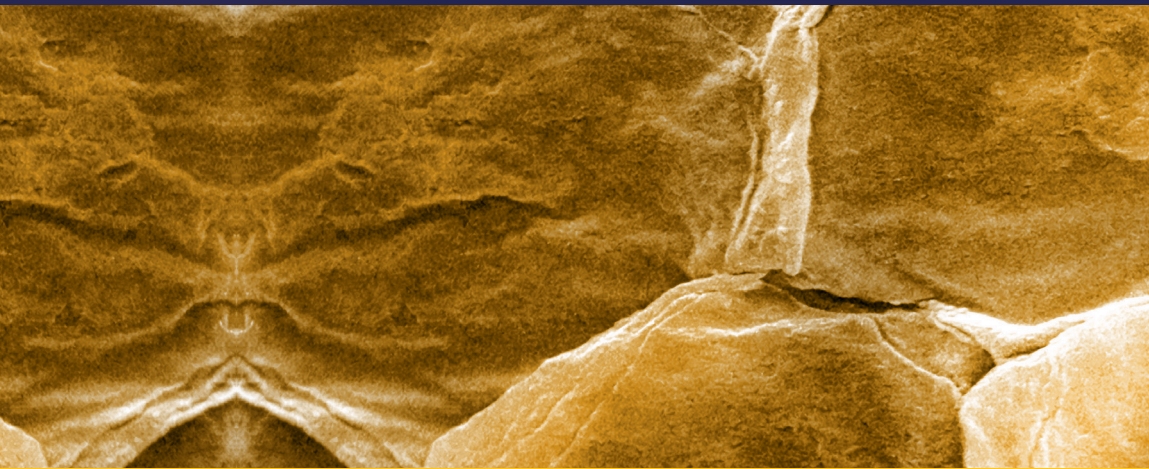


Rook's Dermatology Handbook



EDITED BY

Christopher E. M. Griffiths | Tanya O. Bleiker

Daniel Creamer | John R. Ingram | Rosalind C. Simpson

WILEY Blackwell

Rook's
Dermatology
Handbook

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Preface

Rook's Textbook of Dermatology is a four volume behemoth of information, arguably THE dermatological reference. First published in 1968 Rook has evolved from the visionary text produced by Arthur Rook, Darrell Wilkinson and John Ebling and is now in its 9th edition. Indubitably this is indispensable as a detailed reference on dermatology but is neither a pocket book for ready access in clinic nor a skin disease "101" for trainees, non-dermatologists and hard pressed consultants. The Rook editors had discussed the relative merits of a "Rook Handbook" for several years and eventually decided, by popular demand, that the time had come for the talking to stop and work to begin on such a book.

Rook's Dermatology Handbook is a 1000 page, fully illustrated guide to facilitate the rapid diagnosis of skin diseases and provide ready access to key relevant facts about them. The format comprises epidemiology, pathophysiology, clinical features, differential diagnosis, investigations and management. The basic science is kept to a minimum, there are no photomicrographs of histology and management is top line only. The reader would be expected to turn to more detailed references for

in-depth information. We have pared down the text from Rook's textbook, retained many of its high-quality images and introduced new tables and new sections, including dermatological vocabulary and differential diagnosis of common clinical presentations such as blisters, hair loss and erythroderma. Three of the editors of the textbook – Chris Griffiths, Tanya O. Bleiker and Daniel Creamer – have been joined in the venture by two next generation clinicians, John Ingram and Rosalind Simpson. We are delighted with the book but the proof will come from how it performs in the clinic as an aid to the diagnosis, understanding and management of dermatological disease. We are indebted to all of the authors from the Rook 9th edition who freely gave us permission to recycle and synthesise their chapters.

The book would not have seen the light of day without the unstinting support and expertise of Claire Bonnett, Jenny Seward and Nick Morgan of Wiley, and Production team.

Chris Griffiths
Tanya O. Bleiker
Daniel Creamer
John Ingram
Rosalind Simpson

Glossary

Alopecia	Decreased density or thickness of hairs	Keratoderma	Thickening of the stratum corneum and/or epidermis of the palms and soles, often inherited
Artefact	Induced by exogenous injury, sometimes self-inflicted	Keratosis	Focal thickening of the epidermis, especially the stratum corneum
Callus	Reactive hyperkeratosis, usually due to friction and/or pressure, leading to enhanced skin markings	Kerion	Boggy plaque, due to infection, that often contains pustules
Comedone (open and closed)	<i>Open:</i> dilated hair infundibulum with oxidised (black) keratinous debris ('blackhead') <i>Closed:</i> expansion of hair infundibulum by keratinous debris, usually with no connection to skin surface ('whitehead')	Lichenification	Accentuation of skin markings, often due to rubbing
Dysaesthesia	Inappropriate sensations, e.g. paraesthesias	Poikiloderma	Simultaneous presence of atrophy, telangiectasia and hypo- and hyperpigmentation
Exanthem	Acute widespread eruption, usually due to a viral infection or drug reaction	Prurigo	Papules or nodules due to scratching or picking
Fissure	Linear disruption of stratum corneum; may extend into the dermis	Purpura	Haemorrhage into the skin due to pathological processes, primarily of blood vessels
Infarct	Ischaemia of tissue due to arterial occlusion	Stria	Linear atrophy along tension lines; initially can be red to purple in colour
Induration	Deep thickening of the skin can result from oedema, inflammation, or infiltration	Telangiectasia	Permanently dilated capillaries and venules which are visible to the naked eye

