

Methodic elaboration for practical lesson in
Dermatovenerology
for students of Medicine Faculty nr.2
Topic N1

Anatomy, histology and physiology of the skin.
The methodology of dermatological examination.
The basis of treatment in dermatology.

I. ANATOMY, HISTOLOGY AND PHYSIOLOGY OF THE SKIN

The skin can be divided into three main functional areas. These are the:

- **epidermis** – the major protective layer derived from the fetal ectoderm;
- **dermis** – the major support layer – of mesodermal original;
- **skin appendages** composed of cells derived from both ectoderm and mesoderm (hair follicle, sebaceous gland, apocrine sweat gland, eccrine sweat gland, and nails).
- in addition a fourth area, the subcutaneous **fat**, may be involved in deeply situated skin lesions such as erythema nodosum.

THE EPIDERMIS

The epidermis consists of many layers of closely packed cells, the most superficial of which are flattened and filled with keratins; it is therefore a stratified squamous epithelium. The epidermis contains no blood vessels. It varies in thickness from less than 0.1 mm on the eyelids to nearly 1 mm on the palms and soles. As dead surface squames are shed (accounting for some of the dust in our houses), the thickness is kept constant by cells dividing in the deepest (*basal* or *germinative*) layer. A generated cell moves, or is pushed by underlying mitotic activity, to the surface, passing through the *prickle* and *granular cell layers* before dying in the *horny layer*. The journey from the basal layer to the surface (epidermal turnover or transit time) takes 30 to 60 days. During this time the appearance of the cell changes. Keratinogenesis and melanogenesis are considered the main functions of epidermis.

These are the layers of the epidermis:

- **cornified or horny layer** - outer non-nucleated barrier layer which can be deviated in conjunctum and disjunctum parts. Stratum conjunctum represent a compact part of the horny layer, which attends to the granular layer, and stratum disjunctum represent a superficial part of horny layer, which peels.
- **stratum lucidum**, (present only in areas of thick skin, which are found on the palms of the hands and the soles of the feet). It can sometimes be seen just above the granular layer and is composed of three to five layers of dead, flattened keratinocytes. The keratinocytes of the stratum lucidum do not feature distinct boundaries and are filled with eleidin, an intermediate form of keratin.
- **granular layer** – the zone of 4 - 6 cell rows where keratinocytes lose their nuclei and their cytoplasm appears granular. Lipids, contained into those keratinocytes within lamellar bodies, are released into the extracellular space through exocytosis to form a lipid barrier. Those polar lipids are then converted into non-polar lipids and arranged parallel to the cell surface. For example glycosphingolipids become ceramides and phospholipids become free fatty acids.

- **spinosus or prickle cell layer** – the bulk of the living epidermal keratinocytes which become connected through desmosomes and start produce lamellar bodies, from within the Golgi, enriched in polar lipids, glycosphingolipids, free sterols, phospholipids and catabolic enzymes.
- **bazal layer** – a single row of keratinocytes in normal epidermis which undergo cell division. It is the deepest layer, rests on a basement membrane, which attaches it to the dermis. It is a single layer of columnar cells, whose basal surfaces sprout many fine processes and hemidesmosomes, anchoring them to the *lamina densa* of the basement membrane. In normal skin some 30% of basal cells are preparing for division (growth fraction). Following mitosis, a cell enters the G1 phase, synthesizes RNA and protein, and grows in size. Later, when the cell is triggered to divide, DNA is synthesized (S phase) and chromosomal DNA is replicated. A short post-synthetic (G2) phase of further growth occurs before mitosis (M). DNA synthesis continues through the S and G2 phases, but not during mitosis. The G1 phase is then repeated, and one of the daughter cells moves into the suprabasal layer. It then differentiates, having lost the capacity to divide, and synthesizes keratins. Some basal cells remain inactive in a so-called G0 phase but may re-enter the cycle and resume proliferation. The cell cycle time in normal human skin is controversial; estimates of 50–200 h reflect differing views on the duration of the G1 phase. Stem cells reside amongst these interfollicular basal cells and also amongst the cells of the external root sheath at the bulge in the hair follicle at the level of attachment of the arrector pili muscle. There is decreasing mitotic activity of keratinocytes in the aging skin.

Cell types seen in the epidermis

Keratinocytes

The keratinocytes are the main cell type and forms the great bulk of the cells in the epidermis. The main function of keratinocytes from the basal layer is germinative. In normal skin, keratinocyte division takes place only in the basal layer, so mitotic figures should not normally be seen above this level. After cell division, one daughter keratinocyte remains in the basal layer and the other moves upwards through the epidermis. The keratinocyte is committed to terminal differentiation and death. Within the prickle cell layer, highly specialized cellular bridges, called desmosomes connect the keratinocytes to each other, and these can frequently be seen under the light microscope at high power. They may be particularly easy to see in a biopsy from early dermatitis where there is a lot of epidermal oedema.

There are no desmosomes between keratinocytes and melanocytes, or Langerhans cells, or Merkel cells. There are hemidesmosomes between the basal layer keratinocytes and the underlying basement membrane.

In the granular layer, the living keratinocytes are involved in a complex series of biochemical changes during which the cell nuclei disintegrate, forming the

granules seen in the cytoplasm. Above this level there is the non-nucleated stratum corneum, or cornified layer.

Development and maturation of normal healthy epidermis in this pattern is called orthokeratosis, and produced an outer layer of non-nucleated, dead, flat keratinocytes. In some mucosal sites the normal maturation pattern is different, and there is no granular layer, but an outer layer of nucleated squamous cells. The term for this pattern is physiological parakeratosis (psoriasis).

It is calculated that the transit time for a daughter keratinocyte in the basal layer of normal skin to reach the outer surface is around 50-75 days. In psoriasis this is reduced to 8-10 days.

The strength of the epidermis depends on the cohesion of the keratinocytes. They produce a structural protein, alpha-keratin, which aggregates to form tonofilaments. These tonofilaments are continuous with the desmosomes and are easily seen in the electron microscope as large cytoplasmic bundles. Another communication channel between keratinocytes is a gap junction, tiny channels, which connect the cytoplasm of neighboring cells to each other.

The basal layer of the epidermis synthesizes keratin 5 and 14, and genetic disturbances in the genes coding for these keratin causes the disease epidermolysis bullosa simplex, while the suprabasal keratinocytes synthesize keratin 1 and 10, and abnormalities give rise to bullous ichthyosiform erythroderma or epidermolytic hyperkeratosis.

