Dermatopathology: An abridged compendium of words. A discussion of them and opinions about them. Part 8 (P-S)

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The compendium (Part 8)

– P –

PAGET CELL: specific for mammary Paget’s disease and extramammary Paget’s disease, is typified by a large, round, or plump oval nucleus and pale-staining cytoplasm, the latter being more abundant in extramammary Paget’s disease because there it is replete with acid mucopolysaccharides that are demonstrable by staining with hematoxylin and eosin and better yet with stains specialized for them. Paget cells of mammary Paget’s disease and extramammary Paget’s disease have several attributes in common, but they are dissimilar, just as are mammary Paget’s disease and extramammary Paget’s diseases themselves, despite the fact that they share the word “mammary” and the name Paget’s disease.”

PAGET PATTERN: is scatter of cells constituent of mammary Paget’s disease and extramammary Paget’s disease throughout an epithelium, usually epidermal but also adnexal.

PAGETOID CELL: is one that resembles the cell constituent of Paget’s disease especially of extramammary Paget’s disease by having a large roundish nucleus and abundant pale cytoplasm. It is found, for example, in some lesions of melanoma and of Bowen’s disease.

PAGETOID MELANOCYTE: an abnormal melanocyte with abundant pale cytoplasm, but less abundant than in a ballooned melanocyte and such cells may be dispersed singly or in nests within the epidermis. They resemble the pale cells found within the epidermis of lesions of mammary and extramammary Paget’s disease. Pagetoid melanocytes may contain dusty particles of melanin. If they do not, reliable differentiation from true Paget cells depends on histochemical techniques. Paget cells are DOPA negative and contain either neutral mucopolysaccharides (in mammary disease) or sialomucin (in extramammary disease). Pagetoid melanocytes are DOPA-positive and do not contain mucin. They are usually seen in melanomas, especially those on the trunk and proximal parts of the extremities, but also are found in some melanocytic nevi, especially combined melanocytic nevi.

PAGETOID MELANOCYTIC PROLIFERATION: a phrase coined by Wallace H. Clark, Jr. and advocated by a panel of dermatopathologists appointed by the National Institutes of Health to forge definitions of terms employed in the language of melanocytic neoplasia, they being published in 1992. In brief “pagetoid melanocytic proliferation” was yet another attempt at evasion from a diagnosis by microscopy conventional of “melanoma in situ” and, like all the others, has faded into oblivion.

PAGETOID PATTERN: is descriptive of scatter of cells proliferative throughout an epithelium, usually epidermal but also adnexal, in the manner, that it is seen in mammmary Paget’s disease and extramammary Paget’s disease, e.g., in melanoma, Bowen’s disease, and mycosis fungoides. Thus pagetoid cells may or may not appear in pagetoid pattern in a given histopathologic section (i.e., melanoma).

PALE CELL: is one, whose cytoplasm is deficient in intensity of color, i.e., pallid, as seen in some examples of apocrine (pale-cell) hidradenoma, in pale cell acanthoma, and in pale cell acanthosis. (SEE CLEAR CELLS)
PALE-CELL ACANTHOSIS: refers to a specific histopathologic pattern within a thickened epidermis in which there are discrete zones of large pale-staining keratinocytes separated widely from one another by prominent intercellular spaces traversed by elongated bridges. Neutrophils usually pepper the foci of pale keratinocytes. In short, the changes of pale-cell acanthosis are identical to those that constitute the benign neoplasm named pale-cell acanthoma. (SEE CLEAR CELLS)

PALISADE: refers to the appearance of cells aligned in the fashion of stakes like those that form a line of defense in a fortification, i.e., columnar cells at the periphery of an outer sheath at the bulb of a normal hair follicle; germinative cells at the periphery of a “follicular” germ in an embryo, of aggregations of cells in a trichoblastoma, and of aggregations of cells in trichoblastic (basal cell) carcinoma; histiocytes around a zone of mucin and of degenerated collagen in granuloma annulare and around a zone of fibrin in rheumatoid nodule, and nuclei of Schwann cells apposed directly to one another at the distal end of their respective cytoplasm in Verocay bodies of schwannomas.

PANNICULITIS: an inflammatory disease in which the infiltrate of inflammatory cells is present in the panniculus adiposa (subcutaneous fat). For purposes of facilitating diagnosis by conventional microscopy of panniculitis, the process is divided arbitrarily as “septal” and “lobular,” depending on whether the infiltrate of inflammatory cells, as seen at scanning magnification, mostly is in fibrous septa or mostly in fat lobules. Sometimes, septa and lobules are affected equally. There can be significant overlap.

PAPILLARY: means shaped like a nipple. The root “pap” appears in several names for cutaneous structures, i.e., papillary dermis, dermal papilla, follicular papilla, papilloma, papillomatosis, papillations, and papillated. Nipple-like projections above the skin surface may be seen as an effect of proliferations of keratinocytes as in condyloma acuminata and of extension outward of papillary dermis, i.e., papillomatosis, as in acanthosis nigricans. Papillomatous and papillated are synonymous with papillary.

PAPILLARY DERMA: refers to the uppermost portion of the dermis that has nipple-like projections into hollows in the underside of the epidermis. Collagen in the papillary dermis is seen as delicate fibrils in contrast to collagen in the reticular dermis, which is arranged in bundles. Capillaries in the papillary dermis emerge from venules that are situated in the uppermost portion of the reticular dermis and, for that reason, this plexus is also known as the subpapillary plexus.

PAPILLATION: are projections above the skin surface or into the lumen of a structure tubular or cystic and that may be seen as an effect of proliferations of epithelial cells alone or of fibrous core protrusive covered by epithelial cells.

PAPILLOMA: refers to nipple-like projections above the skin surface as a consequence of extensions outward of dermal papillae (papillomatosis) that may or may not be covered by thickened epidermis. Cutaneous papillomas include those that have fibrous cores, i.e., fibroepithelial papillomas, and those that consist mostly of papillated epithelium, i.e., condylomata. The term “verrucous” sometimes is used synonymously with papillomatous, but the tips of dermal papillae in verrucous lesions tend to be pointed (digitated), i.e., verruca vulgaris, rather than rounded (papillomatous), i.e., condyloma acuminatum.

PAPILLOMATOUS: refers to a nipple-like projection above the skin surface seemingly as a consequence of extensions outward of dermal papillae (papillomatosis) that may or may not be covered by thickened epidermis. Cutaneous papillomas include those that have a fibrous core, e.g., fibroepithelial papillomas and those that consist mostly of epithelium papillate, e.g., condylomata acuminata. The term “verrucous” sometimes is used synonymously with papillomatous, but the tip of epidermal excrescences in lesions verrucous often are pointed (digitated), i.e., verrucae vulgares, rather than rounded (papillomatous), i.e., condyloma acuminatum.

PAPULE: a small, i.e., up to 1.0 cm, slightly raised, solid or cystic lesion. A papule may be formed by abnormalities mostly of surface epidermis, as in a plane wart, by changes mostly in infundibular epidermis and in the dermis adjacent to it, as in lichen planopilaris, by ones mostly in the papillary dermis, as in a lesion of lichen nitidus, by findings mostly in the reticular dermis, as in a small infundibular cyst (a milium), or by aberrations of both epidermis and dermis together, as in a lesion of conventional lichen planus. The surface of a papule maybe flat (i.e. lichenoid, as in lichen planus) as it presents itself usually, hemispherical, as in lichen nitidus, or conical, as in a variety of spongiotic dermatitides, such as allergic contact dermatitis. Papules may be either smooth, like that of an incipient lesion of guttate psoriasis, or scaly, as in a later lesion of guttate psoriasis in which the original cornified layer traversed by elongated bridges. Neutrophils usually pepper widely from one another by prominent intercellular spaces separated by discrete zones of large pale-staining keratinocytes. In short, the changes of pale-cell acanthosis are identical to those that constitute the benign neoplasm named pale-cell acanthoma. (SEE CLEAR CELLS)
should not be used unmodified because it is imprecise, confusing, and potentially harmful to patients; clinicians may be led wrongly to interpret an inflammatory disease as a lymphoma. In fact, there is no need for the term parapsoriasis because the actual diagnoses are either Mucha-Habermann disease or mycosis fungoides or whatever other named disease is being considered.

Parapsoriasis en plaques: is a synonym for one expression of the early patch stage of mycosis fungoides. It is a term that is no longer relevant. (See Parapsoriasis)

Part of a follicle: is a term applied to each of two units that constitute each of the two segments of a follicle. Each part has its own morphologic appearance, and each is delimited by distinct boundaries. From bottom up, the lower transient segment of a follicle is composed of two parts, namely, bulb and stem, and the upper permanent segment of isthmus.

Patch: a broad, i.e., more than 1.0 cm, flat lesion. A macule, in contrast to a patch, is a flat lesion less than 1.0 cm in greatest diameter. A patch may begin as a macule, as in vitiligo, or it may appear as a patch from the outset, such as a café au lait “spot.” Alterations histopathologic that are responsible for patches are the same as those for macules. A café au lait spot of neurofibromatosis results from an increase in melanin within the epidermis, a purple patch of an ecchymosis from extravasation of erythrocytes in the dermis, and a reddish patch of a blush or of a sunburn from erythrocytes congested within dilated blood vessels of the superficial plexus.

Pattern: refers to a design or arrangement of figures or structures. In clinical dermatology, pattern includes distribution of lesions, arrangement of lesions, configuration of lesions, and outline of individual lesions themselves. In dermatopathology, pattern pertains mostly to distribution and arrangement of infiltrates of inflammatory cells and to silhouettes formed by neoplastic cells. They are the vehicles used to arrive at the correct diagnoses with specificity.

In dermatopathology pattern usually refers to patterns of inflammatory cells. However, the concept of pattern is more far reaching. The following discussion illustrates this. Rather than conceive of distinctive expressions of pathologic processes as specific diseases, it may be more accurate, and, therefore, more illuminating, to think of them as “patterns of disease.” For example, dermatologists understand, full well, that conditions like erythema multiforme, erythema nodosum, and leukocytoclastic vasculitis represent repeatable
patterns of disease, clinically and histopathologically, and that each of those patterns can be brought into being by different causes, sometimes many of them, as in the case of erythema multiforme, in which causes are disparate as infection by herpes virus and a drug given systemically. And what has just been stated in regard to erythema multiforme, erythema nodosum, and leukocytoclastic vasculitis obtains equally for urticaria, Sweet’s syndrome, and pyoderma gangrenosum. On reflection further, the very same concept is applicable to psoriasis, lichen planus, and granuloma annulare as well as to all other apparently noninfectious inflammatory diseases of the skin and subcutaneous fat, from pityriasis rosea to nodular vasculitis. And reflection still further makes apparent that even so called infectious diseases really are patterns of disease! Is supplicative granulomatous dermitis and panincillicnus in conjunction with pseudocarcinomatous proliferation of keratocytes brought about by any number of different “deep fungi” and “atypical mycobacteria” not a pattern? And is staphylococcal scalded skin syndrome that stimulates, exactly, the appearance of “superficial pemphigus” (foliaceus/erythematous) not a pattern? And is it not true that except for the presence of hyphae, dermatophytosis may mimic all of the patterns of psoriasis histopathologic? In sum and in short, pattern analysis (in conjunction with an algorithmic method) is the surest route to diagnosis of all “inflammatory diseases” of the skin and subcutaneous fat, each of which may be thought of as a pattern morphologic of disease.