Abbreviations

ACTH	adrenocorticotrophic hormone	HLA	human leukocyte antigen
AE	atopic eczema	HPV	human papilloma virus
AGEP	acute generalised exanthematous pustulosis	HSV	herpes simplex virus
AIDS	acquired immune deficiency syndrome	IF	immunofluorescence
AK	actinic keratoses	IFN	interferon
ALP	alkaline phosphatase	IgE	immunoglobulin E
ALT	alanine aminotransferase	IL	interleukin
ANA	antinuclear antibody	IMF	immunofluorescence
ANCA	antineutrophil cytoplasmic antibodies	IV	intravenous
ASOT	antistreptolysin O titre	KC	keratinocyte carcinoma
AST	aspartate aminotransferase	LDH	lactate dehydrogenase
ATP	adenosine triphosphate	LE	lupus erythematosus
BCC	basal cell carcinoma	LFT	liver function test
BCG	bacille Calmette–Guérin	LP	lichen planus
BP	blood pressure	MHC	major histocompatibility complex
BSA	body surface area	MM	Investigations malignant melanoma
CBT	cognitive behavioural therapy	MMR	mumps measles rubella vaccination
CMV	cytomegalovirus	MRI	magnetic resonance imaging
CNS	central nervous system	MRSA	meticillin-resistant Staphylococcus aureus
CREST	calcinosis, Raynaud phenomenon, oesophageal dysmotility, sclerodactyly and telangiectasia	NSAID	non-steroidal anti-inflammatory drug
CRP	C-reactive protein	PAS	periodic acid–Schiff
CT	computerised tomography	PASI	Psoriasis Area Severity Index
CTCL	cutaneous T-cell lymphoma	PCR	polymerase chain reaction
CVI	chronic venous insufficiency	PUVA	psoralen and ultraviolet A
CXR	chest X-ray	PVL	Panton–Valentine leukocidin
DEJ	dermal–epidermal junction	PXE	pseudoxanthoma elasticum
DLE	discoid lupus erythematosus	RAS	Recurrent aphous stomatitis
DLQI	Dermatology Life Quality Index	RNA	ribo nucleic acid
DNA	deoxyribonucleic acid	QoL	quality of life
EBV	Epstein–Barr virus	SCC	squamous cell carcinoma
EGFR	epidermal growth factor receptor	Sinus	Tract leading from a deeper focus to the skin surface
ELISA	enzyme-linked immunosorbent assay	SLE	systemic lupus erythematosus
ENA	extractable nuclear antigen	SPF	sun protection factor
ESR	erythrocyte sedimentation rate	TCR	T-cell receptor
FBC	full blood count	TFT	thyroid function test
5-FU	5-fluorouracil	TNF	tumour necrosis factor
G6PD	glucose-6-phosphate dehydrogenase	TPMT	thiopurine methyltransferase
GvHD	graft-versus-host disease	U&E	urea and electrolytes
H&E	haematoxylin and eosin	UV	ultraviolet
Hb	haemoglobin	UVR	ultraviolet radiation
HHV	human herpesvirus	VZV	varicella-zoster virus
HIV	human immunodeficiency virus	WCC	white cell count
		WHO	World Health Organization

Introduction

Human skin consists of a stratified, cellular epidermis and an underlying dermis of connective tissue, separated by a dermal-epidermal basement membrane (Figure 1.1). Beneath the dermis is a layer of subcutaneous fat, which is separated from the rest of the body by a vestigial layer of striated muscle.

The skin performs a number of functions, including:

- Providing a physiological barrier against the external environment.
- Maintaining fluid balance by restricting water loss through the skin.
- Forming an innate immune defence against bacteria, fungi and viruses through keratinocyte-derived endogenous antibiotics, defensins and cathelicidins. Langerhans cells have a primary role in epidermal immune surveillance.
- Supporting thermoregulation: vasodilatation or vasoconstriction of the blood vessels in the deep and superficial plexuses helps to regulate body temperature. Eccrine sweat glands, found at all skin sites, also play a role in heat control.
- Providing insulation and trauma protection: subcutaneous fat limits excessive heat loss and shields internal structures from physical trauma.

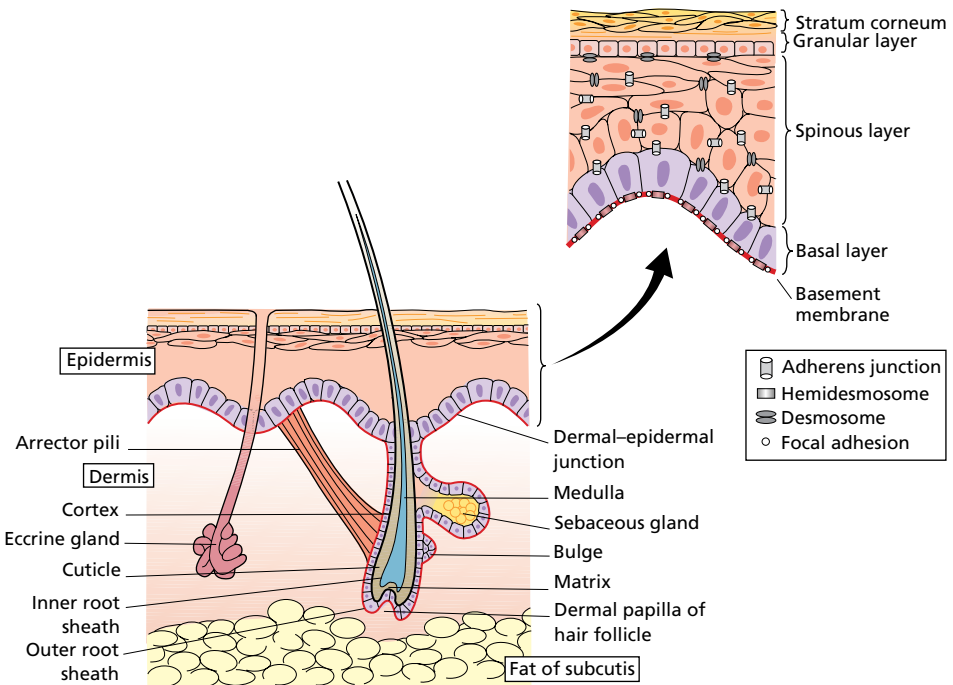


Figure 1.1 The skin and its appendages.