Melanocytes – the pigment-producing cells which are derived from neuroectoderm and founded in the basal layer. The melanocyte has multiple dendrites and these dendritic processes stretch between adjacent keratinocytes. On facial skin there may be as many as one melanocyte for every five basal layer keratinocytes, but in the lower back skin this ratio is usually closer to one in 20. The percentage of the melanocytes in the epidermis is about 10 - 12%. Numbers of melanocytes are the same in equivalent body sites in white and black skin, but the rate of production of pigment and its distribution is different. Melanocytes synthesize the pigment melanin. Melanin granules are seen on ultrastructural examination as small, black, electron-dense, intracytoplasmic structures – the melanosomes. The pigment is formed from DOPA on premelanosomes and this biochemical reaction is catalyzed by the presence in the melanocyte of the enzyme dopa-oxidase and tyrosinase, which are not present in surrounding keratinocytes or other non-melanocytic cells.

Langerhans cells – are dendritic cells of the skin and mucosa which can be found in all layers of the epidermis (most prominent in the stratum spinosum) and occur in the papillary dermis – an important immunologically competent cell. The percentage of the Langerhans cells in the epidermis is near 3 - 8 %. They are immunologically competent and may act as antigen present cells. L.C. in normal skin is the only cell to express MNS class 2 antigens and carry receptors for complement.

Merkel cells are non - dendritic cells of the epidermis which are placed between keratinocytes of basal layer. Their exact normal function is not known but is

thought to be related to cutaneous sensation. Merkel cells are sometimes considered a part of dispersed neuroendocrine system and a member of the amine precursor uptake and decarboxylation (APUD).

THE BASEMENT MEMBRANE

The basement membrane divides the epidermis from the dermis, and is a complex multi-layered structure. Hemidesmosomes attach the basal layer keratinocytes to the lamina lucida (laminin here) area. Below this level is the sub-basal dense plate through which anchoring filaments (type 7 collagen here) connect the lamina lucida to the lamina densa (type 4 collagen here).

THE DERMIS

The dermis lies between the epidermis and the subcutaneous fat and consists of two layers: papillary and reticular. The dermis supports the epidermis structurally and nutritionally. Its thickness varies, being greatest in the palms and soles and least in the eyelids and penis. In old age, the dermis thins and loses its elasticity, the collagen fibers to become is thinner and blood supply decrease.

Like all connective tissues the dermis has three components: cells, fibres and amorphous ground substance. There are a several main cell types in the dermis:

- **the fibroblast** – produced collagen, elastin and mucopolysaccharides;
- **the fibrocyte** - a bloodborne cell able to leave the blood, enter tissue and become a fibroblast;
- **the histiocyte (the tissue macrophage)** – acts as a general scavenger;
- **the mast cell (mastocyte)** – an important cell in type 1 immunological reactions and interactions with the eosinophil.

The dermis is largely made up of interwoven fibres, principally of collagen, packed in bundles. Collagen fibers are composed of glycin, hydroxyprolin and praline. Those in the papillary dermis are finer than those in the deeper reticular dermis. When the skin is stretched, collagen, with its high tensile strength, prevents tearing, and the elastic fibres, intermingled with the collagen, later return it to the unstretched state. Collagen makes up 70–80% of the dry weight of the dermis. Reticulin fibres are fine collagen fibres, seen in foetal skin and around the blood vessels and appendages of adult skin. Elastic fibres account for about 2% of the dry weight of adult dermis. They have two distinct protein components: an amorphous elastin core and a surrounding ‘elastic tissue microfibrillar component’. Elastin (molecular weight 72 kDa) is made up of polypeptides (rich in glycine, desmosine and valine) linked to the microfibrillar component through their desmosine residues. Dermis proteoglycans surround this fibrous material and embedded within the dermis are the: vasculature, lymphatics, nerves and small quantities of smooth and striated muscle. The exclusive functions of the dermis are strength, elasticity and plasticity.

- **The blood supply** - The blood vessels lie in two main horizontal layers. The deep plexus is just above the subcutaneous fat, and its arterioles supply the

sweat glands and hair papillae. The superficial plexus is in the papillary dermis and arterioles from it become capillary loops in the dermal papillae. An arteriole arising in the deep dermis supplies an inverted cone of tissue, with its base at the epidermis, but vessels don't get into epidermis. The blood vessels in the skin are important in thermoregulation. Under sympathetic nervous control, arteriovenous anastomoses at the level of the deep plexus can shunt blood to the venous plexus at the expense of the capillary loops, thereby reducing surface heat loss by convection. In normal skin **the lymphatic drainage system** is not visible, but this is also a profuse network, running from the reticular dermis to the local lymph nodes.

- **Cutaneous nerves** - Both free nerve ending and specialized receptors will be seen in the dermis. These nerve endings are important for the sense of touch, heat, cold, and preoccupation. Nerve endings are best seen on light microscopy with a silver stain, and are numerous in the papillary dermis. Neurocutaneous receptors of touch and mechanical stimuli are Meissner corpuscle, Merkel cell-nerve complexes and Pacinian corpuscle deep pressure, fast vibrations. Neurocutaneous receptors of thermoregulation are Ruffini corpuscle and Krause corpuscle.

SUBCUTANEOUS FAT

Fat is a major component of the human body and approximately 80% of fat is in the subcutis; the rest surrounds internal organs. In non-obese males, 10–12% of body weight is fat, while in females the figure is 15–20%. Fat comprises white and brown adipose tissue. Brown fat is more common in infants and children and is characterized by different mitochondrial properties and increased heat production. The function of fat is to provide insulation, mechanical cushioning and an energy store. In addition, fat may have an endocrine function, communicating with the hypothalamus via secreted molecules such as leptin to alter energy turnover in the body and to regulate appetite. Adipocytes also have important signalling roles in osteogenesis and angiogenesis, and additional physical functions such as phagocytosis. Multipotent stem cells have been identified in human fat, which are capable of developing into adipocytes, osteoblasts, myoblasts and chondroblasts. Molecular biological insight into genes, proteins, hormones and other molecules that influence fat deposition and distribution are gradually being realized, both from research on rare inherited disorders (such as the lipodystrophies or obesity syndromes) as well as population studies on more common forms of obesity.

THE SKIN APPENDAGES

These are pilosebaceous unit, the eccrine sweat glands, and the nails. The pilosebaceous unit includes the hair follicle, the sebaceous gland and erector pili muscle and the apocrine gland which excretory duct opens into hair follicle in some sites, for example axilla.

The hair follicle is the result of interaction between downgrowth of fetal ectoderm, which will form the hair shaft, and the vascular hair bulb papilla, which

is derived from fetal mesoderm. The hair follicles are present on the most surface of the body besides the lips, glans penis, labia minora, palms and soles.

