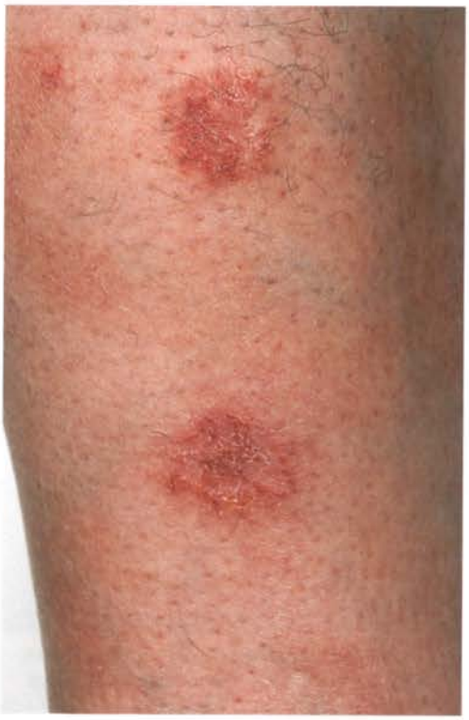

Fig. 1.64 Shape. These lesions are round or discoid in shape. There is no central clearing. The scaling and redness are uniformly distributed. It represents nummular (coin-shaped) or *discoid eczema*.

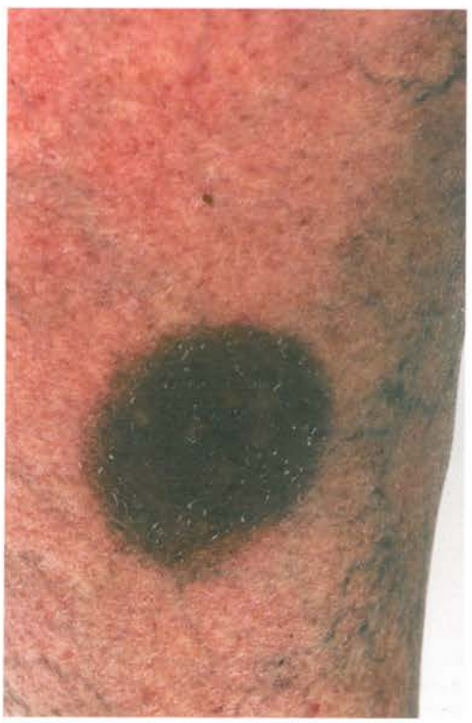

Fig. 1.65 Shape. This completely round pigmented patch is the characteristic appearance of a *fixed drug eruption*.

former have clear or contrasting centres (Fig. 1.66), the latter do not. Polycyclic lesions (Fig. 1.67) are arranged in more than one ring; further distinction can be made on the basis of scaling. Epidermal lesions, such as ringworm (Fig. 1.68) are scaly whereas dermal eruptions, such as sarcoidosis, are not. The individual papules of lichen planus are polygonal in shape.

Surface

The lesion may be rough as in a seborrhoeic wart (Fig. 1.69) or smooth as in dermal melanocytic naevus (a mole). An individual papule may be flat topped, as in lichen planus, pointed (acuminate), as in miliaria rubra, mammilated, as in a compound naevus (Fig. 1.70), or dome shaped and umbilicated, as in molluscum contagiosum.

Consistency

The lesion may be firm as in a dermatofibroma, soft as in a dermal mole, hard as in a secondary deposit or tethered as in scleroderma.