PAUTRIER’S “MICROABSCESS”: a collection of abnormal lymphocytes within the epidermis or epithelial structures of adnexa of lesions of mycosis fungoides; a misnomer because abscess denotes a collection of neutrophils, and Pautrier’s collections consist of abnormal lymphocytes.

PEDUNCULATE: a polypoid excrescence that is attached to the skin or other flat surface by a stalk, as is the case in a fibroepithelial polyp or papilloma.

“PEPPERING” BY LYMPHOCYTES: describes the scatter of lymphocytes as solitary units within an aggregation of epithelial cells of a proliferation, as occurs in spiradenoma and adamantinoid trichoblastoma, or within the epidermis as happens often in mycosis fungoides.

PERSISTENCE: in dermatopathology refers to the continuation of a proliferation benign or malignant, at a primary site after an attempt, usually surgical and unsuccessful, has been made to remove it. Most proliferations described as “recurrent” actually are just persistent. Recurrence should only be used when referring to true metastasis. (SEE RECURRENCE OF MELANOMA)

PETALOID: denotes the appearance histopathologic of aggregations of cells in a proliferation that resembles petals of a flower, as may be observed in some examples of trichoblastoma and trichoblastic (basal cell) carcinoma.

PETECHIA: a pinpoint punctum of extravasated red blood cells in the upper part of the dermis.

PHLEGMON: cellulitis brought into being usually by microaerophilic streptococci. The process tends to extend throughout the dermis and subcutaneous fat to skeletal muscle and other soft tissues. Histopathologically, it is seen to consist of a sparse, but diffuse, infiltrate of neutrophils, mostly. Phlegmonous inflammation differs from suppurative inflammation, which also consists mostly of neutrophils, by displaying sparse polymorphs in diffuse distribution, as in erysipelas, rather than a dense discrete collection of them.

PIGMENT “INCONTINENCE”: the loss of pigment from the epidermis due to damage of epidermal melanocytes and basal keratocytes and its ingestion by macrophages within the dermis. In some conditions, however, pigment transfer to dermal macrophages may be due to phagocytosis of the terminal portions of dendritic processes of melanocytes that have protruded through the basal lamina.

PIGMENTATION: coloration caused by a variety of pigments. Melanin is responsible for a great range of colors in skin, such as black (abundant melanin at all levels of the epidermis, including the cornified layer, as in some simple lentigines), blue (melanin within melanocytes and melanophages in the mid and deep parts of the dermis in many blue nevi), tan (sparse melanin in the epidermis of café au lait spots), gray (diffuse dermal melanosis associated with some metastases of melanoma), and ash melanin increased within the epidermis and present in macrophages within the upper part of the dermis as in a clinical expression of post inflammatory hyperpigmentation known as erythema dyschromicum perstans. Of course, erythema dyschromicum perstans (ashy dermatosis) is not an authentic disease just one manifestation of post-inflammatory pigment alteration. Differences in coloration among peoples also are due to melanin. Loss of epidermal melanin can be seen in diseases as diverse as vitiligo and post-inflammatory hypopigmentation. Skin colors in tattoos result from pigments other than melanin, such as silver (slate gray), carotene (yellowish orange), cobalt (blue), cadmium (yellow), mercury (red), carbon (black or blue), and chromium (green). The brown color of hemochromatosis is a consequence mostly of melanin in keratinocytes rather than of sparse hemosiderin deposited in macrophages.

PILAR: like tricho-, pertains to hair, (the former Latin and the latter Greek), in contrast to follicular, which refers specifically to an entire follicle including a hair. The two terms pilar and tricho-, on the one hand, and follicular, on the other, often are used interchangeably and, therefore, incorrectly (i.e., pilar
sheath acanthoma, trichoblastoma, and pilomatricoma). Each of those proliferations consists of cells non-viable like those in a follicle, not simply of cells non-viable like those of hair. Furthermore, there are no true pilar cysts (although hair shafts often are found in steatocystomas, eruptive vellus hair cysts, and some infundibular cysts), only ones epidermal (i.e., infundibular) and follicular (isthmic-catagen types).

**“PINCHING-OFF” SECRETION:** describes picturesquely the appearance of the distal secretory portion of luminal cells that line apocrine glands. The terms “apocrine” secretion, “decapitation” secretion, “pinching off” secretion, and “snouts” are synonyms.

**PLAQUE:** an elevated, broad, i.e., more than 1.0 cm in diameter, lesion. A plaque of psoriasis may come into being from extension centrifugally of a single papule or it may be a consequence of confluence of many papules. Plaques usually are acquired, as is the case in lichen simplex chronicus, hypertrophic lichen planus, and lichen amyloidosis, but some of them are congenital, as in a gar-ment type of congenital melanocytic nevus, an epidermal nevus like that known as ichthyosis hystrix, and a connective tissue nevus like a shagreen “patch.”

**PLASMA:** is the fluid portion of circulating blood. It is distinguished from serum, which is the fluid obtained after coagulation.

**PLASMOCYTOID:** refers to a cell that exhibits cytologic features similar to those of a plasma cell, i.e., an eccentrically placed nucleus associated with abundant amphophilic cytoplasm. Plasmocytoid cells are found often in some proliferations with apocrine differentiation such as apocrine hidradenoma and apocrine mixed tumor, (not uncommonly in conjunction with polygonal cells). These cells are also seen in benign proliferations, such as “classic” Spitz’s nevus and in malignant proliferations, such as melanoma and malignant apocrine mixed tumor.

**PLATITUDE:** (SEE Cliché)

**PLEOMORPHISM:** denotes variation in size and shape of cells, and particularly nuclei of them, in an infiltrate of a process proliferative. Pleomorphism is the determinant most important of nuclear atypia, the other characteristics conventional of them being large size of nuclei and degree of staining inconsistent markedly of them. No direct correlations exist, however, between pleomorphism of nuclei and behavior biologic of a proliferation. Nuclei may be strikingly pleomorphic in proliferations benign, such as in some “classic” Spitz’s nevi, and even in some conditions inflammatory, such as “monster cells” in an expression distinctive of dermatofibromas. Pleomorphism is not the same as polymorphism, which refers to different types of cells present together in a single infiltrate, i.e., neutrophils, eosinophils, lymphocytes, plasma cells, and histiocytes in a section of granuloma faciale.

**POLYGONAL CELL:** having the shape of a polygon that structure being a closed plane figure bounded by straight lines usually there are at least three sides and typically five or more. When a cell with such a shape is non-epithelial, it is likely to be an abnormal melanocyte (of “classic” Spitz’s nevus or melanoma), and when epithelial, it is likely to be an abnormal myoepithelial cell (as in mixed tumor of apocrine type, that neoplasm benign really being infundibulo-apocrine-sebaceous-follicular). Polygonal cells are often met in company with plasmocytoid cells.

**POLYMORPHIC ERUPTION:** more than, one type of skin lesion occurring concurrently in an individual, i.e., a combination of macules, papules, vesicles, and so on, as occurs in Mucha-Habermann disease.

**POLYPOID:** resembling a polyp, an excrescence above the skin surface having a narrow base and a stalk, such as an acrochordon (i.e., fibroepithelial polyp) or as in an apocrine fibroadenoma.

**POP OUT:** is a colorful description of the way some benign proliferations and cysts shoot from the skin after an incision made above is carried very near to them. The reason for ease of separation from surrounding tissue is that benign proliferations and cysts tend to compress adjacent fibrous tissue and, in the process, create a narrow space between them and adjacent nearly normal skin. The space, combined with the tract of the incision, becomes the route that a neoplasm or cyst takes as it flies from its haven. When a surgeon cradles some benign neoplasms and cysts in order to encourage them to “shell out,” the same space acts as the corridor for their release from the skin.

**POROID CELL:** is one with a round nucleus and scant cytoplasm situated lateral to a cuticular cell in a duct of a normal apocrine or eccrine gland, it usually being the component dominant (the other less conspicuous being a cuticular cell) in poromas and porocarcinomas, both ones apocrine and eccrine.

**PRECANCER (PREMALIGNANCY):** The term precancerous implies a benign stage in development of an authentic cancer. Skin lesions purported to be “precancerous” are solar keratosis, arsenical keratosis, radiation keratosis, Bowen’s disease (and its analogue on genitalia, erythrophasia of Queyrat), lentigo maligna, and extramammary Paget’s disease.
Leukoplakia is a generic term that traditionally has been used for precancerous of keratocytes on mucous membranes or mucocutaneous junctions. Are the conditions just enumerated precancerous, or are they cancers? All are malignant neoplasms.

Histopathologically and biologically, solar keratoses, arsenical keratoses, radiation keratoses, and Bowen’s disease are squamous cell carcinomas. Histopathologically, all fulfill criteria for squamous cell carcinoma, namely, keratinocytes that display crowded nuclei, pleomorphic nuclei, nuclei in mitosis, and premature cornification in the form of dyskeratotic cells. Biologically, each of those lesions may eventuate in metastases, a phenomenon that qualifies them as malignant. It is curious that dermatologists and pathologists the world over diagnose superficial basal cell carcinoma without hesitation but they name superficial squamous cell carcinomas by evasions such as “solar keratoses,” “arsenical keratoses,” “radiation keratoses,” and “Bowen’s disease.” If superficial basal cell carcinoma is considered to be cancer and not precancerous then should solar keratoses, arsenical keratoses, radiation keratoses, and Bowen’s disease be regarded as cancers. They are all, types of squamous cell carcinoma.

Histopathologically, solar keratoses sometimes are associated with suprabasal clefts above which reside acantholytic dyskeratotic cells. When that type of solar keratosis becomes thicker, histopathologists term the lesion pseudoglandular squamous cell carcinoma. No distinct histopathologic boundary exists, however, between any variant of solar keratosis and squamous cell carcinoma. It is not surprising, therefore, that in no textbook of dermatology, dermatopathology, or general pathology can a statement be found where solar keratosis ends and squamous cell carcinoma begins. The reason is that solar keratosis is a squamous cell carcinoma, albeit an “embryologic one.” The decision about whether a particular neoplasm is a solar keratosis or a squamous cell carcinoma is the mere whim and fancy of histopathologists. Because the judgment is entirely arbitrary, it is wholly without repeatability. What has just been written about solar keratosis applies equally to arsenical keratosis, radiation keratosis, Bowen’s disease and bowenoid papulosis—squamous cell carcinomas all.