Hairs are classified into three main types:

1. Lanugo hairs - fine long hairs covering the foetus, but shed about 1 month before birth.
2. Vellus hairs - fine short unmedullated hairs covering much of the body surface. They replace the lanugo hairs just before birth.
3. Terminal hairs - long coarse medullated hairs seen, for example, in the scalp or pubic regions. Their growth is often influenced by circulating androgen levels.

Each follicle passes, independently of its neighbours, through regular cycles of growth and shedding. There are three phases of follicular activity:

1. Anagen – the active phase of hair production.
2. Catagen – a short phase of conversion from active growth to the resting phase. Growth stops, and the end of the hair becomes club-shaped.
3. Telogen – a resting phase at the end of which the club hair is shed.

The duration of each of these stages varies from region to region. On the scalp, said to contain an average of 100 000 hairs, anagen lasts for up to 5 years, catagen for about 2 weeks and telogen for about 3 months. As many as 100 hairs may be shed from the normal scalp every day as a normal consequence of cycling. The proportion of hairs in the growing and resting stages can be estimated by looking at plucked hairs (a trichogram). On the scalp, about 85% are normally in anagen and 15% in the telogen phase. The length of hair is determined by the duration of anagen (e.g. the hairs of the eyebrows have shorter cycles than those of the scalp). Each hair follicle goes through its growth cycles out of phase with its neighbours, so there is no moulting period. However, if many pass into the resting phase (telogen) at the same time, then a correspondingly large number will be shed 2–3 months later.

The sebaceous glands – are clusters of cells with a small dark nucleus and a foamy cytoplasm. They cluster around the hair shafts and their secretion is formed by total destruction of the cells, a mechanism called holocrine secretion. Sebaceous glands secrete the oily, waxy substance called sebum that is made of triglyceride oils, wax, squalene, carbohydrates and cholesterol. This secretion drains into the hair follicle and is discharged on the surface through the hair follicle opening. Sebaceous glands occur all over the body except on the palms and soles. They are usually connected to hair follicles forming the pilosebaceous unit. In some regions, isolated sebaceous glands without hairs are observed. These “ectopic” glands appear as yellowish-white papules of 1 mm diameter. They are called Meibomian glands on the eyelid, Tyson’s glands on the prepuce or the labia minora, Montgomery glands on the mammary areola and Fordyce spots on the lips and buccal mucosa.

The apocrine sweat glands are found predominantly in the axilla, with a few seen also in the skin of the groin, genitalia perianal region and areola. They have a secretory component seen in the deeper dermis. The secretory section has a very wide lumen, and the cells lining this lumen will be seen to be composed of columnar epithelium which appears to form glandular secretion by a ‘nipping off’ of the tops of the cells, a process known as decapitation secretion. The apocrine sweat glands have a holomerocrine mechanism of secretion, which is formed by partial destruction of the cells. The excretory channels of the apocrine glands most commonly drain into the canal of the hair shaft and sebum passes from here out on to the surface of the epidermis.

The eccrine sweat glands are seen on the most body sites, and are anatomically independent from the other appendages. They are absent on the glans penis, internal surface of the prepuce and the labium minus. Their secretory components are much smaller than those of the apocrine gland, with a smaller lumen. They have a merocrine mechanism of secretion, which is formed without of cellular destruction. The excretory duct of these gland winds upward in a spiral pattern through the dermis and epidermis to the surface. Normal sweat consists of water, minerals, lactate and urea.

The nails are very highly modified skin appendages. The nail itself, or nail plate, grows out from the nail matrix and rests on the underlying nail bed. The pale halo at the proximal end of the nail is called the lunula, and around the edge of this there is a protective rim of cuticle.

Nails may become involved in a number of skin diseases, such as psoriasis and fungal infection. The average time for a fingernail to grow out completely from base to outer edge is approximately 6 months, and for toenails the time is 6-18 months or even longer. Thus, therapy of diseased nails requires both time and patience.

II. THE METHODOLOGY OF DERMATOLOGICAL EXAMINATION

PRIMARY AND SECONDARY LESIONS

PRIMARY LESIONS.

1. **Macule** (Latin: *macula*, “spot”) A macule is a circumscribed area of change in skin color without elevation or depression. It is thus not palpable. Macules can be welland ill-defined. In depending on the color the spots can be divided into: white, as in vitiligo; brown, as in cafe-au-lait spots; blue, as in Mongolian spots; red, as in permanent vascular abnormalities such as port-wine stains or capillary dilatation due to inflammation (erythema).

Macules may be the result of hyperpigmentation, hypopigmentation, vascular abnormalities, capillary dilatation (*erythema*), or *purpura* (*extravasated red blood cells*) that is why the spots can be divided into:

- Pigmented (induced by disruption to the production and distribution mechanism of melanin)
 - *hyperpigmented* such the *cafe au lait-colored* macules of neurofibromatosis; *lentigo* which is also called age spots; *melasma*, generally due to hormone imbalances etc.
 - *hypopigmented*, as in vitiligo.
- Vascular (caused by vascular dilatation)
 - *erythema* is redness caused by vascular dilatation, due to inflammation. Disseminated small erythematous macules occur in exanthemas such as *roseola* in syphilis and drug eruptions.
 - *telangiectasia* is the visible, permanent, dilatation of small cutaneous blood vessels (lupus erythematosus, dermatomyositis, rosacea, etc.).
- Hemorrhagic (caused by extravasated red blood cells)
 - *Petechiae* are small, pinpoint (pinhead size) spots that often seen in thrombocytopenic states
 - *Purpura* (up to 2 mm in diameter)
 - *Ecchymoses* are larger, bruise-like purpuric lesions.

The application of pressure with a glass slide (*diascopy*) on the border of a red lesion is a simple and reliable method for differentiating redness due to vascular dilatation (*erythema*) from redness due to extravasated erythrocytes or erythrocyte products (*purpura*). If the redness remains under pressure of the slide, the spots is hemorrhagic.

Some authors in depending on the sizes subdivide spots on:

- a *macule* - a less than 5 mm in diameter,
- a *patch* - larger than 5 mm in diameter

2. Papule (Latin: *papula*, “pimple”) A papule is a superficial, elevated, solid lesion, generally considered <0.5 cm in diameter. Most of it is elevated above, rather than deep within, the plane of the surrounding skin. A papule is palpable. It may be well- or ill- defined. In papules the elevation is caused by metabolic or locally produced deposits, by localized cellular infiltrates, inflammatory or noninflammatory, or by hyperplasia of local cellular elements. Superficial papules are sharply defined. Deeper dermal papules have indistinct borders. Papules may be dome-shaped, cone-shaped or flat-topped (as in lichen planus. Papules may have different color: red – psoriasis; copper - secondary syphilis; violet - lichen planus; yellow – xanthomatosis. A rash consisting of papules is called a *papular exanthem*. Papular exanthems may be grouped (“lichenoid”) or disseminated (dispersed).