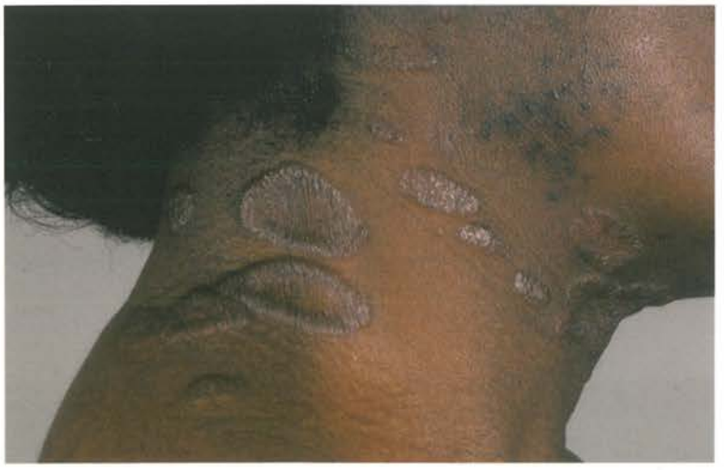

Fig. 1.66 Shape. The lesions are annular or ring shaped with a raised margin and no scaling, indicating that the pathology is not in the epidermis but in the dermis or deeper. This is *sarcoidosis*.

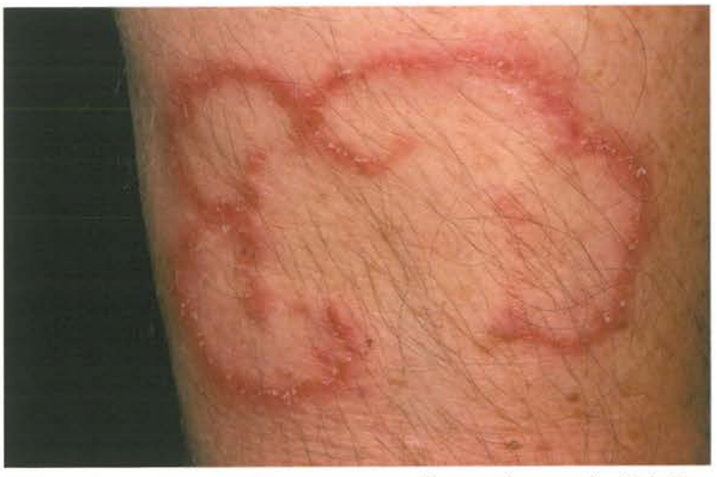

Fig. 1.67 Shape. Polycyclic lesions are arranged in more than one ring. Note the scaling of the slightly raised margin. This is *tinea corporis*.

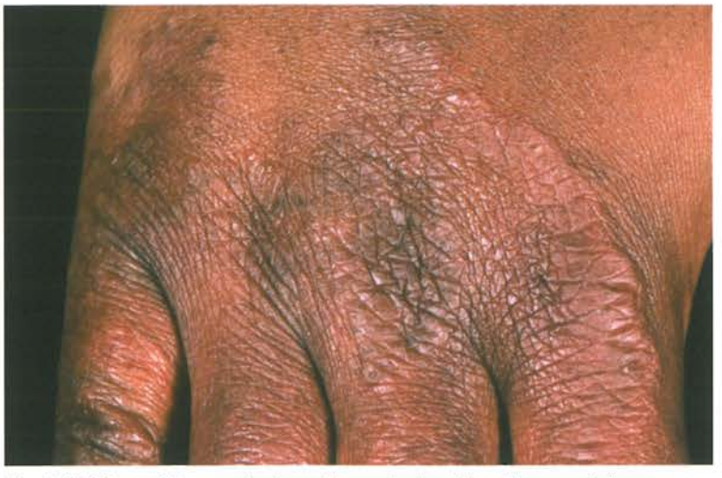

Fig. 1.68 Shape. The eruption is partly annular in outline. The margin is more pronounced and scaly than the pink area within it. Healing has resulted in postinflammatory pigmentation near the fifth knuckle. This is *tinea corporis*.