Fat also has an endocrine function, releasing the hormone leptin, which acts on the hypothalamus to regulate hunger and energy metabolism.

- Vitamin D production.
- Performing a psychosocial function: the appearance of human skin and its associated structures, especially scalp hair, has a major impact on self-image and thus on interpersonal relationships.
- Protection from ultraviolet (UV) radiation through production of melanin from melanocytes. Variation in response to sunlight has historically been divided into six categories according to the Fitzpatrick classification (Table 1.1). It is acknowledged that whilst these 'skin types' describe response to UV exposure, they do not adequately encompass the wide variation of tones seen in skin of colour.

History taking

Clinical assessment of a patient presenting with a skin disorder follows the standard approach of history-taking and physical examination. Investigations may be needed to supplement information obtained at the consultation.

Although a dermatological diagnosis might be swiftly apparent, it is essential to take a full history before examining the skin. The history of the presenting complaint will yield the story of the symptoms and the clinical features of the dermatosis (Box 1.1). Further questions will give details about any associated clinical problems and the patient's background medical history (Table 1.2). The act of questioning (and listening to the answers) contributes to a consultation's therapeutic function.

Table 1.1 Fitzpatrick classification of skin types

Skin type	Skin colour on sun-protected site	Sunburn risk	Tanning ability	Skin cancer risk
I	White	++++	±	High
II	White	+++	+	High
III	White	++	++	Moderate
IV	Olive	+	+++	Moderate
V	Brown	±	++++	Low
VI	Black/dark brown	±	++++	Low

Box 1.1 History of the presenting complaint

Essential points in the history: rashes

- Location: Where did the rash start, where did it spread to?
- Temporal: When did it start, does it come and go, if so, how long does it last?
- Exacerbating factors: What makes it worse?
- Alleviating factors: Physical factors, diet, treatment?
- What are the predominant symptoms: itch, pain, disfigurement?
- Occupational factors: Does it get worse at work? Does it improve away from work?
- Open questions: Do you have any thoughts as to what has caused this? What concerns you most about this problem?

Essential points in the history: lesions

- Where is the lesion? Is it single or multiple?
- Was there any trauma before it arose?
- Temporal: How long has it taken to develop to this size? Is it still growing or is it resolving?
- Symptoms: Is it tender, painful or itchy? Are there any exacerbating factors? Has it bled?
- Past history: Have you had anything similar before?
- Sun exposure: How much sun exposure have you had, including living/ working abroad and use of sun beds? Any history of blistering sun burns?

Table 1.2 Background medical history

Further history	Rationale	Example
General medical history	Systemic diseases may have cutaneous features	Dermatomyositis and other connective tissue disorders
Medication history	Certain dermatoses are induced by drugs	Drug-induced exanthem
Allergy history	Certain dermatoses are caused by an allergy to a food or drug or contact allergen	Oral allergy syndrome
Family history	Certain dermatoses are inherited	Basal cell carcinoma syndrome (Gorlin syndrome)
Occupational history	The trigger for a dermatosis may be found only at the patient's workplace	Allergic contact dermatitis in a hairdresser
Leisure history	Certain dermatoses are related to leisure activities	Allergic contact dermatitis to plants in gardening
Travel history	Certain dermatoses are more commonly encountered abroad	Leishmaniasis
Social history	Certain dermatoses are associated with lifestyle habits	Palmo-plantar pustulosis is associated with smoking
Ethnicity	Certain disorders are more prevalent in particular ethnic groups	Sarcoidosis and lupus erythematosus occur more frequently in patients with black skin
Quality of life	Effects of dermatosis on work, relationships, activities etc. can be quantified	Dermatology Life Quality Index

Clinical examination

As in any medical consultation, examination follows history-taking and the correct assessment of skin signs is only achieved in the context of a patient's symptoms. Effective interpretation of cutaneous clinical features relies on the principle that most skin diseases have characteristic lesions with a predilection for certain body sites. An understanding of these disease-specific patterns is intrinsic to diagnosis in dermatology, an assertion which is especially true in the appraisal of a rash.

The patient should always be examined in a good light, preferably daylight, and with magnification of lesions if necessary. A mobile light on a flexible stand can be helpful in illuminating areas that are in the shade from overhead lights, such as the mouth and the flexures. Ideally, the

entire skin should be examined in every patient, including the scalp and nails. Full skin examination may also reveal suspicious skin lesions that the patient was not aware of, for example lesions on the back.

Examination of a lesion

When assessing a solitary skin lesion there are a number of features relating to its morphology which direct the physician to a diagnosis. All skin lesions can be assigned to one of the descriptive entities defined in Table 1.3 (illustrated in Figure 1.2). Recognition of the lesion type is the basis of clinical examination in dermatology. Thereafter a more detailed appreciation of the lesion's properties will enhance the assessment. Use of the 5Ss is helpful in describing, and thus identifying, a lesion: **S**ite, **S**ize, **S**ymmetry, **S**hape and **S**urface.

