



Dermatopathology: an abridged compendium of words. A discussion of them and opinions about them. Introduction and Part 1

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Introduction

A Bernard Ackerman always had a “dream” that a dictionary would be created for dermatopathology (and dermatology) so that these fields could better communicate with one another, in one “language.” He felt that until a real dictionary came into being neither would be authentic branches of knowledge. He also felt that this dictionary would serve pathology as well. Many tried to complete this “project” (in Philadelphia, New York, and elsewhere) but none succeeded. Bernie considered Morris Leider’s 1976 dermatology dictionary a very limited one for many reasons. Bernie’s own efforts with Almut Böer, M.D. in the journal, *Dermatopathology: Practical & Conceptual* used an “extended discussion of each word.” They managed to get through the “A’s” (over a two year period) and attempted to show others how a dictionary should be completed. They encouraged others (using their model) to continue this work.

This compendium of words is not meant to be “original” nor is, it a “dictionary” as defined. The Oxford Dictionary, second edition revised, says “dictionary: a reference book containing an alphabetical list of words with information given for each word, usually including meaning, pronunciation, and etymology.” This work is essentially based on Bernie’s opinions about words. My task has been to “pluck” the words, ideas, and concepts out, organize them, rewrite some and add some of my own. Discussions of words have been taken from:

- All his glossaries from 1978-2010—the glossaries have been the most useful

- All his books from 1978-2010 (the material “outside” the glossaries)
- The journal, *Dermatopathology Practical & Conceptual* (with Almut Böer, M.D. and other authors) and Bernie’s material from other places, (i.e., Indianapolis meetings, Palm Springs meetings, AAD meetings, ISDP, ASDP, as well as the material from the Institute of Dermatopathology at Jefferson in Philadelphia and the Ackerman Academy in New York, etc.) The Journal has been extremely useful. Its use accounts for why there are so many words in the “A’s” compared to other letters since this was the only letter for which definitions were done. In summary, the glossaries and “The Journal” were the most useful.

In addition the reader should keep in mind the following: Whenever I say Bernie’s works I am including his coauthors, if any. A portion of the material is copied verbatim from Bernie’s material. I fully acknowledge that. (However, this is a tribute to Bernie.) Its essence and beyond is “Ackermanian.” Much of the material has been paraphrased from Bernie’s works. A small amount of material comes from “others” and has been paraphrased or rewritten. (i.e., epidermotropism, neurotropism). Some of Bernie’s “words” have been rewritten by me in order to update the material as old ideas were discarded and new ones came to fruition (i.e., the infundibulum is epidermal). One change in a concept usually leads to many changes in the meanings of a word or many words. I hope my updates of Bernie’s “definitions” which I decided to do have stayed true to Bernie’s ideas and opinions (mine are very similar). Some of the discussions of words are long and

rambling and repetitious (i.e., in-situ). The reason for this is that in many cases (especially in the “A’s”) they follow Bernie and Almut’s method and definitions. Also, some discussions are pieced together from multiple sources. I hope this piecing together of a discussion of a word from various Ackerman sources has not lead to confusion.

The Oxford, Webster, and other well known dictionaries are accepted as “definers of the English language.” They do not differ very much. However, among the different “schools of dermatopathology” there are large differences in the usage, understanding, and the meaning of words (much less so for dermatology.) I feel this is driven by differences in concepts, criteria, plus intangibles that really make one “dictionary” which is accepted by all an impossibility!

Additionally on this subject there is something even more fascinating to me. Each school feels that what they say (or think) regarding certain subjects (i.e., melanocytic neoplasia) makes perfect sense (to them) and the other “school” is “wrong.” Each feels just as strongly about their own concepts, criteria, etc. and cannot “believe” the other school. (In many instances, one school may only marginally read what another school writes.) Many are in between, some ideas from one group and also another. (I feel that before you can disagree with another’s concepts you must first be “clear” about your own and be able to state why “something is what it is.”) It is challenging to translate one language into another and thus know what the other side is “talking about” (unless the writing is unintelligible.)

From this imperfect work I hope you will “take what you want and leave the rest.”