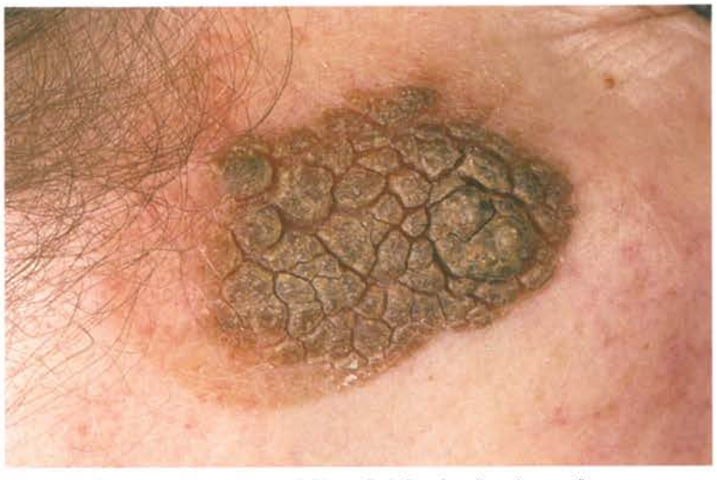

Fig. 1.69 Surface. The surface of this well-defined, yellow-brown (hence seborrhoeic) wart is rough and split (fissured) and appears almost to be stuck to the surface. It is also known as a *basal cell papilloma*.

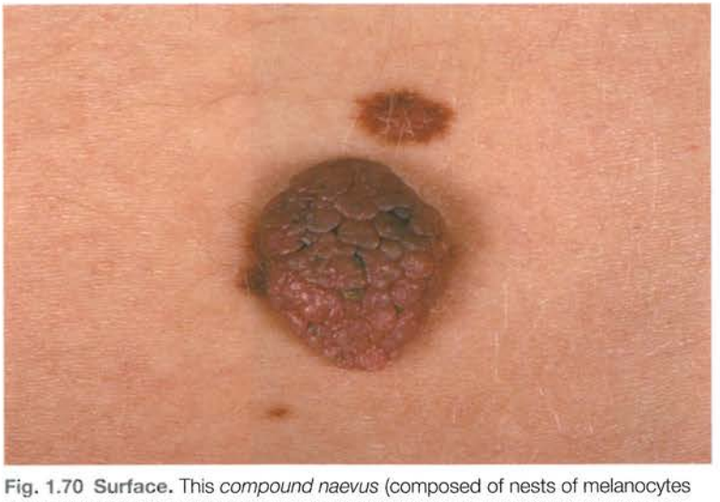

Fig. 1.70 Surface. This *compound naevus* (composed of nests of melanocytes at the dermoepidermal junction and in the dermis) has a mamillated (nipple-like) surface. This benign mole feels soft and the surface is smooth.

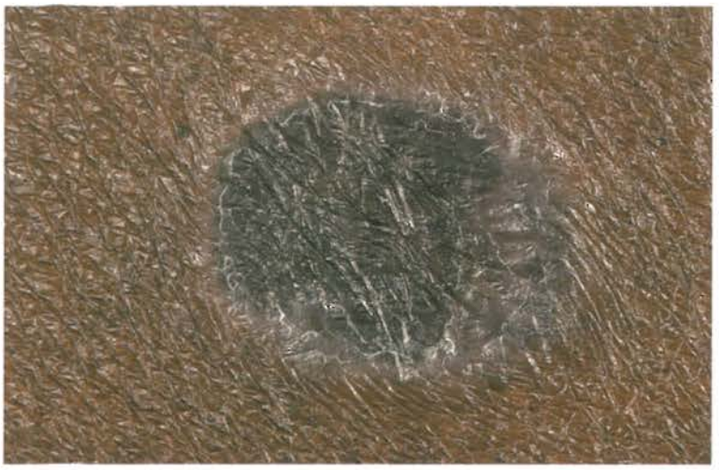

Fig. 1.71 Margin. The majority of this annular lesion shows postinflammatory hyperpigmentation but the margin is purple and papular, which is the clue to the diagnosis of *lichen planus*.