Table 1.3 Descriptive terms for cutaneous lesions (adapted from Nast et al. 2016)

Term	Definition	Example
Bulla (Figure 2a)	A circumscribed lesion >1 cm in diameter that contains liquid (clear, serous or haemorrhagic)	Bullous pemphigoid
Macule (Figure 2b)	A flat, circumscribed, non-palpable lesion that differs in colour from the surrounding skin	Junctional naevus
Nodule (Figure 2c)	An elevated, solid, palpable lesion >1 cm usually located primarily in the dermis and/or subcutis	Squamous cell carcinoma
Papule (Figure 2d)	An elevated, solid, palpable lesion that is ≤1 cm in diameter	Intradermal naevus
Patch (Figure 2e)	A flat circumscribed area of discoloration, >1 cm	Vitiligo
Plaque (Figure 2f)	A circumscribed, palpable lesion >1 cm in diameter; most plaques are elevated	Psoriatic plaque
Pustule (Figure 2g)	A circumscribed lesion that contains pus	Palmoplantar pustulosis
Vesicle	A circumscribed lesion ≤1 cm in diameter that contains liquid (not pus)	Herpes simplex
Weal	A transient elevation of the skin due to dermal oedema	Urticaria
Scale (Figure 2h)	A visible accumulation of keratin, forming a flat plate or flake	Psoriasis
Crust	Dried serum, blood or pus on the surface of the skin	Impetigo
Erosion	Loss of either a portion of the epidermis or the entire epidermis	Pemphigus vulgaris
Excoriation	A loss of the epidermis and a portion of the dermis due to scratching or an exogenous injury	Scratching from any cause
Ulcer	Full-thickness loss of the epidermis plus at least a portion of the dermis	Venous leg ulcer

Source: Adapted from A. Nast et al. The 2016 International League of Dermatological Societies' revised glossary for the description of cutaneous lesions. *British Journal of Dermatology*, 2016, 174, pp. 1351–1358. John Wiley & Sons Ltd on behalf of the British Association of Dermatologists.

Examination of a rash

In the initial assessment of a rash the type of primary lesion, from which the dermatosis is constituted, needs to be identified. Thereafter the configuration of lesional skin on the skin's surface will point to the diagnosis, a deductive process termed pattern recognition. The description of a rash should comment on the morphology of individual lesions, including colour and shape (Table 1.4), as well as information on body sites of involvement and distribution

pattern (Table 1.5) (illustrated in Figure 1.3). Palpation of lesional skin imparts additional information about texture, skin thickness, tenderness and temperature.

Medical photographs

Medical photography is a useful tool to record the current state of a dermatosis and to permit serial comparisons over time to assess change. Patient consent and secure image storage are important considerations.

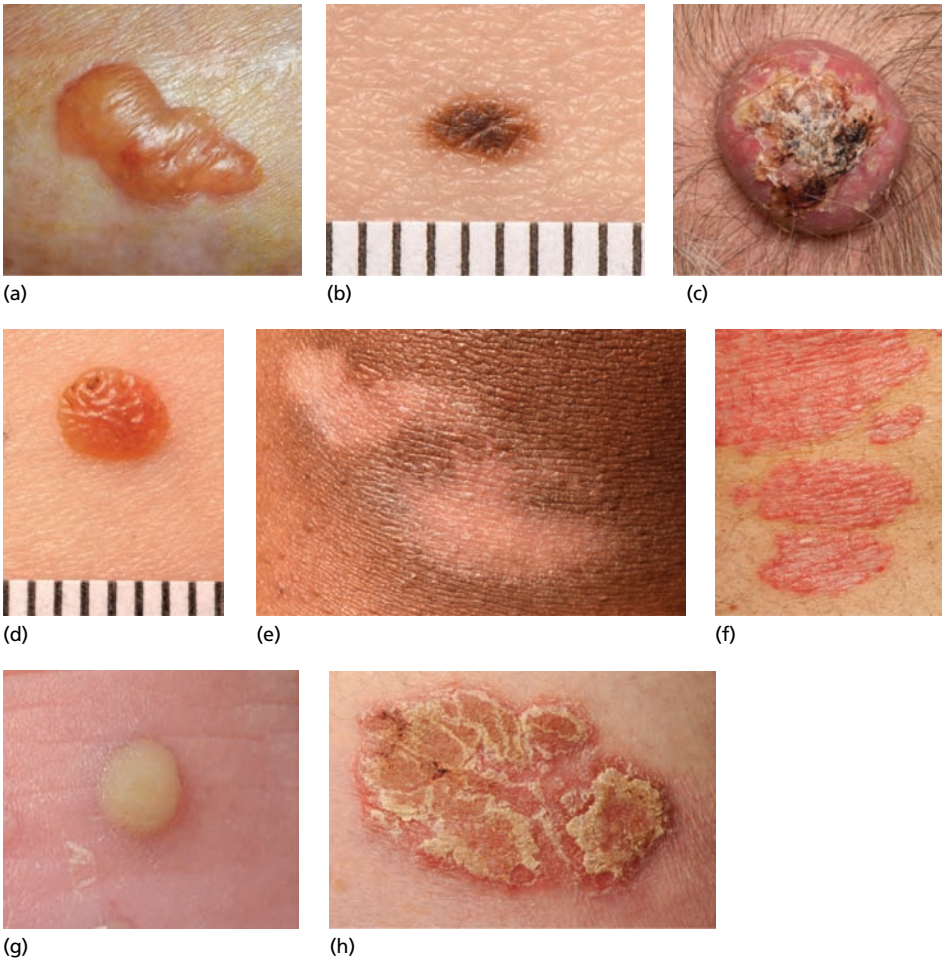


Figure 1.2 Types of cutaneous lesion: (a) bulla: bullous pemphigoid; (b) macule: junctional naevus; (c) nodule: squamous cell carcinoma; (d) papule: intradermal naevus; (e) patch: vitiligo; (f) plaque: psoriasis; (g) pustule: palmoplantar pustulosis; (h) scale: psoriasis. (Source: Reproduced with permission of Cardiff and Vale University Health Board.)