The compendium (Part 1)

– A –

ABNORMAL MELANOCYTE: any melanocyte, particularly one of a melanocytic nevus or of a melanoma, that differs cytopathologically from that of a melanocyte situated at the dermoepidermal junction of normal skin. By definition, a melanocyte positioned at that junction is normal and a melanocyte of a nevus or of a melanoma is abnormal. Nuclei of melanocytes at the junction are small, round or oval, monomorphic, and monochromatic, and do not exhibit a nucleolus. As a rule, nuclei of abnormal melanocytes of a nevus and of a melanoma are larger than those of normal melanocytes, sometimes, as in the case of some “classic” Spitz’s nevi and of some melanomas, being much larger. In nevi other than “classic” Spitz’s nevi, nuclei of abnormal melanocytes tend to be monomorphic and monochromatic; in “classic” Spitz’s nevi, they may be pleomorphic and, at times, heterochromatic. In melanomas, nuclei of abnormal melanocytes often are pleomorphic and heterochromatic. In both “classic” Spitz’s nevi

and melanomas, nuclei may sport a prominent nucleolus, but only in some melanomas is that nucleolus dead center in a vesicular nucleus of virtually every abnormal melanocyte constituent. In nevi other than “classic” Spitz’s nevi, nuclei of abnormal melanocytes organized in aggregations are equidistant from one another; in “classic” Spitz’s nevi and in melanomas; they often are not equidistant, that being a consequence of variability of the size and shape of nuclei, but also of the amount varied of cytoplasm.

ABNORMAL MITOTIC FIGURE: a mitotic figure that is aberrant, such as being tripolar or tetrapolar, or of the configuration of a ring, in contrast with a normal appearance bipolar. Abnormal mitotic figures are seen episodically in melanomas and, rarely, but surely, in “classic” Spitz’s nevi.

ABORTIVE: premature, prematurely, cut short.

Abortive has been defined in a variety of ways despite the fact that the meaning of it originally was “born prematurely.” In medicine in general, abortive refers to arrest in development, meaning that, biologically, a structure does not mature completely. In regard to a particular disease, the term also has the meaning of being “incomplete” clinically and histopathologically. Concerning matters morphologic, however, the word abortive, as it is applied in the language of dermatology and dermatopathology, has yet to be defined in a cogent manner.

In every instance in which the word “abortive” is employed for findings morphologic, the word is used wrongly. Small plaque parapsoriasis is a manifestation of mycosis fungoides (not an abortive lymphoma), and psoriasis that spreads centrifugally is very much psoriasis (not abortive pustular psoriasis). Immature structures in sections of tissue should be designated immature, not abortive. An adnexal anlage is an immature adnexal structure, the word anlage being German for an aggregation of cells in an embryo that represents the first evidence of a future structure. What is meant by an abortive anlage is just as opaque as what is intended by abortive melanosomes; melanosomes may be less than fully melanized, but that does not make them abortive.

ABSCCESS: clinically a circumscribed collection of pus and histopathologically a localized collection of neutrophils.

An abscess may come into being without any cause infectious, and in the skin most abscesses are not a result of infection. The most common abscesses recognizable clinically results from rupture of an infundibular cyst, and the most common circumstance in which that occurs is “cystic” acne vulgaris. Abscesses of noninfectious nature are encountered histopathologically in eruptive lesions of psoriasis, “pustular” psoriasis being a caricature of the process, they having been given designations like Munro’s microabscess and spongiform pustule of Kogoj. Strictly speaking, an abscess

consists of neutrophils and both parts and products of them; “necrotic tissue” and “fluid products of tissue breakdown” are irrelevant to the composition of an abscess. Although signs of inflammation, i.e., redness, may be observable around some abscesses, that surely is not always the case; for example, pustules widespread of von Zumbusch psoriasis sometimes develop in skin devoid of redness. At other times, however, these pustules are situated on erythematous skin. The pus of an abscess need not be flowing and may never flow. In contrast, suppuration refers to the formation or discharge of pus. An exudate is fluid that has exuded from tissues and it need not be pus (purulence); it may be serous or hemorrhagic.

A furuncle is an abscess situated around the base of a hair follicle, the process having begun as a suppurative infundibulitis that extended along the follicle to the base of it. The same may be said for hidradenitis suppurativa and its analogues, namely, acne conglobata, acne keloidalis, and dissecting cellulitis of the scalp (perifolliculitis abscedens and suffodiens). Pathogenetically, hidradenitis suppurativa has nothing whatever to do with eccrine or apocrine glands. Although the term “abscess cavity” is employed so often it has become cliché, it is a contradiction in terms because a cavity is an empty space. The same is true for “chronic abscess”; it is a non-sequitur because an abscess, consisting as it does of neutrophils, always is acute in terms of sequence chronologic of the inflammatory process, it being followed by granulomatous and fibrosing inflammation, the latter truly being “chronic.” Last, Pautrier’s microabscess is wrong on both counts: the phenomenon was described by Darier, not Pautrier, and the cells that make up the collection are lymphocytes, not neutrophils. A cluster of lymphocytes does not qualify as an abscess.