Confluence of papules leads to the development of larger, usually flat-topped, circumscribed, plateau-like elevations known as plaques (French: *plaque*, “plate”). See below.

- 3. Plaque** is an elevated area of skin greater than 2 cm in diameter but without substantial depth. Plaques are a mesalike elevation that occupies a relatively large surface area in comparison with its height above skin level. Plaques are often formed by a confluence of papules, as in psoriasis. The typical psoriatic lesion is a raised, erythematous plaque with layers of silvery scale, often described as micaceous.
- 4. Nodule** (Latin: *nodulus*, “small knot”) is a palpable, solid, round or ellipsoidal lesion that is larger than a papule (>0.5 cm in both width and depth). Depending upon the anatomic component(s) primarily involved, nodules are of five main types:
- epidermal (keratoacanthoma, verruca vulgaris, basal cell carcinoma);
 - epidermal-dermal (nevi, malignant melanoma, invasive squamous cell carcinoma, mycosis fungoides);
 - dermal (granuloma annulare, dermatofibromas);
 - dermal-subdermal (erythema nodosum, superficial thrombophlebitis);
 - subcutaneous (lipomas).

Nodules in the dermis and subcutis may indicate systemic disease and result from inflammation, neoplasms, or metabolic deposits in the dermis or subcutaneous tissue. For example, late syphilis, tuberculosis, the deep mycosis, xanthomatosis, lymphoma, and metastatic neoplasms all can present as cutaneous nodules. A gumma is the granulomatous nodular lesion or tertiary syphilis. Nodules may be well defined (superficial) or ill defined (deep); if localized in the subcutaneous tissue, they can often be better felt than seen. Nodules can be hard or soft upon palpation. They may be dome-shaped and smooth or may have a warty surface or crater-like central depression.

- 5. Wheals.** A wheal is rounded or flat-topped elevated lesion that is characteristically evanescent, disappearing within hours. The epidermis is not affected: there is no scaling. The borders of a wheal, although sharp, are not stable, and in fact move from involved to adjacent uninvolved areas over a period of hours. These lesions, also known as hives or urticaria, are the result of edema in the upper portion of the dermis. Wheals are pale red in color, but if the amount of edema is sufficient to compress superficial vessels, they may be white, especially in the center. Wheals may be tiny papules 3-4 mm in diameter, as in cholinergic urticaria, or giant erythematous plaques of 10-12 cm, as in some cases of urticaria caused by penicillin hypersensitivity. Wheals occur in many shapes: round, oval, serpiginous, annular. Stroking of the skin may produce wheals in some normal persons; this phenomenon is called dermographism and is one of the physical urticarias. When it is associated with significant itching, it is called symptomatic dermographism.

Angioedema is a deep, edematous urticarial reaction that occurs in areas with very loose dermis and subcutaneous tissue, such as the lip. It may occur on the hands and feet as well, and may cause grotesque deformity. A careful search should be made for laryngeal edema, which may cause airway obstruction.

- 6. Vesicle and Bulla (Blister)** (Latin: *vesicula*, “little bladder”; *bulla*, “bubble”). A vesicle is a circumscribed, elevated lesion that contains fluid. Often the vesicle walls are so thin that they are translucent and the serum, lymph, blood, or extracellular

fluid is visible. A vesicle with a diameter greater than 0,5 cm is a bulla. Vesicles and bullae arise from cleavage at various levels of the skin; the cleavage may be within the epidermis, or at or below the dermal-epidermal interface. Cleavage just beneath the stratum corneum produces a subcorneal vesicle or bulla, as in impetigo.

Intraepidermal vesication may result from intercellular edema (spongiosis), as characteristically seen in delayed hypersensitivity reactions of the epidermis (contact eczematous dermatitis) and in dishidrotic eczema. *Spongiotic vesicles* may be detectable microscopically but may not be clinically apparent as vesicles.

Loss of intercellular bridges, or desmosomes, is known as *acantholysis*, and this type of intraepidermal vesication is seen in pemphigus vulgaris, where the cleavage is usually just above the basal layer. In pemphigus foliaceus the cleavage occurs just below the sub-corneal layer.

Viruses cause a curious “ballooning degeneration” of epidermal cells, as in herpes zoster, herpes simplex, variola, and varicella. Viral bullae often have a depressed (“umbilicated”) center.

Pathologic changes at the dermal-epidermal junction may lead to subepidermal vesicles and bullae, as are seen in pemphigoid, bullous erythema multiforme, porphyria catanea tarda, dermatitis herpetiformis, and some forms of epidermolysis bullosa. The thickness of the wall of bulla may be estimated by its translucency and flaccidity. The amount of pressure required to collapse the lesion may help predict whether the bulla is intraepidermal or subepidermal. It has been said that a relatively large, tense bulla suggests pemphigoid, whereas a flaccid bulla suggests pemphigus. There is, however, no reliable means of distinguishing these two diseases except by histologic examination of the lesion and immunofluorescence. When epidermis is lost, usually as a result of vesication, the circumscribed denudation is known as an erosion and appears as a moist, slightly depressed lesion.

7. Pustule (Latin: *pustula*, “pustule”) is a circumscribed, raised lesion that contains a purulent exudate. Pus, composed of leukocytes with or without cellular debris, may contain bacteria or may be sterile, as in the lesions of pustular psoriasis. Pustules may vary in size and shape and, depending on the color of the exudate, may appear white, yellow, or greenish yellow. Follicular pustules are conical, usually contain a hair in the center, and generally heal without scarring.

Pustules are characteristic of rosacea, pustular psoriasis, Reiter- s disease, and some drug eruptions, especially those due to bromide or iodide. Vesicular lesions of some viral diseases (varicella, variola, vaccinia, herpes simplex, and herpes zoster), as well as the lesions of dermatophytosis, may become pustular. A Gram-stain and culture of the exudate from pustules should always be performed.

SECONDARY LESIONS.