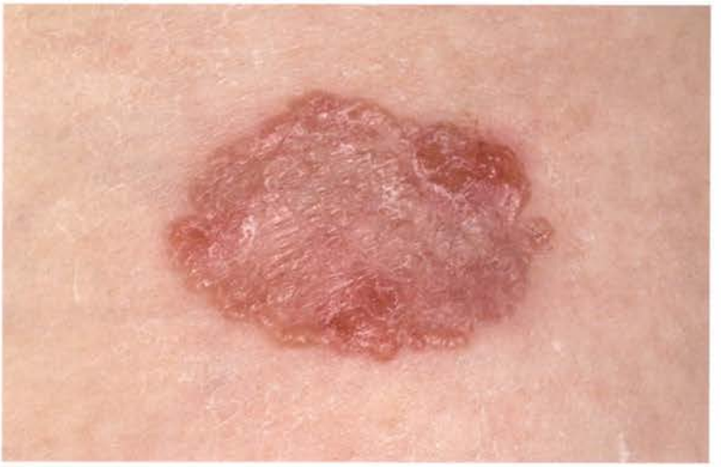

Fig. 1.72 Margin. The border of this well-defined, red, slightly scaly plaque has a distinct rolled pearly edge. It is a *superficial basal cell carcinoma*.

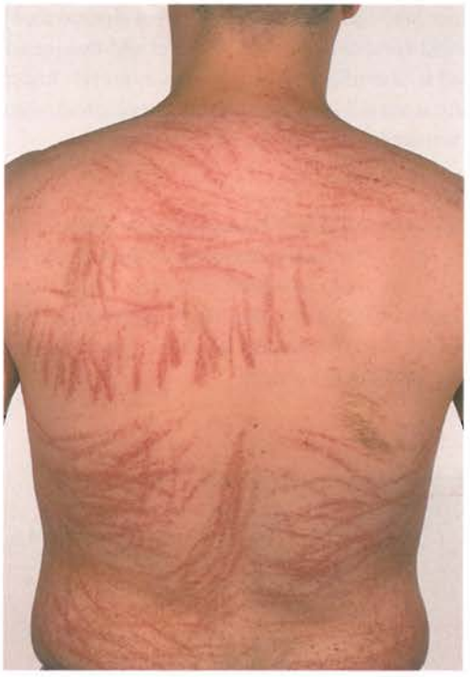

Fig. 1.73 Linearity. This bizarre linear streaky patterned eruption occurred 48 hours after ingesting raw or half-cooked *Shiitake mushrooms* (*Lentinus edodes*). A similar flagellate reaction may occur with bleomycin.

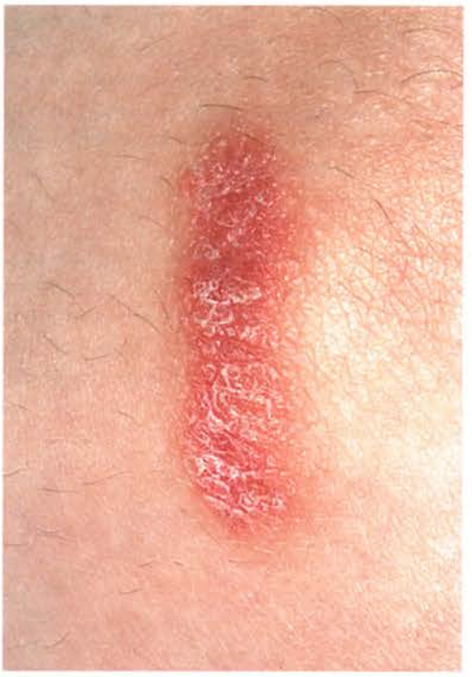

Fig. 1.74 Linearity. This linear deep red lesion with prominent scaling is *psoriasis* occurring in an area of traumatized (in this case scratched) skin. It is known as the *Koebner phenomenon*.

Margin

The lesion may be discrete as in psoriasis or indistinct as in many forms of eczema. There may be more activity peripherally, with a tendency to central healing, as in tinea or annular lichen planus (Fig. 1.71). The margin may be raised and rolled, as in basal cell carcinoma (Fig. 1.72), or irregular and notched as in a malignant melanoma (see Fig. 1.58).

Pattern

The lesions may be arranged in a particular manner. They may be linear, annular, grouped or reticulate.