Bedside tests

Dermoscopy

(Syn. Dermatoscopy)

A dermatoscope (Syn. Dermoscope) provides polarised light and magnification to aid examination of the skin. The dermatoscope lens is applied to the skin surface with a film of oil on the lesion to enhance examination of sub-corneal structures. Analysis of the colours and appearances of structural elements, such as the pigment network, is especially useful in the

diagnosis of pigmented lesions. The images may be viewed directly, photographed or recorded digitally for subsequent or sequential analysis. Dermatoscopes can also be useful in distinguishing haemangiomas, angiokeratomas, pigmented basal cell carcinomas and seborrhoeic keratoses. Additional uses include the identification of scabies mites and other parasitic infections. Trichoscopy is use of a dermatoscope for hair and scalp lesions, and aids diagnostic accuracy in scalp disorders.

Table 1.4 Shapes of cutaneous lesions (adapted from Nast et al. 2016)

Term	Definition	Example
Acuminate	Elevated lesion with a sharp point	Cutaneous horn
Annular (Figure 3a)	Shape of a ring (clear centrally)	Tinea corporis
Arciform, arcuate	A segment of a ring; arch-like	Erythema annulare centrifugum
Digitate	Finger-shaped	Digitate dermatosis, a form of parapsoriasis
Discoïd; nummular (Figure 3b)	Circular or coin-shaped	Nummular eczema
Linear	Lesional skin forming a band or line	Lichen striatus
Papillomatous	Lesion with multiple surface projections	Epidermal naevus
Pedunculated	Papule or nodule attached by a thinner stalk	Skin tag
Polymorphic	Variable sizes and shapes as well as types of lesions	Acne vulgaris
Polycyclic	Coalescence of several rings	Subacute cutaneous lupus erythematosus
Reticulate (Figure 3e)	Net-like or lacy pattern	Mucosal lichen planus
Serpiginous (Figure 3c)	Wavy pattern, reminiscent of a snake	Cutaneous larva migrans
Targetoid	Lesion composed of concentric rings	Erythema multiforme
Umbilicated	Lesion with a small surface depression	Molluscum contagiosum
Verruciform	Lesion with multiple projections resembling a wart	Viral wart

Source: Adapted from A. Nast et al. The 2016 International League of Dermatological Societies' revised glossary for the description of cutaneous lesions. *British Journal of Dermatology*, 2016, 174, pp. 1351–1358. John Wiley & Sons Ltd on behalf of the British Association of Dermatologists.

A dermatoscope can also be used in the assessment of nail fold capillaries in connective tissue diseases (e.g. dermatomyositis).

Skin swabs (bacterial/viral)

In a suspected bacterial infection skin swabs for bacteriology should be sent to confirm the organism and provide antibiotic sensitivities to guide antibiotic selection.

Detection of viral skin infection, for example herpes simplex, requires a specific viral swab for viral culture. Increasingly polymerase chain reaction (PCR) amplification of viral DNA/RNA can provide a diagnosis within hours.

Mycological sample collection

Skin. Scraping samples of surface scale from an active margin are taken with a disposable scalpel blade or banana-shaped scalpel. The scrapings should be transported in folded paper, which keeps the specimen dry, thus preventing overgrowth of bacterial contaminants.

Hairs. If tinea capitis is suspected, the hairs should be plucked with the roots intact; cut hairs are unsuitable. Brush samples from the scalp are excellent for culture, but with this technique microscopy is not possible.

Table 1.5 Distribution patterns of cutaneous lesions (adapted from Nast et al. 2016)

Term	Definition	Example
Acral	Lesions involving distal extremities (e.g. ears, nose, fingers and toes)	Acrocyanosis
Asymmetrical	Distribution pattern which lacks symmetry along an axis (e.g. the midline)	Lichen striatus
Blaschkoid; along Blaschko lines	Lesions occurring on embryonic growth lines (Blaschko lines)	Incontinentia pigmenti
Dermatomal (zosteriform)	Lesions confined to one or more dermatome (a segment of skin innervated by a single spinal nerve)	Shingles (herpes zoster)
Disseminated	Lesions distributed randomly over most of the body surface area	Viral exanthem
Exposed skin	Areas exposed to external agents (e.g. airborne allergens, irritants, sunlight)	Airborne allergic contact dermatitis
Extensor sites	Areas overlying muscles and tendons involved in extension (e.g. dorsal forearm, elbow, posterior upper arm)	Psoriasis
Flexural sites	Areas overlying muscle and tendons involved in flexion of joints (e.g. antecubital fossa)	Atopic dermatitis
Follicular	Lesions located within or around hair follicles	Keratosis pilaris
Generalised/widespread	Distributed over most of the body surface area (see above)	Viral exanthem
Intertriginous	Present in major body folds (axillae, submammary folds, inguinal creases, natal cleft)	Flexural psoriasis
Kobnerised (displaying Kobner phenomenon)	Lesions arranged in a distribution which reflects physical stimuli (e.g. scratching, sunburn)	Lichen planus
Palmo-plantar	Involving palmar and plantar skin	Palmo-plantar pustulosis
Periorificial (e.g. perioral, periorbital)	Involving the skin around orifices	Peri-oral dermatitis
Seborrhoeic	Involving areas with the highest density of sebaceous glands (e.g. scalp, face, upper trunk)	Seborrhoeic dermatitis
Sporotrichoid (Figure 3d)	Lesions occurring along lymphatic vessels, usually of arm or leg	<i>Mycobacterium marinum</i> infection
Symmetrical	Lesions occurring with symmetry along an axis, commonly the midline	Psoriasis