- 1. Erosions.** An erosion is a moist, circumscribed, usually depressed lesion that results from loss of all or a portion of the viable epidermis. After the rupture of vesicles or bullae, the moist areas remaining at the base are called erosions. Extensive areas of denudation due to erosions may be seen in bullous diseases such as pemphigus. Unless they become secondarily infected, erosions usually do not scar. If inflammation extends into the papillary dermis, an ulcer is present and scarring results, as in vaccinia and variola, and less often in herpes zoster and varicella.
- 2. Ulcers.** An ulcer is a lesion in which there has been destruction of the epidermis and at least the upper (papillary) dermis. Certain features that are helpful in determining the cause of ulcers and that must be considered in describing them include location, borders, base, discharge, and any associated topographic features of the lesion or surrounding skin such as nodules, excoriations, varicosities, hair distribution, presence or absence of sweating, and adjacent pulses. Stasis ulcers are accompanied by pigmentation and, occasionally, by edema or sclerosis. Ulceration occurs in granulomatous nodules of various types due to deep fungi, tuberculosis, syphilis, and yaws, as well as in a variety of parasitic and bacteriologic disorders. Nodules adjacent to ulcerations suggest granulomatous or neoplastic disease.
- 3. Fissures** are linear cleavages or cracks in the skin and may be painful. They occur particularly in palmar/plantar psoriasis and in chronic eczematous dermatitis of the hands and feet, especially after therapy that has caused excessive drying of the skin. Fissures are frequently noted in perianal psoriasis or at the angles of the mouth (perleche). Perleche may be caused by avitaminosis, moniliasis, ill-fitting dentures, or unknown factors.
- 4. Scar** occurs wherever ulceration has taken place and reflects the pattern of healing in those areas. Scars may be hypertrophic or atrophic. They may be sclerotic, or hard, as a consequence of collagen proliferation. The scarred epidermis is thin, generally without normal skin lines and without appendages. A depressed scar may resemble the primary atrophy. Scars may occur in the course of acne, some porphyrias, herpes zoster, and varicella. Raynaud's disease, syphilis, tuberculosis (especially on the face), leprosy, or carcinoma may produce mutilations, or a loss of tissue that alters major anatomic structures.
- 5. Desquamation (scaling).** Abnormal shedding or accumulation of stratum corneum in perceptible flakes is called scaling. Under normal circumstances the epidermis is completely replaced every 27 days. The end product of this holocrine process of keratinization is the cornified cell of the outermost layer of the skin – the stratum corneum. The cornified cell is packed with filamentous proteins, normally does not contain a nucleus and is usually lost imperceptibly. When keratinocytes production occurs at an increased rate, as in psoriasis, immature keratinocytes that retain nuclei reach the skin surface – this is called parakeratosis. Parakeratotic cells may pile up and contribute to the formation of scales. In psoriasis, scales may appear in

thin, mica-like sheets or accumulate massively, suggesting the appearance of an oyster shell. Densely adherent scales that have a gritty feel like sandpaper are typically seen in solar keratosis. Fishlike scale occurs in a group of disorders known as ichthyoses, in some of which prolonged retention of the stratum corneum occurs even though it is produced at a normal rate. Scaling lesions also occur in dermatophyte infections, pityriasis rosea, secondary and tertiary syphilis.

- 6. Crusts (encrusted exudates).** Crusts result when serum, blood, or purulent exudate dries on the skin surface, and are characteristic of pyogenic infections. Crusts may be thin, delicate, and friable, or thick and adherent. Crusts are yellow when formed from dried serum, green or yellow-green when formed from purulent exudate, or brown or dark red when formed from blood. Crusts may be present in acute eczematous dermatitis and impetigo (honey-colored, glistening crusts). When the exudate or crust involves the entire epidermis, the crusts may be thick and adherent: this condition is known as ecthyma. A scutula is a small, yellowish, cup-shaped crust especially characteristic of superficial fungal infection of the scalp caused by *Trichophyton schoenleinii*.
- 7. Excoriations** are superficial excavations of epidermis that may be linear or punctate and result from scratching. They are findings in all types of pruritus and are concomitants of pruritic skin disease, such as atopic eczema, dermatitis herpetiformis, or infestations.
- 8. Lichenification.** Repeated rubbing, especially in people with chronic eczema, leads to areas of lichenification. Proliferation of keratinocytes and stratum corneum, in combination with changes in the collagen of the underlying dermis, causes lichenified areas of skin to appear as thickened plaques with accentuated skin markings. The lesions may resemble tree bark.
- 9. Pigmentation,** either more or less than surrounding skin, can develop after lesions heal.

Having identified the lesions as primary or secondary, adjectives can be used to describe them in terms of their other features.

1. Colour (e.g. salmon-pink, lilac, violet).
2. Sharpness of edge (e.g. well-defined, ill-defined).
3. Surface contour (e.g. dome-shaped, umbilicated, spire-like).
4. Geometric shape (e.g. nummular, oval, irregular, like the coast of Maine).
5. Texture (e.g. rough, silky, smooth, hard).
6. Smell (e.g. foul-smelling).
7. Temperature (e.g. hot, warm).

Dermatologists also use a few special adjectives which warrant definition.

1. *Nummular* means round or coin-like.
2. *Annular* means ring-like.
3. *Circinate* means circular.
4. *Arcuate* means curved.
5. *Discoïd* means disc-like.

6. *Gyrate* means wave-like.
7. *Retiform* and *reticulate* mean net-like.
8. *Targetoid* means target-like or ‘bull’s eye’.
9. *Polycyclic* means formed from coalescing circles, or incomplete rings.

To describe a skin lesion, use the term for the primary lesion as the noun, and the adjectives mentioned above to define it. For example, the lesions of psoriasis may appear as ‘salmon-pink sharply demarcated nummular plaques covered by large silver polygonal scales’. Try not to use the term ‘erythema’ as it means a shade of red and therefore is less specific than for example ‘fire engine red’. Try also not to use the terms ‘lesion’ or ‘area’. Why say ‘papular lesion’ when you can say papule? It is almost as bad as the ubiquitous term ‘skin rash’. By the way, there are very few diseases that are truly ‘maculopapular’. The term is best avoided except to describe some drug eruptions and viral exanthems. Even then, the terms ‘scarlatiniform’ (like scarlet fever – punctate, slightly elevated papules) or ‘morbilliform’ (like measles – a net-like blotchy slightly elevated pink exanthem) are more helpful.

Configuration

After unravelling the primary and secondary lesions, look for arrangements and configurations that can be, for example, discrete, confluent, grouped, annular, arcuate, segmental or dermatomal. Note that while individual lesions may be annular, several individual lesions may arrange themselves into an annular or polycyclic configuration. Other adjectives, discussed under the morphology of individual lesions, can apply to their groupings too. The Köbner or isomorphic phenomenon is the induction of skin lesions by, and at the site of, trauma such as scratch marks or operative incisions.