Clinically, histopathologically, and biologically, lentigo maligna is melanoma. Clinically, the lesion, irrespective of size, is asymmetrical with notched borders and variegated color, usually shades of brown. Histopathologically, the neoplasm fulfills all of the criteria for melanoma in-situ, e.g. an increased number of melanocytes disposed as solitary units (and later in nests) within the epidermis (and epithelial structures of adnexa), solitary melanocytes that are not equidistant from one another (and neither are the nests), nests that usually have become confluent in foci, some melanocytes that are present above the dermoeipidermal junction, and nuclei of melanocytes that may be pleomorphic; that constellation of histopathologic findings signifies melanoma in-situ. Biologically, some examples of so-called lentigo maligna, once having extended into the reticular dermis, eventuate in metastases of melanoma. In brief, lentigo maligna is not a “precanceroses,” but a melanoma that is confined to the epidermis and epithelial structures of adnexa.

Extramammary Paget’s disease is not “precancerous,” but an apocrine carcinoma that begins within the epidermis and extends far down epithelial structures of adnexa (in the same manner as does melanoma). Although many lesions of extramammary Paget’s disease remain patches, i.e., in-situ, for the lifetime of the patient, some do not; neoplastic cells may descend into the dermis and from there metastasize.

In conclusion, a clinician cannot determine by gross examination alone which solar keratosis will eventually become a metastasizing squamous cell carcinoma, which “lentigo maligna” will become a metastasizing melanoma, or which extramammary Paget’s disease will metastasize as apocrine carcinoma. Each of these conditions is cancer, and each must be removed completely. No clinician or histopathologist can predict which lesion of solar keratosis, arsenical keratosis, radiation keratosis, Bowen’s disease, melanoma in-situ, or extramammary Paget’s disease, left untreated, will cause death as a consequence of metastasis. Clinicians should be educated further to appreciate the fact that radical surgery is not appropriate for any of these cancers; all that is required is total removal nothing more.

By sanitizing cancers with such terms as solar keratosis, lentigo maligna, and extramammary Paget’s disease, clinicians and pathologists sustain a belief in the erroneous concept of “precanceroses.” In actuality, solar keratosis is a squamous cell carcinoma, lentigo maligna is a melanoma, and extramammary Paget’s disease is an apocrine carcinoma.

In addition, there is neither need nor place for the word parapsoriasis in the language of dermatology and dermatopathology. The diseases now referred to as parapsoriasis can be diagnosed accurately as either Mucha-Habermann disease or mycosis fungoides. No disease termed parapsoriasis transforms into mycosis fungoides; it is either mycosis fungoides or it is Mucha-Habermann disease. This is a considerable advance beyond thinking of Brocq, who spawned the concept of parapsoriasis and who wrote these lines about it in 1903: “It is then quite evident that the group of parapsoriasis, such as I define it, established bonds of union between psoriasis and seborrhea psoriasiform on one side, and between lichen planus and the mild form of pityriasis rubra and mycosis fungoides on the other.”
PROLIFERATION: an increase in number, usually of cells. A proliferation of epithelial cells in skin may be divided into those that are epidermal and those that are adnexal. A proliferation of epidermal keratinocytes may be psoriasiform evenly, i.e., elongated rete ridges of about equal length, those alternating with dermal papillae of about equal length, as in psoriasis at its apogee, psoriasiform unevenly i.e., elongated rete ridges that are not of uniform length but that alternate nonetheless with dermal papillae to create an undulate pattern, as in longstanding nummular dermatitis, jagged, i.e., serrations at the base of a thickened epidermis, as in lichen planus, and mammillated, papillated, or digitated, i.e., the surface of the epidermis resembling breasts, nipples, or fingers, respectively, as in some examples of nevus sebaceous, acanthosis nigricans, and verruca vulgaris, respectively. “Verrucous” is a synonym for “digitate.” Proliferation of cells of epithelial structures of adnexa, i.e., the upper part of eccrine ducts and of infundibular epidermis, may be slight or marked the latter sometimes simulating squamous cell carcinoma (pseudocarcinomatous), as seen in some examples of halogenodermas, infections by deep fungi and atypical mycobacteria, and the verrucous stage of incontinentia pigmenti. Proliferation of melanocytes may be categorized according to an increase in the absolute number of normal appearing melanocytes per unit area of dermoepidermal junction or basal layer of epidermis, either as solitary units entirely or associated with nests.

Proliferation also may be a term generic for any increase in the number of cells other than ones inflammatory and, therefore, applicable to what are designated conventionally neoplasms (both benign and malignant), hyperplasias, hamartomas, malformations, structures ectopic, etc. No agreement has been reached after more than 150 years about definition of the above. Moreover, and more important, these definitions are not essential to diagnosis with specificity. Once the diagnosis with specificity has been accomplished by naming things for what they are such as in the case of verruca vulgaris with sebaceous differentiation, fibrofolliculoma/ trichodiscoma, nevus comedonicus, spiradenoma, and microcystic adnexal carcinoma, a knowledgeable clinician should understand immediately the implications biologic of the specific diagnosis just stated in regard to management of the patient.

PROLIFERATION OF ATYPICAL MELANOCYTES: an increase in number of melanocytes whose nuclei are atypical those cells being present in, and sometimes above, the basal layer of the epidermis and of epithelial structures of adnexa. The phrase is descriptive and does not convey a diagnosis with specificity; such a proliferation may occur in melanoma in situ, as well as in a junctional “classic” Spitz’s nevus.

PROMONTORY SIGN: describes a projection of a normal venule into a larger space created by a newly formed, often bizarre-shaped, thin-walled vessel in a patch or plaque of Kaposi’s sarcoma.

PSEUDOCANTHOLYSIS: the process whereby non-epithelial cells, melanocytes in particular of some examples of “classic” Spitz’s nevi and of some melanomas, are separated from one another in a manner reminiscent to that of acantholysis in which epithelial cells are detached from one another.

PSEUDOCAPSULE: refers to simulation of a capsule, i.e., a structure that envelops a proliferation. In general pathology, disagreement considerable exists concerning the terms “capsule” and “pseudcapsule.” Some histopathologists consider a capsule to be a membrane fibrous formed by stroma of normal tissue at the periphery of a proliferation that usually is benign. Other histopathologists regard a proliferation as encapsulated when the layer of fibrous tissue that surrounds it is intrinsic to the proliferation itself. Those latter pathologists contend that there are few truly encapsulated proliferations and they cite as examples of those few, schwannomas, thyromas, and thyroid adenomas. For them, the layer of fibrous tissue that surrounds most other proliferations may be ascribed to fibrous stroma of normal tissue and, therefore, qualifying as pseudocapsule. Neither a “capsule” nor a “pseudocapsule” is requisite for any diagnosis with specificity in dermatohistopathology. The alterations fibrous in point nearly always are caused by compression of surrounding normal tissue by a benign proliferation or cyst. Clefths tend to form between compressed fibrous tissue that surrounds a benign proliferation and the relatively normal tissue adjacent. As a consequence of such clefts encircling, a benign proliferation tends to “pop out” when incision is made sufficiently deep above it. The attributes just described are not seen as a rule in malignant proliferations. In sum, so-called capsule and pseudocapsule are merely compressed connective tissue.

PSEUDOCARCINOMATOUS HYPERPLASIA: is a proliferation of epithelial cells that, by silhouette, simulates a carcinoma, usually a squamous-cell carcinoma, but by cytologic features does not. The “Pseudocarcinomatous” appearance results from marked hyperplasia of epithelial structures of adnexa, i.e., infundibular epidermis and eccrine ducts. Those adnexal epithelial structures normally extend far into the dermis, and it is for that reason hyperplasia of them may result in simulation of an epithelial malignant neoplasm, i.e., a carcinoma. Although some keratinocytes in the pseudocarcinomatous hyperplastic epithelium may be in mitosis, nuclei are not strikingly crowded or pleomorphic, in contrast to the usual situation in many squamous-cell carcinomas.

Pseudocarcinomatous hyperplasia may be secondary to another pathologic process at that site, i.e., granular neoplastic cells in granular-cell schwannoma, infectious agents such as atypical mycobacteria in swimming-pool granuloma and
**PSEUDOMALIGNANCY:** malignancy is fundamentally a concept predicated on biologic behavior. A malignant neoplasm has the capability to kill either by destruction of tissue locally or by metastasis. The irrepressible behavior of malignant neoplasms is reflected in certain morphologic correlates, grossly (clinically) and histopathologically. Viewed through a microscope, those morphologic attributes usually are expressed as a particular silhouette and as certain cytologic features. As a rule, malignant neoplasms tend to be asymmetrical and poorly circumscribed, and to be composed of neoplastic cells disposed in aggregations that vary markedly in size and shape, that may have jagged outlines, and that tend to become confluent to form sheets of cells. Nuclei of cells that constitute malignant neoplasms often are pleomorphic, and some are in mitosis. Some mitotic figures may be abnormal.

The concept of pseudomalignancy is based on disparity between histopathologic appearance and biologic behavior of a condition. A pseudomalignancy has either the silhouette or the cytologic features usually associated with a malignant neoplasm, yet its behavior is uniformly and wholly benign. Pseudomalignancies usually are benign neoplasms, but, rarely, they may be inflammatory diseases, such as dermatofibromas with “monster cells.” Irrespective of the essential pathologic process, the term pseudomalignancy (pseudocarcinoma, pseudolymphoma, pseudosarcoma, and pseudomelanoma) is vague and descriptive of a benign condition; it is not a specific diagnosis framed in the language of clinical dermatology, i.e., trichoblastoma, response to the bite of a tick and “classic” Spitz’s nevus. Pseudomalignancies neither precede nor transform into authentic malignancies. The word should rarely be used.

Furthermore, pseudocarcinoma may refer to a benign neoplasm, a hyperplasia, or an inflammatory process that mimics a carcinoma, usually a basal cell carcinoma or a squamous cell carcinoma. For example, desmoplastic trichoepithelioma, a particular expression of trichoblastoma typified by columns of follicular germinative cells, often mimics a morpheaform basal cell carcinoma histopathologically; the hyperplasia of surface epithelium above a granular cell neoplasm may simulate squamous cell carcinoma, and a very early lesion of xanthogranuloma, an inflammatory process that consists of dense, diffuse infiltrates of histiocytes, some nuclei of which are pleomorphic and in mitosis, may resemble a poorly differentiated squamous cell carcinoma. What some authors consider to be “pseudocancerous,” i.e., florid oral papillomatosis, epithelioma cuniculatum, and giant condyloma, are really authentic verrucous carcinomas. “Solitary” keratoacanthoma cannot be a pseudomalignancy because it sometimes metastasizes as the squamous cell carcinoma that it is.