Source: Adapted from A. Nast et al. The 2016 International League of Dermatological Societies' revised glossary for the description of cutaneous lesions. *British Journal of Dermatology*, 2016, 174, pp. 1351–1358. John Wiley & Sons Ltd on behalf of the British Association of Dermatologists.



(a)



(b)



(c)



(d)



(e)

Figure 1.3 Shapes and distribution patterns of skin lesions: (a) annular: tinea cruris; (b) discoid (round): nummular eczema; (c) serpiginous: cutaneous larva migrans; (d) sporotrichoid: *Mycobacterium marinum* infection; (e) reticulate: lichen planus on the lower lip. (Source: (a), (b) and (c) reproduced with permission of Cardiff and Vale University Health Board.)

Nails. Isolation of the pathogen from nail material is more difficult than in other samples. The full thickness of the nail should be sampled. Debris from under the nail is a fruitful source of material.

Wood's light

This is a source of UV light from which visible light has been excluded by a Wood's (nickel oxide) filter. Variations in epidermal pigmentation are more apparent under Wood's light than under visible light, whereas variations in dermal pigment are less apparent. For example, Wood's light accentuates the epidermal depigmentation of vitiligo, whereas the pallor from localised dermal vasoconstriction in naevus anaemicus disappears under Wood's lamp. Some organisms produce chemicals that fluoresce under Wood's lamp, including *Corynebacterium minutissimum*, the bacterium responsible for erythrasma

(Figure 1.4). Wood's light examination is a useful tool in the diagnosis of superficial mycoses, particularly infections due to *Microsporum* species which fluoresce green (Table 1.6).

Specific investigations

Biopsy

A biopsy of lesional skin provides essential information on the dermatopathology of almost all skin disorders. Sections from a paraffin-embedded biopsy specimen are usually stained with haematoxylin and eosin (H&E) for standard histopathological reporting. Special stains can be used to detect the presence of microorganisms (e.g. dermatophytes), the distribution of particular components of the skin (e.g. elastin) and the deposition of pathological substances (e.g. amyloid). Antibody deposition in the skin in immunobullous diseases is assessed

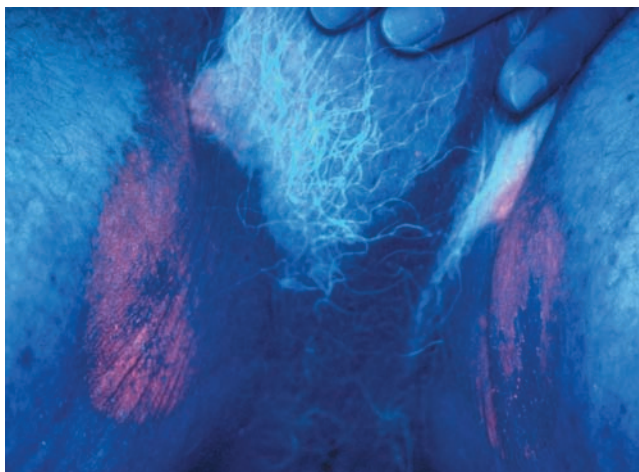


Figure 1.4 Wood's light illumination of erythrasma of the groins. The fluorescence in erythrasma is coral pink.

Table 1.6 Colour under Wood's light linked to clinical examples (adapted from Nast et al. 2016)

Colour under Wood's light	Clinical example(s)
Blue-green to yellow-green	Tinea capitis due to <i>Microsporum</i> spp.
Coral pink	Erythrasma
Red	Urine in some forms of porphyria
White	Well-developed lesions of vitiligo
Yellow to yellow-green	Pityriasis (tinea) versicolor

Source: Adapted from A. Nast et al. The 2016 International League of Dermatological Societies' revised glossary for the description of cutaneous lesions. *British Journal of Dermatology*, 2016, 174, pp. 1351–1358. John Wiley & Sons Ltd on behalf of the British Association of Dermatologists.

by direct immunofluorescence. Skin biopsies are processed specifically for this purpose; the immunofluorescence laboratory technique is performed on frozen sections.

Several different types of biopsy can be performed under local anaesthetic. Punch biopsies provide a small full thickness sample of skin, shave biopsies provide information on superficial skin layers and incisional biopsies may be used to sample a full-thickness section of a larger skin lesion or rash.

Patch tests

Patch tests are typically used to detect contact allergy of the delayed hypersensitivity type.

Multiple chemicals are applied to the patient's back to assess for an eczematous reaction. Patch tests are usually read at 2 days and 4 days (see Chapter 69). A patch test technique can be used to detect contact urticaria when the results are read at 15–30 min.