III. LABORATORY DIAGNOSIS

(direct microscopic examination, culture on Sabouraud’s medium, Wood’s light examination, Tzanck smear, skin biopsy, immunofluorescence microscopy, skin tests to suspected allergens, etc.).

SPECIAL TOOLS AND TECHNIQUES

1. A **Wood’s light**, emitting long wavelength ultraviolet radiation, will help with the examination of some skin conditions. Fluorescence is seen in some fungal infections, erythrasma and *Pseudomonas* infections. Some subtle disorders of pigmentation can be seen more clearly under Wood’s light (e.g. the pale patches of tuberous sclerosis, low-grade vitiligo and pityriasis versicolor, and the darker *café au lait* patches of neurofibromatosis). The urine in hepatic cutaneous porphyria often fluoresces coral pink, even without solvent extraction of the porphyrins (see Fig. 21.10).

2. **Diascopy** is the name given to the technique in which a glass slide or clear plastic spoon is pressed on vascular lesions to blanch them and verify that their redness is caused by vasodilatation and to unmask their underlying colour. Diascopy is also used to confirm the presence of extravasated blood in the dermis (i.e. petechia and purpura, the appearance of which do not change on pressure).
3. **Photography**, mostly digital nowadays, helps to record the baseline appearance of a lesion or rash, so that change can be assessed objectively at later visits. Small changes in pigmented lesions can be detected by analysing sequential digital images stored in computerized systems.
4. **Dermoscopy** (also called *epiluminescence microscopy*). A hand lens with built-in lighting and a magnification of 10× to 30× is called a *dermatoscope* and permits the noninvasive inspection of deeper layers of the epidermis and beyond. This is particularly useful in the distinction of benign and malignant growth patterns in pigmented lesions. *Digital dermoscopy* is particularly useful in the monitoring of pigmented skin lesions because images are stored electronically and can be retrieved and examined at a later date to permit comparison quantitatively and qualitatively and to detect changes over time. Digital dermoscopy uses computer image analysis programs that provide (1) objective measurements of changes; (2) rapid storage, retrieval, and transmission of images to experts for further discussion (teledermatology); and (3) extraction of morphologic features for numerical analysis. Dermoscopy and digital dermoscopy require special training.
5. **Cytology (Tzanck smear)** Cytology can aid the diagnosis of viral infections such as herpes simplex and zoster, and of bullous diseases such as pemphigus. A blister roof is removed and the cells from the base of the blister are scraped off with a No. 10 or 15 surgical blade. These cells are smeared on to a microscope slide, air-dried and fixed with methanol. They are then stained with Giemsa, toluidine blue or Wright's stain. Acantholytic cells are seen in pemphigus, and multinucleate giant cells are diagnostic of herpes simplex or varicella zoster infections. Practice is needed to obtain good preparations. The technique has fallen out of favour as histology, virological culture and PCR have become more accessible.
6. **Skin biopsy.** Biopsy (from the Greek *bios* meaning 'life' and *opsis* 'sight') of skin lesions is useful to establish or confirm a clinical diagnosis. A piece of tissue is removed surgically for histological examination and sometimes for other tests (e.g. culture for organisms). When used selectively, a skin biopsy can solve the most perplexing problem but, conversely, will be unhelpful in conditions without a specific histology (e.g. most drug eruptions, pityriasis rosea, reactive erythemas). Skin biopsies may be *incisional*, when just part of a lesion is removed for laboratory examination, or *excisional*, when the whole lesion is cut out. Excisional biopsy is preferable for most small lesions (up to 0.5 cm diameter) but incisional

biopsy is chosen when the partial removal of a larger lesion is adequate for diagnosis, and complete removal might leave an unnecessary and unsightly scar. Ideally, an incisional biopsy should include a piece of the surrounding normal skin although this may not be possible if a small punch is used. The main steps in skin biopsy are:

- a. Obtain written and informed consent from the patient before starting the procedure;
 - b. Administration of local anaesthesia; and
 - c. Removal of all (excision) or part (incision) of the lesion and repair of the defect made by a scalpel or punch.
7. **Patch testing** is used to document and validate a diagnosis of allergic contact sensitization and identify the causative agent. Substances to be tested are applied to the skin in shallow cups (Finn chambers), affixed with a tape and left in place for 24–48 h. Contact hypersensitivity will show as a papular vesicular reaction that develops within 48–72 h when the test is read. It is a unique means of in vivo reproduction of disease in diminutive proportions, for sensitization affects all the skin and may therefore be elicited at any cutaneous site. The patch test is easier and safer than a “use test” with a questionable allergen, for test items can be applied in low concentrations in small areas of skin for short periods of time.
8. **Prick testing** is used to determine type I allergies. A drop of a solution containing a minute concentration of the allergen is placed on the skin and the skin is pierced through this drop with a needle. Piercing should not go beyond the papillary body. A positive reaction will appear as a wheal within 20 min. The patient has to be under observation for possible anaphylaxis.
9. **Microscopic Examination of Scales, Crusts, Serum, and Hair** Gram stains of smears and cultures of exudates and of tissue minces should be made in lesions suspected of being bacterial or yeast (*Candida albicans*) infections. Ulcers and nodules require a scalpel biopsy in which a wedge of tissue consisting of all three layers of skin is obtained the biopsy specimen is divided into one-half for histopathology and one-half for culture. This is minced in a sterile mortar and then cultured for bacteria (including typical and atypical mycobacteria) and fungi.