Pseudocarcinomatous hyperplasia refers specifically to silhouettes that may simulate those of either a basal cell carcinoma or a squamous cell carcinoma. A simulator of superficial basal cell carcinoma is follicular germ induced by fibrohistiocytic elements of dermatofibroma, and a mimic of squamous cell carcinoma is the adnexal epithelial hyperplasia often associated with infections by deep fungi and atypical mycobacteria. A distinction must be made between the terms pseudocarcinoma, on one hand, and pseudocarcinomatous hyperplasia on the other. The former is a clumsy attempt to render a specific diagnosis, whereas the latter is a description of adnexal epithelial hyperplasia the outline of which resembles a carcinoma. The term pseudocarcinomatous hyperplasia is not meant to be a specific diagnosis. Pseudocarcinomatous hyperplasia usually consists of a proliferation of infundibular and/or eccrine ductal epithelium that assumes the outline of a squamous cell carcinoma, but nuclei of spinous cells are neither crowded nor atypical.

The same principle that obtains for pseudomalignancy in general applies equally to pseudolymphomas of the B-cell type in which the architectural pattern resembles roughly that of a lymphoma of the B-cell type, namely, nodular aggregations composed mostly of lymphocytes distributed throughout the dermis and sometimes in the subcutaneous fat, but the lymphocytes are small and devoid of prominent nuclear pleomorphism. It has become increasingly apparent that many lesions on faces diagnosed as pseudolymphomas have proved to be authentic lymphomas. Lymphomatoid papulosis often is listed among the pseudolymphomas, but, in reality, it is a Ki-1 lymphoma. Actinic reticuloid, a persistent light reaction, does not mimic a lymphoma histopathologically, and neither does pityriasis lichenoides (Mucha-Habermann disease). The lymphocytes in actinic reticuloid and Mucha-Habermann disease are small and monomorphous.

Pseudosarcomas tend to have the silhouette of a benign neoplasm, but nuclei of neoplastic cells display pleomorphism, i.e., the mesenchymal cells (“floret cells”) of pleomorphic fibromas and pleomorphic lipomas.

There are several “pseudomelanomas,” only one of which is accompanied, and only episodically, with striking pleomorphism of nuclei, namely, “classic” Spitz’s nevus. That nevus, however, like all nevi, has the architectural pattern of a benign
neoplasm, to wit, it is symmetrical and sharply circumscribed. Some “classic” Spitz’s nevi have several architectural features in common with melanoma, such as a scatter of melanocytes at all levels of the epidermis, a phenomenon that is also encountered in the other distinctive melanocytic nevi such as con genital nevi biopsied shortly after birth; some junctional and compound nevi situated on the palms and soles; junctional and compound nevi situated on particular anatomic sites such as the nipple and areola, genitalia, perianal region, intertriginous regions, and umbilicus; and junctional and compound nevi that persists after inadequate surgical removal.

In sum, whenever possible the diagnoses should be made for what they are. The term “pseudo” anything has limited usefulness.

PSORIASIFORM: resembling a fully developed lesion of psoriasis clinically, i.e., a reddish plaque covered by scales, and/or histopathologically, i.e., elongated rete ridges of about equal length that alternate with long dermal papillae to form a strikingly undulate pattern. The concept of psoriasiform just stated is parochial, however, because it applies only to lesions formed fully of that remarkably protein disease. Clinical expressions of psoriasis range from smooth-surfaced guttate papules devoid of scale to exfoliative erythroderma, from discrete pustules on palms and soles to widespread pustules accompanied by signs and symptoms of systemic character, and from plaques with slight scale to “extremely thick side.” Each of these manifestations of psoriasis has an analogue histopathologically. In theory, therefore, simulation of any of the manifold expressions of psoriasis, clinically and histopathologically, is psoriasiform.

PUNCH BIOPSY: refers to a procedure used to obtain a cylindrical portion of skin that consists of epidermis and dermis, and even of subcutaneous tissue. The procedure is performed with an instrument known as a punch, the diameter of which may range from 2 to 8 mm and whose cutting edge is usually made of steel but sometimes of plastic. A sample of skin is taken by introducing the round blade of the punch into the skin, to which it is oriented perpendicularly, and, under pressure, rotating the instrument, back and forth, until it has penetrated the entire dermis and entered the subcutaneous fat. The procedure is simple and rapid and leaves a wound that is easy to close with a suture or that may heal with second intention and with little scar. Of course, there are many indication and contraindications for the use of this procedure. (SEE BIOPSY, RAZOR BLADE REMOVALS)

PURPURA: a purple color caused by hemorrhage in the skin. Petechiae are pinpoint hemorrhagic macules and ecchymoses are hemorrhagic patches. A hematoma is extensive hemorrhage in a somewhat discrete locus in the reticular dermis and/or the subcutaneous fat. Purpura can be classified as subsequent to inflammatory disease, as in Schamberg’s disease, leukocytoclastic vasculitis, and Mucha-Habermann disease, and as unaffiliated with an inflammatory disease, as is the case for solar purpura, thrombomocytopenia, and disseminated intravascular coagulopathy.

PUSHING MARGINS: an image used to convey the smooth, round borders of a well-circumscribed (usually benign) neoplasm that appears to grow centrifugally. This term is to be eschewed. It is a cliché since it cannot be seen under a microscope. (i.e., pushing).

PUSTULE: clinically, an elevated, circumscribed collection of pus (neutrophils and necrotic debris of neutrophils) and, histopathologically, a collection of neutrophils within an epithelium (surface and/or infundibular) and, much less often, an eccrine unit. A variety of names, some eponymic, have been given to types of pustules situated within cutaneous epithelia. Discrete pustules in the mid-spinous zone of psoriasis are termed “Munro’s microabscesses”; sponge-like pustules in the upper reaches of the epidermis, particularly of psoriasis and variants of it, ranging from acrodermatitis continua (Hallopeau) through keratoderma blennorrhagicum to subcorneal pustular dermatitis (Sneddon and Wilkinson), are known as spongiform pustules of Kogoj; abscesses positioned beneath the cornified layer of an epidermis are referred to as subcor neal pustules. None of those types of pustules is specific for anyone disease. So-called Munro’s micro abscesses also may be seen in dermatophytosis, spongiform pustules in halogenodermas, and subcorneal pustules in pyoderma gangrenosum. The various appearances of pustules histopathologically reflect a moment in time at which a biopsy interrupted migration of neutrophils from capillaries in dermal papillae to the cornified layer of the epidermis, which is their destination ultimately. For reasons not understood, neutrophils in eruptive (guttate) psoriasis hone to the summit of mounds of parakeratosis.

Most pustules in skin are infundibular epidermal rather than surface epidermal, and most of them are idiopathic. Many of the remainder are infectious, i.e., bacterial (staphylococci), fungal (dermatophytes), viral (herpes), and spirochetal (Treponema pallidium). Sometimes, what looks like a pustule in infundibular epidermis is seen by conventional microscopy to consist of countless eosinophils and many fewer neutrophils. Examples of this phenomenon, termed badly “eosinophilic folliculitis” (the condition, actually, is an infundibulitis, not a folliculitis, and it is not eosinophilic, but rather is dominated by eosinophils), are encountered in Ofuji’s disease and in some patients with HIV. The term pustule is reserved for any nonsolid lesion that clinically is filled with “pus” and pustule histopathologically only for collections of neutrophils in
an epithelium, usually, the epidermis; we do not use the word pustule for aggregations of eosinophils within an epithelium.

**PYKNOSIS:** one of the specific signs of necrosis marked by shrinkage and intense hyperchromasia of nuclei. During cell death, some nuclei may shrink as chromatin condenses to a solid, densely basophilic mass. That combination of findings constitutes pyknosis.

**PYRIFORM:** means having the form of a pear and, when applied to proliferation epithelial, refers usually to lobules of epithelial cells that assume the shape of a pear, such as normal lobules of a sebaceous gland and abnormal ones of nevus sebaceous.

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**RACEMIFORM:** describes an appearance like a cluster of grapes or a bunch of berries formed by epithelial cells in some proliferations. In cutaneous pathology, the best example of racemiform arrangement of cells is found in trichoblastoma.

**RADIAL GROWTH PHASE:** a concept introduced by Richard Reed and co-workers who stated that the “variants of malignant melanoma that grow radially at the dermoeipidermal interface are expressions of an evolutionary process in which melanoma cells proliferate and spread along the dermal-epidermal interface or in the immediate subadjacent papillary dermis for a variable period of time before they acquire the capacity to survive and produce aggregates of tumor cells in the dermis (vertical growth phase).” Those collaborators have suggested that the vertical growth phase, defined by them as the presence of an “expansile nodule” in the papillary dermis, may indicate potential for metastasis, whereas the radial growth phase is associated with no capability for metastasis. The terms radial and vertical, however, are not contrasting; vertical is a component of radial. Vertical and horizontal are contrasting. Moreover, “radial growth phase” and “vertical growth phase” of melanoma simply are replacements for the now abandoned concept of Clark’s “levels of invasion of melanoma.” Radial growth phase superseded Clark’s levels I and II, whereas vertical growth phase substituted for levels III, IV, and V. Notions of radial growth phase and vertical growth phase of melanoma are not relevant either to diagnosis or to management of melanoma. In reality, no histopathologist is able to determine truly where the so-called radial growth phase ends and the so-called vertical growth phase begins, and no surgeon’s hand should be guided by those “phases”; every melanoma, irrespective of thickness of it, should be removed with just enough normal skin around it to ensure that that goal has been achieved. In short, assignment of phases radial and vertical is engagement in mysticism and should be eschewed.

**RAZOR BLADE REMOVALS:** in this technique a razor blade which bends (it is usually the equivalent of a “Gillette blue blade” cut in half or whole) is used to perform shave horizontal or tangential removals. The blade may also be bowed into different shapes to perform saucerization removal/excisions by a dermatologist who is experienced with it. Some find this far superior to a “knife blade” to perform saucerization. Some refer to saucerization as ”scoop removals.” (SEE BIOPSY, SHAVE REMOVALS, SEE SAUCERIZATION)

**RECURRENCE OF MELANOMA:** the following applies to all other proliferations as well varying of course if the proliferations are benign or malignant. (i.e., benign proliferations can also persist)

The issue of “recurrence of melanoma” is central to the matter of extent of margins for excisions of melanoma and to the issue of prognosis for a person who bears a melanoma. Virtually every textbook of dermatology and of general surgery advocates wider excisions for thicker melanomas based on the assertion that the likelihood for recurrence of melanoma is enhanced by narrow margins of excision, especially for thicker lesions. That declaration is without validity. Recurrence means to occur again, and in the realm of melanoma the phenomenon of recurrence may assume one of two forms, namely, persistence of a primary melanoma at the original local site, as a consequence of incomplete removal, or metastasis. The implications of those very different expressions of “recurrence” are profound, to wit, persistence of a primary melanoma may not spell a grave prognosis (i.e., re-excision may be curative, especially if the neoplasm is still very thin), whereas metastasis, for practical purposes, may signal a grave prognosis.