Prick tests

Prick testing investigates immediate type I hypersensitivity reactions. A small quantity of the test solution is placed on the skin and a prick is made through it with a sharp needle. The size of the weal and flare is measured after 15 min and compared with an adjacent control solution.

2

Introduction to dermatological therapeutics

Treatment modalities for skin disease comprise:

- Topical treatment
- Dressings (not discussed further in this book)
- Local injection
- Systemic agents (Chapter 82)
- Phototherapy
- Surgical removal of tissue
- Physical destruction of tissue such as with cryotherapy, cautery/diathermy, hyfrecation, and laser.

Topical treatment

There are different ‘vehicles’ (Box 2.1) that can be used to apply topical medication or emollients to the skin. As a rule, acutely inflamed skin is best treated with bland preparations that are least likely to irritate. Moist or exudative

eruptions are conventionally treated with lotions or creams, whilst dry skin responds well to ointments.

Prescribing topical treatment

The following should be considered when making a prescription for a topical treatment:

Prescription requirements: In general, a prescription of topical agent should comprise drug name, vehicle (cream or ointment), quantity to be dispensed, frequency, site of application and duration of treatment.

Frequency: Emollients should be applied frequently enough to maintain their physical effect, which may mean several applications per day. Active preparations are usually applied once or twice daily. Twice-daily application of topical corticosteroids is only marginally more effective than once-daily application.

Box 2.1 Different types of vehicle

Ointments: Semi-solid vehicles composed of lipid, such as white soft paraffin BP (petrolatum). Contain fewer preservatives than other vehicles. Occlusive and emollient properties.

Creams: Semi-solid emulsions containing both lipid and water. Emollient, lubricant and mildly occlusive.

Pastes: Semi-solid preparations containing a high proportion of finely powdered material such as zinc oxide or starch. Occlusive, protective and hydrating.

Lotions: Liquid formulations, usually simple suspensions or solutions of medication in water, alcohol or other liquids. Suitable for treating the scalp and other hairy areas of skin.

Gels: Thickened lotions. Suitable for treating the scalp and other hairy areas of skin.

Powders: Occasionally used to deliver drugs such as antifungal agents applied to the feet.

Paints: Liquid preparations which are usually applied with a brush to the skin or mucous membranes.

Dressings: Impregnated dressings, e.g. bandages containing ichthammol or zinc oxide, or tapes containing topical steroid.



Figure 2.1 The fingertip unit: from the distal crease of the forefinger to the ventral aspect of the fingertip

Quantity: The quantity of active topical agent (such as topical corticosteroids) needed for effective treatment should be explained to the patient. 'Fingertip units' are a useful guide for topical corticosteroid application (see Figure 2.1 and Table 2.1). Emollients should be applied more liberally.

Timing of application: Leave a suitable amount of time between emollient and active agent. This avoids dilution of the active medication and prevents spread over areas of skin where it is not required.

Potential hazards: Localised irritant or allergic reactions are the most frequent adverse effects (see Chapter 69). All topically applied drugs are absorbed to some degree, but systemic side effects are relatively rare. Absorption varies considerably depending on the region of skin being treated (absorption greatest from the genital area and least from the soles and palms). Occlusion greatly enhances drug penetration. Inflammation of the skin significantly increases drug absorption, especially in erythrodermic patients. Bath oils tend to make the bath slippery; paraffin-based ointments are flammable, which is a particular risk in smokers.

Table 2.2 lists frequently used topical treatments, but is not intended to provide an exhaustive list.

Topical corticosteroids

Topical corticosteroids (TCS) are the mainstay of treatment in eczematous dermatoses and are used either regularly or occasionally in the management of most inflammatory skin

diseases. TCS vary in potency from mild to very potent (Table 2.3). Classification of potency is important to predict response and possible adverse effects. Penetration of TCS is increased by occlusion using polythene film, dressings, gloves or bandages. This improves beneficial effects but also increases the potential for adverse effects.

Side effects

Significant side effects are rare, especially with short term use. The most common side effects are localised to application sites: skin atrophy, striae, erythema, telangiectasia and purpura; atrophic changes can become irreversible with long-term use of potent preparations. Areas most vulnerable to atrophy are those where the skin is already relatively thin, e.g. flexures and face.

Other local side effects are development of contact allergy, exacerbation of infection and if used on the face acneiform eruptions (see Chapter 40). It is advisable to avoid the use of topical corticosteroids in the presence of active viral infection, including herpes simplex, viral warts or molluscum contagiosum.

It is recommended that patients should use no more than 50 g of a superpotent steroid or 100 g of a potent steroid preparation per week and that prolonged usage at this high rate should be avoided to minimise the risk of systemic absorption leading to Cushing syndrome and hypothalamic-pituitary-adrenal axis suppression.

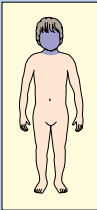
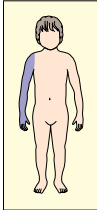
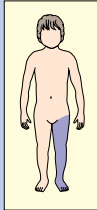
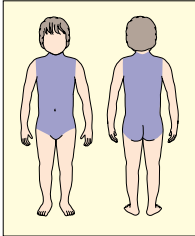
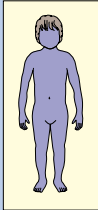
Rebound worsening of disease may occur when topical corticosteroids are withdrawn, particularly in psoriasis. This is most likely after withdrawal of potent or very potent corticosteroids.

'Steroid phobia' describes the fear of using topical corticosteroids which is out of proportion to the likelihood of side effects developing.