IV. BASIC PATHOLOGIC REACTIONS IN THE SKIN

1. **Dyskeratosis** -This term relates to some abnormality in the process of epidermal cell keratinization. The changes usually consist of nuclear pyknosis and bright pink condensation of the cytoplasm of keratinocytes. The process occurs in two main contexts. Firstly, in malignant and premalignant epithelial lesions, such as squamous cell carcinoma, Bowen’s disease and solar keratosis. Secondly, in various forms of acantholytic

disorder such as Darier's disease. In this latter condition, specific types of dyskeratotic cell include corps ronds and grains

2. **Hyperkeratosis** refers to increased thickness of the stratum corneum, and may be associated with acanthosis of the Malpighian layer. Hyperkeratosis may occur in various disorders of keratinization, such as the keratodermas and some ichthyotic disorders, and relative hyperkeratosis is quite common in chronic discoid lupus erythematosus. The stratum corneum is normally thick on the palms and soles, and very thin, or even absent, around the eyelids and near mucous membrane junctions.
3. **Parakeratosis** can be defined as the retention of keratinocyte nuclei within the horny cell layer. It represents a disturbance of keratinization, and is normally associated with an absence or reduction in thickness of the granular cell layer. The histological feature of parakeratosis is commonly seen in many different forms of inflammatory dermatosis, and is closely associated either with increased epidermal cell turnover or with inflammatory changes in the epidermis itself. It is commonly seen in psoriasis and subacute eczematous reactions, and in conditions such as pityriasis lichenoides where the change reflects an earlier disturbance in the underlying epidermis. In chronic inflammatory conditions where epidermal turnover is unaffected, such as in lichenoid reactions, parakeratosis is rarely seen. Dysplastic epithelial changes, such as those occurring in actinic keratoses and Bowen's disease, are normally accompanied by parakeratosis.
4. **Hypergranulosis**- this refers to an increase in thickness of the granular layer of the epidermis, and is commonly accompanied by hyperkeratosis and acanthosis. It is often seen in chronic lichenification and lichen planus and related disorders.
5. **Acanthosis** -this term is used to describe an increase in number of cells in the Malpighian or prickle cell layer of the epidermis (from the Greek for prickle or thorn). Sometimes, a distinction is made between increased thickness of the epidermis due to enlarged keratinocytes (pseudoacanthosis) and true acanthosis due to increased numbers of keratinocytes. In practice, acanthosis is commonly used to cover both senses. Increased thickness of the epidermis may result from increased length of rete ridges, as in a psoriasiform tissue reaction, or may affect the whole epidermis, such as in lichenification. Acanthosis is commonly accompanied by other histological changes such as hypergranulosis, hyperkeratosis and papillomatosis.
6. **Spongiosis** is also known as intercellular oedema, and describes the widening of intercellular spaces between keratinocytes due to fluid accumulation. Spongiosis is the characteristic histopathological change seen in acute and subacute eczematous reactions, but is also seen in a wide variety of other conditions; when spongiosis is marked it leads to intraepidermal vesiculation. Spongiosis of follicular epithelium may be

associated with increased mucin deposition in the histopathological reaction pattern known as follicular mucinosis.

7. **Acantholysis** is the term used to describe loss of cohesion between keratinocytes, due to breakdown of intercellular bridges. It results in the formation of intraepidermal clefts, vesicles and bullae. It appears to be the primary pathological change in a group of diseases including pemphigus and its variants, Darier's disease, transient and persistent acantholytic dermatosis and warty dyskeratoma. The site of acantholysis in these disorders is important. In pemphigus foliaceus and pemphigus erythematosus, acantholysis is usually confined to the upper portion of the epidermis, whereas in pemphigus vulgaris the split is formed at a lower level in the epidermis. In benign familial pemphigus (Hailey–Hailey disease), although acantholysis is often focal or incomplete, where it does occur it tends to affect the full thickness of the epidermis. Acantholysis may also be seen secondary to some other pathological change, where there is alteration or damage to epidermal cells. It may occur, for example, in bullous impetigo, viral disorders, solar keratoses and some forms of squamous cell carcinoma. When acantholysis occurs in these disorders, the term secondary acantholysis is often used to distinguish the process from primary acantholysis, which occurs in pemphigus and related diseases.
8. **Ballooning degeneration** -this form of degeneration of keratinocytes is associated with marked swelling and pallor of individual cells, with loss of intercellular bridges. A blister forms as a result of the consequent acantholysis. Ballooning degeneration along with reticular degeneration is characteristic of virus disorders affecting epithelia, such as herpesvirus infections.
9. **Hydropic degeneration** -this is also known as liquefaction degeneration, and refers to a vacuolar change that affects the basal cell layer of the epidermis. Small droplets and vacuoles develop within and in between basal cells. The process is commonly associated with pigmentary incontinence, and when marked may lead to subepidermal blister formation. It occurs typically in the whole range of lichenoid tissue reactions, including lupus erythematosus, lichen planus, dermatomyositis etc.
10. **Exocytosis** -this term describes the migration of inflammatory cells from the blood vessels of the dermis into the overlying epidermis. The process may be associated with spongiosis, as in eczema, or occur in the absence of spongiosis, such as may be seen in mycosis fungoides. In the later setting, the word epidermotropism is usually preferred.
11. **Papillomatosis** -this change is characterized by elongation upwards of the dermal papillae, giving an accentuated and sometimes irregular, undulating configuration to the dermal–epidermal junction. The feature is commonly seen in psoriasis, and a wide variety of other inflammatory and neoplastic cutaneous disorders.

V. THE BASIS OF TREATMENT IN DERMATOLOGY:

The treatments used in dermatology can be deviated into:

- Drugs
 - Topical
 - Systemic
- Physical
 - Surgical
 - Excision
 - Curettage
 - Intralesional injection
 - Electrodesiccation
 - Cryotherapy
 - Radiotherapy
 - Phototherapy
 - Laser therapy

1. **TOPICAL TREATMENT** has the advantage of direct delivery and reduced systemic toxicity. It consists of a vehicle or base which often contains an active ingredient.

- **VEHICLES (BASES)**
 - **Powder** -is either the pure drug by itself (talcum powder), or is made of the drug mixed in a carrier such as cornstarch or corncob powder.
 - **Paste** - combines three agents - oil, water, and powder. It is an ointment in which a powder is suspended.
 - **Lotion** are similar to solutions but are thicker and tend to be more emollient in nature than solution. They are usually an oil mixed with water, and more often than not have less alcohol than solutions. Lotions can be drying if they contain a high amount of alcohol. There is a significant variability in the ingredients of base of generic lotions when compared to brand name lotions.
 - **Shake lotion** - a mixture that separates into two or three parts with time. Frequently an oil mixed with a water-based solution. Needs to be shaken into suspension before use. "Shake well before use".
 - **Gel** is thicker than a solution. Gels are often a semisolid emulsion in an alcohol base.
 - **Cream** is an emulsion of oil and water in approximately equal proportions. It penetrates the stratum corneum outer layer of skin well. Cream is thicker than lotion, and maintains its shape when removed from its container. It tends to be moderate in moisturizing tendency. For topical steroid products, oil-in-water emulsions are common. Creams have a significant risk of causing immunological sensitization due to

preservatives. It has a high rate of acceptance by patients. There is a great variation in ingredients, composition, pH, and tolerance among generic brands.