Unfortunately, all too many general pathologists and surgeons use the term recurrence indiscriminately, i.e., they fail to make a distinction between recurrence of a primary neoplasm at the initial local site (persistence) and recurrence that presents itself as neoplastic cells at a site some distance from the primary one (metastasis). (SEE PERSISTENCE.) The literature of pathology and surgery with regard to “margins of excision for melanoma” is peppered by the word “recurrence,” yet rarely is an attempt made by authors to qualify the type of recurrence, i.e., persistence or metastasis. Without such clarification, a reader is unable to judge the authenticity of the statement that recurrence of a melanoma is more likely if margins of the excision are narrow. If by recurrence the authors of these papers actually mean metastasis (as they usually do), then a patient’s prognosis is grim and could not have been altered, no matter how wide and deep the margins of excision because the metastasis must have occurred before the excision of the primary melanoma, assuming that the primary neoplasm was removed entirely. Parenthetically, authors of
papers about this subject often define local recurrence as the appearance of a melanoma within 2 to 5 cm of the primary site. Those melanomas however represent satellite metastases, not persistent primaries.

If, one day, dermatologists, pathologists, and surgeons are to communicate lucidly with one another about recurrence of melanoma and its implications for margins of excision of melanoma and for prognosis, then they must not employ the same word recurrence for two entirely different phenomena, i.e., persistence and metastasis. Only then can meaningful studies be undertaken of the relationship between extent of margins of excision for primary melanomas and prognosis of those neoplasms. And only then can those studies be assessed critically.

**RE-EPITHELIALIZATION:** refers to a process by which a new epidermis is generated from infundibular and eccrine ductal epithelia.

**REGRESSION:** in the realm of cutaneous neoplasia, refers to involution of a benign or a malignant proliferation, usually as a consequence of the effects of inflammatory cells on proliferations. The commonest benign proliferation that undergoes regression is solar lentigo (or its more advanced expression, reticulated seborrheic keratosis) to which a dense lichenoid infiltrate of lymphocytes is attracted. That lesion, known as a lichen planus-like keratosis, eventually disappears, leaving in its wake a papillary dermis thickened by fibroplasia, a sprinkling of lymphocytes, melanophages, and telangiectasias.

Solitary keratoacanthomas nearly always regresses after months as a consequence, in part, of a dense mixed infiltrate of inflammatory cells, leaving as residuum a dermal scar. Regression of primary melanoma is a phenomenon that involves the superficial vascular plexus, a thickened papillary dermis, and the epidermis. Requirements for induction of regression are: (1) melanocytes of melanoma in the epidermis and papillary dermis, and the epidermis. Requirements for induction of regression are: (1) melanocytes of melanoma in the epidermis and papillary dermis, and (2) lymphocytic infiltrates around the vessels of the superficial plexus, in lichenoid array within the “melanomatous” component of the papillary dermis, and scattered among neoplastic melanocytes of melanoma within the epidermis. In brief, cytotoxic products of lymphocytes kill neoplastic cells of melanoma situated in the papillary dermis and epidermis. Rarely, all of the cells of the melanoma in the epidermis are destroyed by the effects of lymphocytes, i.e., a condition termed “complete regression” of melanoma. Often, all of the melanocytes of melanoma in a discrete focus of the papillary dermis and epidermis are obliterated by the action of lymphocytes; a circumstance designated “focal regression” of melanoma. Episodically, melanocytes of melanoma are eliminated partially in the papillary dermis and in the epidermis or entirely in the papillary dermis and not at all in the epidermis, a phenomenon known as “partial regression.”

The concepts stated above vary depending on the dermatopathologist.

Morphologically, the effects of the battle between lymphocytes and melanocytes of melanoma express themselves in three fashions: fibrosis, melanosis, and a combination of fibrosis and melanosis. These descriptive terms, i.e., fibrosis and melanosis, apply to the residual changes of complete and focal regression of primary melanoma in a thickened papillary dermis beneath an epidermis whose normal undulations have been muted or effaced. Fibrosis refers to fibroplasia, usually of delicate fibrillar bundles of collagen, in a thickened papillary dermis. Sometimes the papillary dermis may be more than five times its normal thickness and measures more than 1.0mm as a consequence of formation of new collagen by fibrocytes. Fibrosis often is accompanied by diffuse deposits of mucin, sparse lymphocytic infiltrates, variable numbers of melanophages, i.e., from practically none to many, and telangiectasias. In contrast, melanosis denotes a dense band of melanophages in a papillary dermis that may be as thickened by macrophages as it may be by fibroplasia. The epidermis above a zone of melanosis also shows diminution in the normal pattern of rete ridges and dermal papillae. In some specimens, sections exhibit features of fibrosis and melanosis in the same thickened papillary dermis. In those cases, fibrosis tends to be present in the upper part of the expanded papillary dermis and melanosis in the lower part. When melanosis is noted, a diagnosis of regression of melanoma may be issued without equivocation. The findings of melanosis are specific. That is not the case for fibrosis in regression of melanoma. Changes indistinguishable from it may be found in regression of lichen planus like keratosis, a solar lentigo-reticulated seborrheic keratosis that attracts lichenoid infiltrates of lymphocytes to it and is subsequently destroyed by them. Even the numerous necrotic keratocytes so often observed in the epidermis and papillary dermis of a regression of lichen planus like keratosis may be noted in some lesions of melanoma undergoing regression by fibrosis. Regression of “halo” nevus as a result of the effects of lymphocytes on melanocytes also takes the form of fibroplasia but never of melanosis. A “halo” nevus that has regressed completely can be distinguished, usually, at scanning magnification from a melanoma that has regressed completely by fibrosis: the two have different silhouettes. In most instances, a regressed “halo” nevus has the silhouette of a Clark’s nevus, i.e., it is small, symmetrical, and slightly domed, whereas a melanoma usually is broader, asymmetrical, and flattish.

What is the significance of regression of primary melanoma? Complete regression of melanoma, in our experience is synonymous with the existence of metastases from that primary melanoma. As we conceive it, prior metastasis to a regional lymph node is mandatory for occurrence of complete regres-
sion of primary melanoma. Sensitized lymphocytes return from the involved node to the skin where they “hone in” on the melanoma and destroy it. Different authors have different interpretations concerning the significance of focal regression of primary melanoma. Some aver that it is a good prognostic sign, whereas others claim that it has no prognostic significance. We are not certain of the meaning of focal regression of primary melanoma, biologically, but we infer that if complete regression signifies a grave prognosis focal regression probably does not herald a good one.

However of this later point there is no certainty.

Last, there is the subject of regression of metastasis of melanoma to skin. That phenomenon is seen rarely, but when it is encountered, the setting tends to be a satellite metastasis in the same histopathologic section as a primary melanoma. When melanophages in discrete collections marked by jagged outlines are discerned in foci within reticular dermis and even within the subcutaneous fat, the diagnosis is melanosis as a consequence of regression of a metastasis of melanoma.

**RETICULAR ALTERATION:** (SEE BALLOONING)

**RETICULATE:** refers to one manifestation of a pattern net-like that can be visualized in the skin by both inspection gross and examination histopathologic. Examples of patterns reticulated observable clinically are livedo reticularis, confluent and reticulated papillomatosis, and parapsoriasis reticulata (parakeratosis variegata), which is a manifestation of mycosis fungoides. Patterns reticulate seen histopathologically result from interconnection of cells epithelial in conditions as diverse as trichoblastoma, mantle adenoma, and fibroepitheliomatous trichoblastic (basal cell) carcinoma.

**RETIFORM:** retiform is a synonym for reticulate.

**RIBBONS OF COLLAGEN:** describe the appearance of strips of collagen that resembles ribbons in proximity close to one another as in the situation in fibrofolliculoma/trichodiscoma, they being aligned perpendicular to struts of mantle epithelium that forms a pattern fenestrated at a stage “early” in that particular hamartoma (fibrofolliculoma) and being distributed haphazardly later in the course of the same hamartoma (trichodiscoma).

**RICHLY FIBROCYTIC STROMA:** in a proliferation of epithelial cells adnexal describes connective tissue in which fibrocytes are present in large number and in which bundles of collagen tend to be delicate and fibrillar, often being joined by mucin in quantity, as is the case in small nodular trichoblastoma, type racemiform of trichoblastoma, and fibroepitheliomatous trichoblastic (basal cell) carcinoma.

**RIMS OF BASEMENT MEMBRANE MATERIAL:** describes a layer homogenous and eosinophilic of uniform thickness composed of elements like those that constitute basement membranes normal in the skin and that is found around aggregations of cells in cylindroma and cylindrocarcinoma, as well as, at times, in spiradenoma.

**ripples PATTERN:** for purposes practical the epithelial elements essential always being immature sebocytes, refers to an image histopathologic created by epithelial cells seemingly undifferentiated arrayed in lines wavy that gives the impression of the appearance of rippling of water, it being a sign specific of sebaceous.

**ROUND MELANOCYTE:** small, round, “lymphocytoid” melanocytes are commonly present in the middle or lower portions of intradermal melanocytic nevi. They are smaller in diameter than the more superficially situated cuboidal or epithelioid melanocytes, have centrally placed compact nuclei, and usually do not contain melanin. Small round melanocytes with atypical nuclei sometimes are found in melanomas. In these cases there may be only a few nests of such cells or they may constitute the bulk of the neoplasm. Large, seemingly round cells are seen in “classic” Spitz’s nevi and some blue nevi as the result of section of spindle cells perpendicular to their long axes. Large round cells with atypical nuclei are commonly found in melanomas.

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**SARCOMA:** A neoplasm malignant made up of cells non-epithelial.