Linear pattern

Linearity may be explained by involvement of a dermatome (e.g. herpes zoster), of blood vessels (e.g. thrombophlebitis) or of lymphatics (e.g. sporotrichosis or lymphangitis). Exogenous agents such as plant allergens or their derivatives produce linear streaking (e.g. phytophotodermatitis and poison ivy dermatitis) and pigmentation (e.g. Berloque dermatitis) on exposed skin. Drugs, especially bleomycin and shiitake mushrooms (Fig. 1.73) may produce bizarre flagellate-like streaking patterns on the skin. Some self-induced lesions such as artefacts are linear in arrangement.

Linear lesions may be of developmental origin (e.g. epidermal naevi) or may follow Blaschko's lines, which do not conform to any known vascular or nervous structure. Other lesions may be determined by the Koebner (or isomorphic) phenomenon (Fig. 1.74), which is the induction of an eruption at the site of trauma, be it scratching or ultraviolet light. Certain diseases manifest this phenomenon (e.g. psoriasis, lichen planus and plane warts). However, there are many linear configurations that are unexplained (e.g. lichen striatus, lichen sclerosus, linear morphoea and porokeratosis of Mibelli). Finger-like shapes are typical of digitate dermatosis (Fig. 1.75).

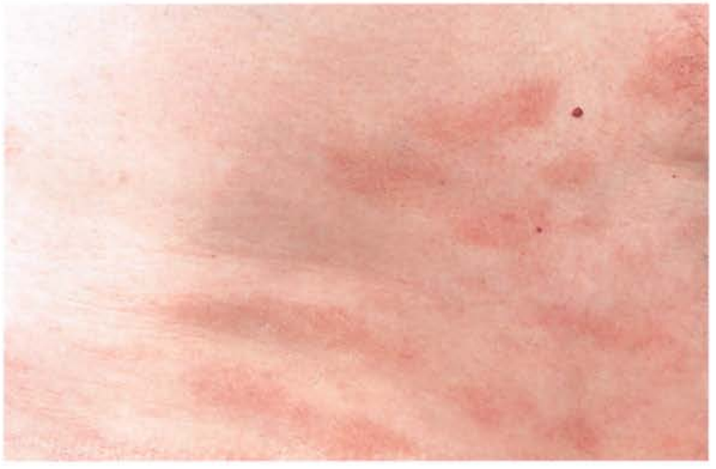

Fig. 1.75 Linearity. These finger-like processes are a distinctive part of *digitate dermatosis* (chronic superficial scaly dermatosis), a probable aborted form of mycosis fungoides.

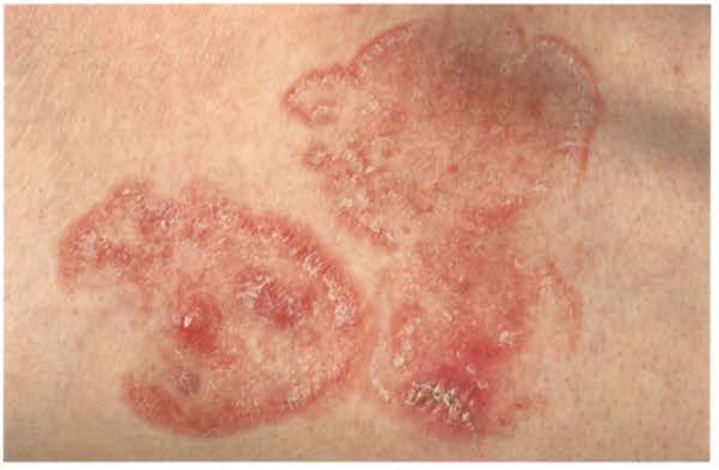

Fig. 1.76 Arciform and polycyclic lesions. The red, scaly plaque on the left is arciform (an incomplete circle) and on the right is polycyclic. Biopsy confirmed *mycosis fungoides*.