- **Ointment** is a homogeneous, viscous, semi-solid preparation, most commonly a greasy, thick oil (oil 80% - water 20%) with a high viscosity, that is intended for external application to the skin or mucous membranes. Ointments have a Water number that defines the maximum amount of water that it can contain. They are used as emollients or for the application of active ingredients to the skin for protective, therapeutic, or prophylactic purposes and where a degree of occlusion is desired.

Vehicles and their properties

Base	Used on	Effect	Points of note
Dusting powders	Flexures (may be slightly moist)	Lessen friction	If too wet, clump and irritate
Alcohol-based application (tinctures)	Scalp hair	Clean vehicle for corticosteroid application	Cosmetically elegant, do not matt hair May sting raw areas
Watery and shake lotions	Acutely inflamed skin (wet and oozing)	Drying, soothing and cooling	Tedious to apply Frequent changes (lessened by polyethylene occlusion) Powder in shake lotions may clump
Creams	Both moist and dry skin	Cooling, emollient and moisturizing	Short shelf life Fungal and bacterial growth in base Sensitivities to preservatives and emulsifying agents
Ointments	Dry and scaly skin	Occlusive and emollient	Messy to apply, soil clothing Removed with an oil
Pastes	Dry, lichenified and scaly skin	Protective and emollient	Messy and tedious to apply (linen or calico needed) Most protective if applied properly
Sprays	Weeping acutely inflamed skin Scalp	Drying, non-occlusive	Vehicle evaporates rapidly No need to touch skin to treat it
Gels	Face and scalp	Vehicle for corticosteroids, salicylic acid and tretinoin	May sting when applied to inflamed skin can be covered by makeup
Mousse	Scalp	Clean vehicle for corticosteroid application	Does not matt the hair

▪ ACTIVE INGREDIENTS

These include corticosteroids, tar, dithranol, antibiotics, antifungal and antiviral agents, benzoyl peroxide, retinoic acid and many others (Table 1). The choice depends on the action required, and prescribers should know how each works.

Table 1 An overview of topic therapy

Drug	Indications	Pharmacology
Corticosteroids	Eczemas, psoriasis, lichen planus, discoid lupus erythematosus, sunburn, pityriasis rosea, mycosis	Mode of action is through vasoconstrictive, anti-inflammatory and anti-proliferative effects; medication is available in different

	fungoides, photodermatoses, lichen sclerosus	strengths; side-effects need to be considered
Antiseptics	Skin sepsis, leg ulcers, infected eczema	Chlorhexidine, benzalkonium chloride, silver nitrate and potassium permanganate are used
Antibiotics	Acne, rosacea, folliculitis, impetigo, infected eczema	Chlortetracycline, neomycin, bacitracin, gramicidin, polymixin, sodium fusidate, and mupirocin are available; resistance and sensitization are potential problems. Metronidazole is used for rosacea
Antifungals	Fungal infections of the skin, <i>Candida albicans</i> infections	Nystatin, clotrimazole, miconazole, econazole, terbinafine, ketoconazole, sulconazole and amorolfine are available
Antiviral agents	Herpes simplex, herpes zoster	Aciclovir, penciclovir
Parasiticidals	Scabies, lice	Benzyl benzoate, permethrin and malathion for scabies; malathion, permethrin and carbaryl for lice — applied as lotion or shampoo
Coal tar	Psoriasis, eczema	Presumed anti-inflammatory and anti-proliferative effects; available as creams, shampoos and in paste bandages
Dithranol	Psoriasis	Anti-proliferative effects; available as creams, pastes and ointments
Vitamin D analogues	Psoriasis	Calcipotriol and tacalcitol inhibit keratinocyte proliferation and promote differentiation; creams and ointments available
Keratolytics	Acne, scaly eczemas	Salicylic acid, benzoyl peroxide and tretinoin
Retinoids	Acne, psoriasis	Isotretinoin [acne), tazarotene (psoriasis)

2. SYSTEMIC THERAPY

Are used particularly for the more serious conditions and for infections. Details are given in table 2:

Table 2 An overview of systemic therapy

Group	Drug	Indications
Corticosteroids	Prednisolone usually	Bullous disorders, connective tissue vasculitis
Cytotoxics	Methotrexate Hydroxyurea Azathioprine	Psoriasis, sarcoidosis Psoriasis Bullous disorders, chronic actinic dermatitis

		eczema
Immunosuppressants	Ciclosporin	Psoriasis, atopic eczema, pyoderma gangrenosum
	Gold	Bullous disorders, lupus erythematosus
Immunomodulators	Inosine pranobex	Viral warts (genital), herpes simplex (genital)
Retinoids	Acitretin	Psoriasis, other keratinization disorders
	Isotretinoin	Acne
Antifungals	Griseofulvin, terbinafine	Fungal infection
	Ketoconazole	Fungal infection (<i>C. alb/cans</i> too)
	Itraconazole, fluconazole	Fungal infection, candidiasis
Antibiotics	Various	Skin sepsis, acne, rosacea
Antivirals	Aciclovir, valaciclovir	Herpes simplex, herpes zoster
	Famciclovir	Herpes zoster, genital herpes simplex
Antihistamines	H 1 blockers	Urticaria, eczema
Antiandrogens	Cyproterone	Acne (females only)
Antimalarials	Hydroxychloroquine	Lupus erythematosus, porphyria cutanea tarda
Antileprotic	Dapsone	Dermatitis herpetiformis, leprosy, vasculitis

Other treatments

A wide variety of other, more specialized treatments exists for specific skin conditions. Corticosteroids are sometimes injected directly into lesions (e.g. to treat keloids). Certain disorders are responsive to ultraviolet B or photochemotherapy. In the past, X-irradiation was used to treat a range of skin conditions including psoriasis, acne, tinea capitis, tuberculosis of the skin and hand eczema. There are now very few indications for X-ray treatment of non-malignant disease, although irradiation is of great value in several types of skin tumour. Cryotherapy, in which liquid nitrogen is applied to the skin, is currently widely used in dermatology. It is mainly employed for the treatment of benign or pre-malignant skin tumours.

Basics of medical therapy

- Correct diagnosis is essential to ensure appropriate treatment.
- When using topical steroids:
 - a. use the lowest potency that is effective
 - b. look out for side-effects, especially atrophy
 - c. emollients can help reduce the steroid requirement.
- Explain the treatment to the patient and preferably give a written handout; this helps compliance. The fingertip unit is a convenient way to indicate the amount of cream to apply.
- Use the simplest treatment possible; patients easily get mixed up if they have several different tubes to use.
- Prescribe adequate amounts. Patients are
- often given too little, 'run out' of their creams, and return to clinic no better as the treatment has been inadequate.