**S-100 PROTEIN:** is an acidic protein composed of a and b subunits. It was thought initially to be specific for neuro-ectodermal cells and tissues. Subsequently, the protein has been shown to be ubiquitous in distribution, including tissues derived from ectoderm (i.e., astroglial cells, melanocytes, neuroblasts, Schwann cells), mesoderm (i.e., Langerhans’ cells, adipocytes), and endoderm (i.e., neuroendocrine cells of respiratory and gastrointestinal tracts). Many neoplasms that differentiate toward those normal cells and tissues also react to this protein. S-100 protein is found in secretory cells of eccrine glands, but the presence of S-100 protein in a proliferation with adnexal differentiation does not necessarily signify eccrine differentiation. S-100 protein also is present in some proliferations with indubitable apocrine differentiation, evidenced by apocrine secretion, and in many neoplasms in the breast, probably because mammary glands and lactiferous ducts have capability for apocrine metaplasia.

Despite its lack of total specificity, antibodies directed against S100 protein continue to play a central role in establishing a diagnosis of melanoma in the skin. The routinely used, commercially available polyclonal antibodies are directed against both subunits of S100 protein and recognized protein
expressed in melanocytes, Langerhans cells, neutrophils, and nerves within the skin. In some settings, macrophages may also be detected with anti S100 protein antibodies. It is important to note that virtually all melanocytes, whether occurring singly in the epidermis, as benign nevus nests, or as melanoma cells, express S100 protein within their cytoplasm. Reported sensitivity rates have been reported for less common subtypes including mucosal, sinonasal and desmoplastic melanomas. Metastatic melanoma is also almost always detected with anti-S100 protein antibodies. Melanomas may fail to express S100 protein, though this is exceedingly uncommon.

When attempting to identify a subpopulation of cells within the epidermis, it is important to evaluate the immunostaining in concert with the routine histology in order to separate Langerhans cells from melanocytes. (The addition of anti-CD1a antibody would further help in this distinction, as Langerhans cells invariably express this antigen, while melanocytes do not). More commonly, dermatopathologists are asked to identify a population of poorly differentiated spindle-shaped cells within the dermis. In this setting, anti-S100 protein antibodies can be an invaluable aid. Virtually all desmoplastic melanomas express this protein, and virtually none of the other neoplasms in this histologic differential diagnosis do so. Thus, results from this test are very helpful in narrowing a differential diagnosis, and when used in conjunction with other antibodies, can help establish a diagnosis in most cases.

**SATELLITE LESION:** (SEE METASTASIS OF MELANOMA)

**SATELLITOSIS:** a metastasis within 5 centimeters of the primary neoplasm, which sometimes is apparent in the same tissue section; an evidence of distant metastases. The terms satellite, in-transit, regional and distant applied to metastasis are artificial and misleading and not necessary.

**SAUCERIZATION:** a variant of shave removal using a razor blade, which is bowed for deeper removals. (SEE RAZOR BLADE REMOVAL)

**SCALE:** a collection of cornified cells seen clinically as a dry, thin flake which may assume various sizes, shapes, and colors. Scale may consist of orthokeratotic cells, parakeratotic cells, or both of them jointly, it being lodged usually atop surface epidermis but sometimes being contained within the invagination formed by infundibular epidermis. Scale is described as micaceous (i.e., the confluent parakeratosis of psoriatic plaques), branny (i.e., the focal scale-crust of seborrheic dermatitis), powdery (i.e., the orthokeratosis of tinea versicolor in which the normal basket-weave pattern of the thickened cornified layer is preserved), adherent (i.e., the confluent compact and laminated orthokeratosis of ichthyosis vulgaris and X-linked ichthyosis), coarse (i.e., the focal vertical parakeratosis of Darier’s disease and of porokeratosis), and greasy (i.e., the delicate, laminated, pigmented orthokeratosis of seborrheic keratosis).

Scale must be distinguished clinically from keratosis, i.e., a horny excrescence, which is not a flake of cornified cells, but which may be orthokeratotic in a lesion of isolated epidermolytic hyperkeratosis, parakeratotic in one of acantholytic dyskeratotic acanthoma, and both together in a solar keratosis. Diagnosis histopathologic of many skin diseases can be accomplished by study of the epidermal cornified layer alone, using only low magnification of a conventional microscope. For example, mounds of parakeratosis that house neutrophils at their summit signify eruptive psoriasis or dermatophytosis, mounds of scale-crusts at lips of infundibular ostia indicate seborrheic dermatitis, alternation of short rectangular or square zones of orthokeratosis and parakeratosis in both horizontal and vertical directions telegraphs pityriasis rubra pilaris, marked compact orthokeratosis on hair bearing skin denotes lichen simplex chronicus, and a slice of orthokeratosis above a slice of parakeratosis, i.e., the “sandwich sign,” should call to mind dermatophytosis in which hyphae repose in orthokeratotic cells very near the middle of the sandwich. Corneocytes in excessive number may cause infundibula to widen, as in keratosis pilaris and discoid lupus erythematosus, and even to become sac-like, as in an infundibular cyst.

**SCALE-CRUST:** a combination of scale (cornified cells, usually parakeratotic ones) and crust (serum that contains blood cells, either, red, white, or both). Scale-crusts cover spongiosiform dermatitides, allergic contact dermatitis, and nummular dermatitis in particular, but they are found also at the lips of ostia of infundibula in seborrheic dermatitis. Scale usually results from acceleration in epidermopoiesis that is associated with an increase in the number of spinous cells. Crust usually is a consequence of spongiosis or of erosion secondary to excoriation, following which serum flows to the surface of the skin.

**SCANNING:** in histopathology pertains to observing an entire section quickly, from one end to another, using an objective with the lowest magnification, i.e., between X 1.0 and X 2.5 objective of a conventional microscope. Scanning magnification is crucial to accurate diagnosis in dermatopathology because it permits analysis of patterns formed by cells, i.e., infiltrates of inflammatory cells around vascular plexuses, in the interstitium, and within epithelial structures, and infiltrates of neoplastic cells as they have become arranged in distinctive silhouettes. Architectural features visualized with scanning magnification are requisite if an algorithmic method based on pattern analysis for specific diagnoses of skin diseases is to be used effectively for inflammatory diseases and for proliferations of various kinds with
regard to benign versus malignant proliferations the silhouette reflects the biologic behavior. There are exceptions.

**SCAR:** a type of fibrosis that represents the end stage of an inflammatory process or the end result of a wound healing, that early in its course resulted in destruction of preexisting tissue, evolved through granulation tissue, and eventuated in fibroplasia. Formation of granulation tissue, the scaffold on which fibroplasia usually proceeds, is a sign that healing of the wound has commenced. Histopathologically, a scar rather early in its course is made up of altered bundles of collagen in conjunction with a marked increase in the number of fibrocytes, both of those elements being oriented mostly parallel to the skin surface, and in association with what seems to be an increase in the number of dilated venules aligned mostly perpendicular to that surface and in the amount of mucin. Years later, the number of fibrocytes is so markedly decreased that sometimes they are identifiable with difficulty. In contrast, a keloid is a type of fibrosis that consists of strikingly thickened, brightly eosinophilic, homogeneous-appearing collagen bundles disposed randomly and affiliated with an increased number of plump fibrocytes that parallel the long axis of the thickened bundles. A dermatofibroma is another type of fibrosis in which coarse collagen bundles and many fibrocytes are arranged haphazardly, sometimes being joined by siderophages and lipophages that signify extravasation in the dermis previously of erythrocytes secondary to trauma at that particular site. Other findings encountered commonly in a dermatofibroma are fibrocytes and/or histiocytes interspersed between distinctly thickened bundles of collagen at the periphery of the lesion, acanthosis, and hyperpigmentation of the epidermis. Normal skin markings tend to be effaced when those three forms of fibrosing inflammation, i.e., scars, keloids, and dermatofibromas, encroach severely on the papillary dermis and impinge on the epidermis.

**SCLEROSIS:** clinically, a condition of hardness of the skin and, histopathologically, a type of fibrosis characterized by near obliteration of boundaries between bundles of collagen, the result being the appearance of the bundles having become blended (“homogenization”) and by marked decrease in the number of fibrocytes. Sclerosis usually indicates that fibroplasia has been longstanding. Examples of sclerosis are the thickened papillary dermis of lichen sclerosus et atrophicus and much, or all, of the dermis of chronic radiation dermatitis. Morphea, other than the form of it confined to the upper part of the dermis and known as lichen sclerosus et atrophicus, is not typified by sclerosis; collagen bundles in a lesion of morphea formed fully are crowded but, nonetheless, discrete. At that stage, fibrocytes in morphea are decreased in number.

“**SEBACEOUS FOLLICLE:**” refers to a vellus-hair follicle on a face characterized by a prominent epidermal infundibulum, a puny inferior segment, and one or more large sebaceous glands.

**SEBACEOUS SECRETION:** is holocrine secretion of over-mature sebaceous cells. It represents the end product of maturation of sebocytes.

**SEBOCYTE:** is a synonym for sebaceous cell, a cell derived in embryonic life from a follicular germ. Immature sebocytes have round nuclei and scant, slightly vacuolated cytoplasm; mature sebocytes have scoloped nuclei and markedly vacuolated cytoplasm. Sebocytes in an embryo originate in the middle bulge at the junction of the infundibulum and isthmus. Sebocytes are aggregated in lobules that, in conglomerate, form a gland; a duct collects sebaceous (holocrine) secretion and transports it to an infundibular epidermis, from whence it is carried, as sebum, through an ostium to the surface of the skin.

**SEBORRHEIC:** purportedly relating to sebaceous glands or sebum. In actuality, however, conditions such as seborrheic dermatitis and seborrheic keratoses bear no direct relationship to sebaceous glands or to sebum. Seborrheic distribution of lesions refers to sites like those involved by seborrheic dermatitis, to wit, the forehead, malar region, paranasal and nasolabial folds, retroauricular region, and sternal region.

**SEBUM:** is constituted mostly of sebaceous secretion and other materials that are carried with it in its journey through a sebaceous duct and epidermal infundibulum to the skin surface, namely, squames, *Pityrosporum ovale* and *orbiculare*, *Staphylococcus epidermidis*, *Propionibacterium acnes*, and *Demodex*.

**SECRETION:** is a process whereby a specific product is elaborated as a result of the activity of a gland. Secretion also may refer to any substance that is produced by the act of secreting. The major kinds of secretion are those that are discharged on an internal or external surface of the body (i.e., by way of an exocrine gland) and those that secrete hormones that are dispatched into blood and lymph (i.e., by way of an endocrine gland). The glands in skin produce secretions of different types, i.e., merocrine by eccrine glands, apocrine by apocrine glands, and holocrine by sebaceous glands. Merocrine, apocrine, and holocrine secretions are all exocrine.