Annular pattern

Annular, arciform (incomplete circular lesions; Fig. 1.76) and polycyclic forms are common. They have to be defined further as to whether they are macular (e.g. annular erythema), papular (e.g. granuloma annulare), scaling (e.g. tinea corporis) or nodular (e.g. sarcoidosis, tertiary syphilis or mycosis fungoides). A special configuration is the iris or target lesion (Fig. 1.77), which is almost unique to erythema multiforme.

Grouped lesions

If lesions occur in a group and make a particular pattern, a diagnosis may sometimes be readily obtained (e.g. insect bites). The cluster of vesicles in herpes simplex (Fig. 1.78) is so characteristic that the term herpetiform is used to describe other conditions that simulate grouped vesicles (e.g. dermatitis herpetiformis). Similarly, the linear pattern of vesicles in groups seen in herpes zoster lends the title zosteriform to certain eruptions that occur in a band-like manner (though not conforming to a dermatome) and, in particular, to naevoid conditions. Another rather old-fashioned term that is sometimes used is corymbiform. This means a central cluster of lesions beyond which are scattered individual lesions, a phenomenon that is sometimes seen in verrucae.

Reticular arrangements

A net-like (Latin *reticulum* means a little net) arrangement is seen in particular in livedo reticularis (Fig. 1.79), cutis marmorata and erythema ab igne, where the horizontal pattern of blood vessels under the skin is highlighted. Lichen planus produces just such a pattern in the mouth (Fig. 1.80). The individual papule of this disease may be seen to have a white lace-like change in its surface (Wickham's striae).

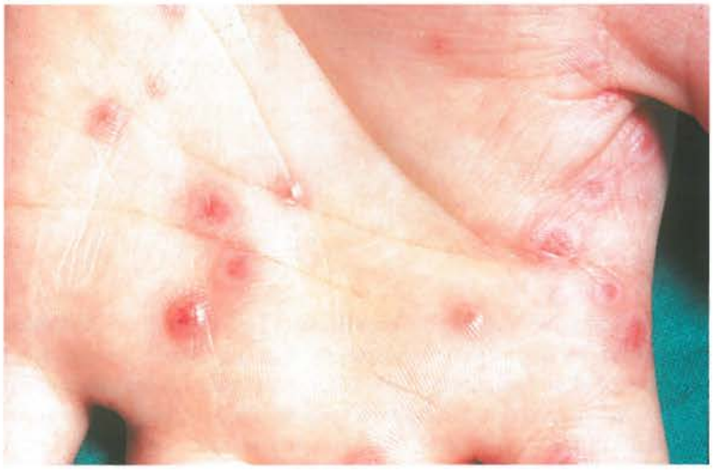

Fig. 1.77 Target lesion. These plaques with a target or iris annular configuration are virtually diagnostic for *erythema multiforme* caused by herpes simplex.

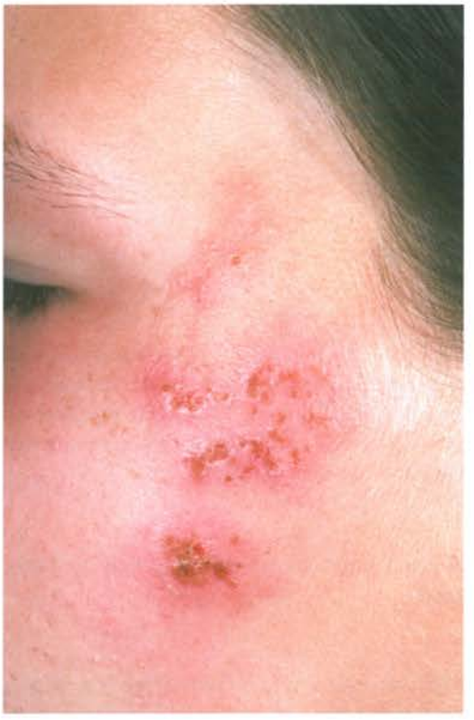

Fig. 1.78 Grouped lesions. These vesiculopustular lesions are grouped together on an erythematous background. This is *herpes simplex*; such an arrangement in other conditions is often known as herpetiform.