Topical calcineurin inhibitors

Topical calcineurin inhibitors (TCIs) have been developed for topical treatment of atopic eczema and have numerous additional applications. TCIs (e.g. tacrolimus and pimecrolimus) exhibit their anti-inflammatory effect by inhibition of calcineurin, which suppresses lymphocyte activation. They do not induce cutaneous atrophy. Theoretically, the local immunosuppression related to these

Table 2.1 Fingertip units required for a single treatment of various regions in children and adults (the unit is measured using an adult finger)

					
Age	Face and neck	One upper limb	One lower limb	Trunk (including buttocks)	Whole body
3–6 months	1	1	1.5	2.5	8.5
1–2 years	1.5	1.5	2	5	13.5
3–5 years	1.5	2	3	6.5	18
6–10 years	2	2.5	4.5	8.5	24.5
Adult	2.5	4.5	7.6	13.5	40

Source: Finlay AY, Edwards PH, Harding KG. 'Fingertip Unit' in dermatology. *Lancet*, 1989, 11, 155 and Long CC, Mills CM, Finlay AY. A practical guide to topical therapy in children. *Br J Dermatol* 1998, 138, 293–296.

Table 2.2 Different types of topical treatments and their main uses

Type of agent	Common example(s)	Indication
Antimicrobial agents	Alcohols: isopropyl alcohol, ethanol and <i>n</i> -propanol Benzalkonium chloride Chlorhexidine, iodine Antibiotics	Alcohols can be used for skin cleansing Present in antiseptic creams Used as a skin cleanser prior to surgery Topical antibiotics are frequently used in the treatment of superficial infections, acne vulgaris and rosacea
Antifungal agents	Allylamines (e.g. terbinafine), imidazoles (e.g. clotrimazole), morpholines (e.g. amorolfine) and polyenes (e.g. nystatin)	Treatment of mild dermatophyte and yeast infections
Antiparasitic agents	Pyrethroids (e.g. permethrin), malathion, dimeticone, Ivermectin	Treatment of lice infestations and scabies
Antiperspirants	Aluminium chloride	Hyperhidrosis of the axillae, palms and soles
Antiviral agents	Aciclovir, podophyllin, cidofovir	Treatment of herpes simplex virus types I and II, treatment of genital warts
Astringents	Aqueous solutions of potassium permanganate, aluminium acetate, and silver nitrate	Used to reduce exudation by precipitation of protein. These also have antiseptic properties.
Calcineurin inhibitors	Tacrolimus, pimecrolimus	Licensed for treatment of atopic eczema, especially in facial and flexural areas Also used in multiple other inflammatory dermatoses
Corticosteroids	Topical: see Table 2.3 Intralesional, e.g. triamcinolone	Topical: mainstay of treatment in eczematous dermatoses and other inflammatory dermatoses Intralesional: recalcitrant dermatoses, e.g. alopecia areata, keloid scars, lichen simplex, nodular prurigo
Cytotoxic and antineoplastic agents	5-fluorouracil, diclofenac, ingenol mebutate, imiquimod	Actinic keratosis, Bowen disease, superficial BCC, viral warts (refer to individual drug regarding indication)
Depigmenting agents	Hydroquinone, azelaic acid	Melasma
Emollients	White soft paraffin	Used to protect, lubricate and moisturise dry skin Also use instead of soap ('soap substitute') on inflamed skin
Keratolytic agents Miscellaneous agents	Salicylic acid, urea Brimonidine Capsaicin Dithranol Nicotinamide/nicotinic acid	To treat hyperkeratosis Rosacea Neuralgia, nodular prurigo, other localised intractable itch Psoriasis Acne vulgaris

(Continued)

Table 2.2 (Continued)

Type of agent	Common example(s)	Indication
Retinoids	Retinoic acid (tretinoin), isotretinoin, adapalene, Bexarotene	Acne Early plaque stage mycosis fungoides
Sensitising agents	Dinitrochlorobenzene, diphenycprone	Treatment of alopecia areata and warts
Soothing agents	Menthol, calamine	To help soothe itching and discomfort
Special dermatological formulations ('specials')	Examples: Coal tar solution BP 5% w/w in betamethasone valerate 0.025% w/w ointment 100 g Salicylic acid 5% w/w/propylene glycol 47.5% w/w in Dermovate® cream 100 g Reflectant (Dundee) sunscreens (available in coffee, coral pink, beige) 50 g	For moderate-severe psoriasis of trunk and limbs when other treatments such as vitamin D analogues have been ineffective For use on palmoplantar skin for hyperkeratotic eczema, palmopustular pustulosis and psoriasis not responding to Clobetasol propionate and emollients alone To treat photosensitivity disorders where the patient is sensitive to visible light, e.g. solar urticaria and porphyrias
Tars	Coal tar	Psoriasis
Vitamin D analogues	Calcipotriol, calcitriol, maxacalcitol, tacalcitol	Psoriasis

Table 2.3 Potency of some common topical corticosteroids as per *British National Formulary*

Topical corticosteroid potency	Examples
Very potent	Clobetasol propionate Diflucortone valerate 0.3%
Potent	Beclometasone dipropionate 0.025% Betamethasone valerate 0.1% Fluocinolone acetonide 0.025% Hydrocortisone 17-butyrate Mometasone furoate 0.1% Fluticasone propionate
Moderate	Betamethasone valerate 0.025% Clobetasone butyrate Fludrocortide 0.0125% Fluocinolone acetonide 0.00625% Alclometasone dipropionate 0.05%
Mild	Hydrocortisone 1% Hydrocortisone 2.5% Fluocinolone acetonide 0.0025%