**SECRETORY:** pertains to a process whereby a specific product is elaborated by cells of a gland. Secretion and excretion are two processes by which cells extrude their products. If the extruded material is to be used by the organism, the process is called secretion; if the extruded material is waste, the process is called excretion. All three glands in skin, namely, eccrine, apocrine, and sebaceous, are secretory.
SEGMENT: refers to two fundamentally different parts of a hair follicle, namely, the upper and the lower. The upper segment consists of isthmus. It does not participate in the ever-repeating cyclic changes of a follicle. The lower segment is made up of two parts, namely, the stem above and the bulb below. The lower segment undergoes characteristic morphologic and functional changes during a follicular cycle that are designated growing (anagen), involuting (catagen), and resting (telogen).

SEMANTICS: The meaning of a word, phrase, or text

SEPTA: refers usually to struts of connective tissue that divide a normal or pathologic structure into compartments, for example, fibrous septa that partition fat lobules of normal subcutaneous tissue, granulomatous and fibrotic septa that form fenestrations in subcutaneous fat of erythema nodosum, and fibrous septa that create units composed of clusters of proliferative apocrine cells and mucin in mucinous carcinoma.

SEPTAL PANNICULITIS: an inflammatory process in the panniculus adiposus in which, as viewed at scanning magnification, the infiltrate of inflammatory cells is situated mostly in the septa rather than in the lobules. In many instances the overlap with lobular may make distinction difficult.

SERUM: is the fluid portion of the blood obtained after removal of the fibrin clot and blood cells, distinguished from plasma in circulating blood.

SESSILE: means attached by a base broad, in contrast to a polyp which is attached by a peduncle.

SHADOW CELLS: are ones cornified in which, as a consequence of karyolysis, nuclei have faded but an outline vague of them still can be recognized. These cells represent attempts faulty at differentiation toward hair and are found only in conditions in which some follicular matrical cells are present, examples of that being panfolliculoma, pilomatrixoma, matricoma, matrical carcinoma, desmoplastic trichoepithelioma, and mixed tumors with apocrine, follicular and sometimes sebaceous differentiation. Rarely, “shadow cells” may be present in trichoblastomas and trichoblastic (basal cell) carcinomas, both of those proliferations consisting mostly of germinative cells. “Shadow cells” tend to become calcified, a phenomenon that is visualized at first as sprinkling of delicate particles basophilic within corneocytes arranged compactly and later as occlusion complete of corneocytes by material homogeneous and purple. In pilomatrixoma, “shadow cells” often act as a nidus for osseous metaplasia by fibrocytes.

SHEAVE (TANGENTIAL, HORIZONTAL) BIOPSY TECHNIQUE: describes a procedure performed with a surgical blade or razor blade directed mostly parallel to the skin surface for the purpose of sampling exophytic lesions such as verrucae, seborrhic keratoses, fibroepithelial polyps, melanocytic nevi, and cherry hemangiomas. Some flat lesions of melanoma-in-situ in sites like the tip of the nose and in Bowen’s disease and basal cell carcinomas are sometimes amenable to the shave technique. Unfortunately, the procedure is abused when it is used to biopsy inflammatory diseases of all kinds, proliferations that are mostly endophytic, and elevated pigmented lesions suspected of being melanoma. These all-too-common practices are to be deplored because they often harvest inadequate specimens that prevent histopathologists from using criteria effective for diagnosis, criteria that have been formulated on the basis of sections cut from satisfactory biopsy specimens taken by scalpel excision. This technique using a razor blade can be modified to perform saucerization which can provide satisfactory “biopsies” of pigmented and other lesions if used properly and selectively. (SEE RAZOR BLADE REMOVAL, SEE SAUCERIZATION)

SHEATH: denotes a structure that encloses or surrounds a body. In skin, most sheaths are composed of fibrous tissue, the most notable being the perifollicular connective tissue sheath that extends along the entire outer sheath to the base of the infundibular epidermis. The follicular sheath is separated by a basement membrane from the epithelial component of a follicle. Nerve fascicles in skin also are associated with three connective tissue sheaths, the epineurium, perineurium, and endoneurium.

SHELL OUT: describes a method whereby certain benign proliferations and cysts are eased from their housings with a scalpel or other instrument and transported to the skin surface along a path formed by an artificial cleft that developed between compressed encompassing fibrous tissue and normal skin or subcutaneous fat.

SIDEROPHAGE: a macrophage that has ingested iron.

SIGNET-RING CELL: describes a mucin producing adenocarcinomatous cell whose cytoplasm is filled completely with mucin and is distended by it. Mucin causes the nucleus to be compressed and displaced to the side of a cell, thereby causing the entire cell to resemble a signet ring. The signet part is the nucleus at the side of the cell, the ring portion is the cytoplasm at the periphery of the cell, and the space reserved to accommodate a finger is the mucin. Other proliferations have been said to have cells that simulate this appearance (i.e. signet ring cells in some melanomas).

SILHOUETTE: is a representation of the outline of something, usually filled in with black or another solid color. In the mind’s eye of a histopathologist, the contours of a proliferation or a cyst can be filled in with solid colors and made into silhouettes. Observation of a proliferation at scanning
magnification of a conventional microscope enables histopathologists to imagine, among other things, its silhouette. The silhouette of a proliferation is the morphologic representation of the biological behavior of that proliferation. For example, a silhouette that is symmetric, vertically oriented, wedge-shaped, and sharply circumscribed with smooth borders indicates that a proliferation, if primary in skin, is likely to be benign. A silhouette that is asymmetric, poorly circumscribed, and jagged in outline nearly always signifies that a proliferation, if primary in skin, is malignant. All too often, textbooks of general pathology emphasize cytologic details almost exclusively as criteria for differentiating benign from malignant proliferation, whereas, in actuality, features of silhouette are more compelling in regard to that distinction because they are more consistent morphologic reflections of biological behavior. In short, accurate diagnosis of cutaneous proliferation requires assessment of silhouette at scanning magnification and of cytologic features at higher magnifications. The entire range of magnifications, from scanning to high, is complementary in enabling a histopathologist to come to a specific, accurate diagnosis.

**SINUS:** an epithelium-lined channel in the skin that opens on the surface, usually through an infundibular ostium. In contrast to a fistula that is open on both ends, neither of which needs to be in continuity with an infundibulum, a sinus is open at one end only. Examples of a cutaneous sinus are seen in lesions of the so-called follicular occlusion triad, all of which begin as explosive suppurative infundibulitides/folliculitides, namely, acne conglobata, hidradenitis suppurativa, and dissecting cellulitis of the scalp (to which should be added a fourth analogue, i.e., acne keloidalis). A sinus forms as a result of re-epithelialization of an infundibulum as it attempts to restore itself to its original state after having burst and ejected its contents, mostly of neutrophils, into the dermis, where those polymorphs are joined, in time, by histiocytes in company with lymphocytes and plasma cells, and, eventually, by fibrocytes. The failed attempt of an infundibulum to reconstitute itself in that setting is manifested as a sinus which invariably, is accompanied by fibrosis.

**SINUS TRACT:** (SEE SINUS)

**SOLAR ELASTOSIS:** elastotic material, i.e., the altered, bluish, spaghetti-like connective tissue produced by fibrocytes that have been affected by sunlight over the course of decades. This is a sign of longstanding damage to the skin by the effects of ultraviolet light.

**SOLID:** designates a substance or tissue that is not fluid, gaseous, or hollow. The term is applied in dermatopathology to proliferations that are neither tubular nor cystic. For example, poromas and cylindromas are mostly solid. Apocrine hidradenomas are solid and cystic. Apocrine papillary cystoadenomas are mostly cystic. Solid carcinoma designates a distinctive histopathologic variant of apocrine carcinoma.

**SPECIMEN:** designates a sample of tissue taken by a surgical procedure in preparation for examination by conventional microscopy in order to determine particular characteristics of it from which inferences (diagnoses) may be drawn. A distinction exists: between a specimen of skin removed by a surgical instrument from a person and sections of skin that represent slivers cut by a microtome from a specimen.

**SPINDLE-SHAPED CELL:** having the shape of a spindle, i.e., being like a stick with ends tapered used to form and twist yarn in spinning by hand. True spindle-shaped melanocytes are thin and are met with more often in melanoma than in the type of nevus paid heed most by Spitz in 1948 and in which large cells constituent tend to be fusiform, often in conjunction with ones round, plump oval, polygonal, and plasmacytoid; never do spindle-shaped melanocytes arrayed in fascicles monopolize in “classic” Spitz’s nevus, although they do predominate in what Spitz in 1948 referred as a “spindle cell tumor.” Last, it must be stressed that spindle-shaped melanocytes do not qualify as “Spitz’s cells,” the sine qua non for which is cytoplasm copious; a true “spindle cell” of any kind, including one melanocytic, has cytoplasm paltry. In short, although spindle-shaped melanocytes may appear among “Spitz’s cells” in a “classic” Spitz’s nevi, they are not, in themselves “Spitz’s cells.” Thus “true” spindle shaped cells contrast with fusiform shaped cells, whether referring to the above or those found in other melanocytic nevi, including “blue nevi.”

**SOLID CYSTIC:** describes the two elements that constitute various proliferations, benign and malignant, one of them being made up of cells arranged compactly (solid) and of the other cells that line a sac containing fluid or material (cystic), the example quintessential of the phenomenon being one presentation histopathologic of apocrine (solid cystic) hidradenoma.

**SPIR-:** is a prefix used to convey a sense for relation to a structure that spirals. In dermatopathology, spir- traditionally has referred to the spiral of the component intraepidermal of an eccrine duct, i.e., to the acrosyringium, and perforce to proliferations thought to be eccrine in character. The terms eccrine spiradenoma and eccrine acrospiroma, however, are misnomers because both spiradenoma and some proliferations included under the rubric of acrospiroma (i.e., pale or clear-cell hidradenoma) show apocrine, rather than eccrine, differentiation. Because the segment distal of an apocrine duct spirals through infundibular epidermis, the concept and the word acrosyringium is applicable equally to the apocrine, as well as the eccrine duct.
SPITZOID: in general, any lesion that resembles histopathologically a “classic” Spitz’s nevus. The term spitzoid melanoma has been applied to a melanoma that has some attributes histopathologic of that nevus.

SPITZ’S CELLS: melanocytes of the size and shape of those pictured in most photomicrographs by Spitz in her article seminal of 1948, they being ones of “classic” Spitz’s nevus and having a large nucleus, abundant cytoplasm, and round, fusiform, and polygonal shapes especially.

SPONGIFORM PUSTULE: a collection of neutrophils in the spinous and granular zones of the epidermis. Remnants of cell membranes give the pustule a sponge-like appearance.

SPONGIOSIS (Intercellular edema): of the epidermis and of epithelial structures of adnexa, expressed morphologically by widened spaces between spinous cells and by intercellular bridges that stretch across those extended spaces in company with a sprinkling of cells, usually inflammatory ones foreign normally to the epidermis. Although spongiosis nearly always is mediated by inflammatory cells, particularly by lymphocytes but sometimes by eosinophils and even by neutrophils, it may be induced by neoplastic lymphocytes, such as those of mycosis fungoides. In short, spongiosis is a distinctive pattern of cutaneous epithelium brought into being usually by inflammatory cells and unusually by neoplastic ones. Some spongiosic inflammatory processes may eventuate in vesicles clinically, such as allergic contact dermatitis, nummular dermatitis, dyshidrotic dermatitis, and “id” reactions, whereas others, such as seborrheic dermatitis, erythema annulare centrifugum, and pityriasis alba, do not progress to vesicles. Practically never does spongiosis in mycosis fungoides culminate in a vesicle.

SPONTANEOUS: means self-generated, which is incompatible with biologic processes. Therefore, all so-called spontaneous phenomena like spontaneous keloids and spontaneous regression of melanoma are not spontaneous at all. The former results from external trauma and the latter from the effects of lymphocytes on the melanocytes.

SQUAME: designates a lone corneocyte, particularly one that has become detached from its neighbors, as happens, commonly following rupture of an infundibular type of follicular cyst. In that circumstance, squames often are found free in the dermis or subcutaneous fat, or within multinucleate histiocytic giant cells. The term “squame” does not apply to physiologic desquamation of clumps of epidermal corneocytes.

SQUAMOID: means resembling cells of the spinous zone of surface or the infundibular epidermis, in contrast to basaloid cells that look like basal cells of that epithelium. Proliferations of basaloid and squamoid cells are found together in some proliferations common, i.e., verruca vulgaris and seborrheic keratoses.

SQUAMOID CELLS: (SEE SQUAMOID)

SQUAMOUS EDDIES: are whorls made up of spinous cells mostly, but also of granular and cornified cells, that presumably come into being as a consequence of the spiral of sebaceous ducts through infundibular epidermis. The circumstance most common for development of “squamous eddies” is verruca vulgaris, and the next most common is seborrheic keratosis. Most of the time, “squamous eddies” are a sign of a benign condition and also of irritation of the lesion (i.e. seborrheic keratosis.)

STAGE: usually refers to the extent of spread of a malignant proliferation. Clinical stages for melanoma have been defined by the American Joint Committee on Cancer and the New York University Melanoma Cooperative Group. The value of these stagings is debatable by some.

STELLATE: shaped like a star, arranged in a roset or rosetes.

STEM: describes the upper part of the lower segment of a follicle that resembles the stem of a flower, a slender stalk that derives from a bulb and that supports a “flower” (upper segment of isthmus and infundibular epithelium). The course of a stem is from Adamson’s fringe below to desquamation of corneocytes of the inner sheath above (from the top of the bulb to the base of the isthmus). In terminal follicles in anagen, the stem is the longest part.

STORIFORM: refers to a pattern created by the interweaving of fascicles of oval and spindle cells that causes it to resemble the intersecting intertwining pattern of a doormat.

STRAND: refers to epithelial and nonepithelial cells arranged in single file, in contrast to a cord, in which cells are arranged in rows of two, and columns, in which cells are arranged in formations more than two cells wide. Strands may be seen in benign conditions, such as melanocytes splayed in a single file between bundles of collagen of the reticular dermis in a superficial type of congenital nevus, and in malignant ones, such as metastatic carcinoma to the skin from a breast.

STRETCHED STEM: describes the upper part of the lower segment of a follicle that resembles the stem of a flower, a slender stalk that derives from a bulb and that supports a “flower” (upper segment of isthmus and infundibular epithelium). The course of a stem is from Adamson’s fringe below to desquamation of corneocytes of the inner sheath above (from the top of the bulb to the base of the isthmus). In terminal follicles in anagen, the stem is the longest part.

STRATIFIED SQUAMOUS EPITHELIUM: consists of cells of different shapes, i.e., columnar, cuboidal, and polygonal, that flattens progressively as they come nearer to the free surface. Stratified squamous epithelium, such as the epidermis, may cornify completely and be free of nuclei in its outermost cells or, as in mucous membranes, may cornify partially and retain nuclei in its outermost cells.

STROMA: designates the connective tissue component of a proliferation of epithelial cells. It may be edematous, mucinous, or fibrous or it may contain globules of amyloid. Some
proliferations may be associated with stroma copious, i.e., fibrofolliculoma/trichodiscoma, trichoblastoma, and the fibroepitheliomatosus type of trichoblastic (basal cell) carcinoma, whereas other proliferations are accompanied by little or no stroma, i.e., cylindroma, sebaceoma, and infundibulocystic trichoblastic (basal cell) carcinoma. Understanding of stroma can be very helpful in determining whether a lesion is benign or malignant.

**STROMA LIKE THAT OF PERIFOLLCULAR SHEATH IN AN EMBRYO:** refers to highly fibrocytic, richly vascular connective tissue made up chiefly of delicate fibrillary bundles of collagen in combination with mucin plentiful, the constellation of findings being reminiscent of that seen around a follicle developing in an embryo.

**SUBCORNEAL PUSTULE:** a collection of neutrophils situated immediately beneath the cornified layer of the epidermis.

**SUBCUTANEOUS:** denotes being situated beneath the dermis and therefore beneath the skin. The skin itself consists only of epidermis and dermis. The subcutaneous fat was known in times past as the hypodermis because it lies immediately beneath the skin. A distinction exists between subcutaneous fat and subcutaneous tissue. Subcutaneous fat consists predominantly of adipocytes. At the base of the subcutaneous fat is fascia, or aponeurosis, and beneath it sits skeletal muscle. Lesions that arise in or are situated in the dermis and subcutaneous fat are considered to be housed in superficial soft tissues, whereas those found below the fascia are regarded as being within deep soft tissues. Dermatopathology overlaps with “soft tissue” pathology; although most regard it as a separate field of pathology.

**SUPERFICIAL ATYPICAL MELANOCYTIC PROLIFERATION OF UNCERTAIN SIGNIFICANCE (SAMPUS):** a phrase employed for diagnosis by David Elder, his associates, and followers of them to designate a “category” that “includes predominantly junctional melanocytic proliferations and melanocytic proliferations that are confined to the epidermis and papillary dermis, “without evidence of tumorigenic proliferation or mitotic activity there.” Because the term is generic, rather than specific, it is not a diagnosis precise but a description rondelette and, at the same time, an acknowledgment of doubt about behavior biologic, adding nothing to what should be an effort with determination to arrive at a diagnosis with precision based on an understanding profound of melanocytic proliferations. Now is the time to jettison that phrase fuzzy before it becomes yet another example of gobbledygook in the language of general pathology and dermatopathology.

**SUPERFICIAL VASCULAR PLEXUS:** refers to venules and arterioles situated beneath the papillary dermis in the upper part of the reticular dermis. Capillaries from the superficial plexus loop into the dermal papillae.

**SUPRAMATRICAL ZONE:** designates the zone in a follicular bulb situated between the matrix below and the keratogenous zone above. It is bounded by the critical line at its base and by the B-fringe at its surface. Multiplication of cells that originated in the matrix has almost stopped by the time the supramatrical zone has been reached, in preparation for formation of hair. (SEE KERATOGENOUS ZONE, SEE B FRINGE)

**SUPRAPAPILLARY PLATE:** The portion of the epidermis situated immediately above the summit of dermal papillae.

**SUPPURATION:** Formation and exudation of pus, i.e., a creamy viscous fluid that consists of a daunting collection of neutrophils mostly, but also debris, both cellular and non-cellular, that results from necrosis and degeneration consequent to the effects of destructive enzymes released from polymorphonuclear leukocytes. Accumulation of pus in a cavity of a cavernous organ is called empyema. Suppuration in skin may manifest itself as tiny pustules (i.e., Munro’s micro abscesses and Kogoj’s spongiform pustules) and as large abscesses (i.e., secondary to rupture of an infundibular cyst and in infectious processes, such as those caused by atypical mycobacteria and deep fungi). Within cutaneous epithelium, foci of suppuration may appear in both surface and infundibular epidermis, and in eccrine glands and ducts. Abscesses also may develop in the midst of the dermis or in the subcutaneous fat.

**SYRINGO-** designates a tube such as is characteristic of some structures constituent of syringoma and of syringomatous carcinoma, but also of many other proliferations of character eccrine, apocrine, and sebaceous.

**SYRINGOTROPISM:** a biological phenomenon that indicates growth or turning movement of a cell or a collection of cells toward an eccrine gland. In a strictly morphologic sense it is not definable. Adj. syringotropic.

The following terms would be better; intrasyringeal: placed within an eccrine gland; perisyringeal: present around an eccrine gland.

Syringotropism is not defined in medical dictionaries, or in textbooks of dermatopathology. However, it is constantly used in articles, especially in those about histopathology of mycosis fungoides. Strictly speaking, the suffix *tropism* implies movement, the best example being the turning or bending phenomenon that plants undergo in response to light as the environmental stimulus, called phototropism. Literally, syringotropism means a “turning toward the eccrine gland” or having an affinity for the eccrine gland respectively. A review of this word in the literature reveals a constant association with one condition only: mycosis fungoides. The
words syringotropism and syringotropic are employed in the cases of mycosis fungoides in which neoplastic lymphocytes are found in the epithelia of eccrine glands. In fact, this criterion has led to two subtypes of mycosis fungoides, namely, the so-called syringotropic and folliculotropic. (SEE FOLLICULOTROPISM)

There are only occasional exceptions in the usage of these words in reference to entities other than mycosis fungoides, but interestingly nearly all are malignant: melanoma, Bowenoid carcinoma, skin metastases of other carcinomas, etc. Occasionally, syringotropic is used for an inflammatory disease.

What dermatopathologists intend to describe by the word syringotropic is the biopsies in which lymphocytes are placed mostly in the eccrine units, i.e., those specimens in which cells have a clear affinity for these adnexa. But this is not exclusive of mycosis fungoides. Many other diseases, much more common and benign, display cells with tendency for adnexa. Examples are neutrophilic hidradenitis or lichen striatus to mention but some of them. The reason why none of these are described by words that finish with tropism or tropic is that dermatopathologists use these words only for malignant conditions. Confronted with exactly the same morphologic finding, namely, lymphocytes in eccrine glands, syringotropism is used only after having decided already on a malignant condition, usually mycosis fungoides. When the diagnosis of a benign condition, i.e., lupus erythematosus or neutrophilic hidradenitis, is made the cell infiltrates are not termed tropic.

In addition, the term seems only to apply to lymphocytes, not eosinophils or neutrophils.

SYSTEMATIC: concerning a system or organized according to a system.

SYSTEMIC: pertinent to a whole body rather than to one of its parts; somatic.

SYSTEMIZATION: the process of organizing something according to plan.

SYSTEMATIZED: arranged according to an organized system.