ACANTHO-: a prefix meaning spinous or prickle and in dermatology/ dermatopathology applied to spinous (prickle) cells of an epithelium

ACANTHOLYSIS: a process whereby epithelial cells, as a consequence of loss of connections (desmosomes) between them, separate from one another, becoming round and eventually dying, the process ending in formation of either a cleft or a blister.

Acantholysis refers to a process and, that being the case cannot properly be said to be a “disruption” or “dissolution.” Moreover, cells of the spinous, granular, and cornified layers may become acantholytic, for example, in focal acantholytic dyskeratosis as it presents itself in Darier’s disease, Grover’s disease, a particular kind of epidermal nevus, and a distinctive benign keratosis (acantholytic dyskeratotic acanthoma). Not only cells of the epidermis (both surface and infundibular) are affected, but those of cutaneous ducts and even glands, namely, the sebaceous, apocrine, and eccrine. When epithelial

cells separate completely from one another, they are doomed to become necrotic, which, in classic pathology is a particular type of degenerative phenomenon typified by pyknosis, karyorrhexis, and karyolysis, not by hyalin, which is a generic, antiquated designation for any substance, ranging from fibrin to amyloid, that resembles glass, and, because of its utter non-specificity, should be eschewed because it is uninformative.

As stated earlier acantholysis pertains to a process pathologic that leads to formation of round epithelial cells that are separate distinctly from their neighbors, those cells being called acantholytic ones. Acantholytic cells are observed in different kinds of processes pathologic, among those being inflammatory ones, such as pemphigus vulgaris, pemphigus foliaceus, paraneoplastic pemphigus, infection by herpes virus (herpes simplex, zoster, and varicella), and Grover’s disease; neoplastic ones, such as squamous-cell carcinoma of the solar keratotic type (so-called pseudoglandular squamous-cell carcinoma); cystic ones, such as warty dyskeratoma; hamartomatous ones, such as an epidermal nevus punctuated by foci of acantholytic dyskeratosis, and one that escapes categorization readily in the classification of classic pathology, namely, Darier’s disease. Moreover, acantholytic cells may be brought into being by different mechanisms, the two most common being immunologic, as in pemphigus vulgaris and pemphigus foliaceus, and secondary to the effects of products of neutrophils as occurs in bullous impetigo and dermatitis herpetiformis. By virtue of these different mechanisms, acantholytic cells may appear above the basal layer, as in Hailey-Hailey disease, in the spinous zone mostly, as in infection by herpes virus, in the granular zone, as in pemphigus foliaceus, and in the cornified layer, as in Darier’s disease. Whenever neutrophils enter the epidermis in large numbers, the effects of enzymes released by them may induce formation of acantholytic cells in the spinous and granular zones, as occurs in inflammatory diseases as disparate from one another as pustular psoriasis and a pustular expression of dermatophytosis. And whenever neutrophils in subepidermal spaces and in subepidermal blisters are extraordinarily numerous, as is expected in lesions developed fully of dermatitis herpetiformis and bullous lupus erythematosus, acantholytic keratocytes may come into being at the base of the epidermis.

When acantholysis occurs rapidly in an inflammatory disease, such as in pemphigus vulgaris, acantholytic cells die soon and become necrotic. When, however, acantholysis develops more slowly in an inflammatory disease, such as is the reality in Hailey-Hailey disease, acantholytic cells die more slowly, permitting time for cornification to occur as evidenced by the presence of acantholytic cells that are dyskeratotic. Acantholytic cells in squamous cell carcinoma, warty dyskeratoma, the epidermal nevus typified by foci of acantholytic dyskeratosis, and in Darier’s disease all cornify abnormally.

The notion of “villi” appearing in any disease of the skin characterized by acantholysis is misguided; what are said to be villi are merely prominent, but normal, dermal papillae crowned by an epidermal basal layer above which a cleft or blister has formed secondary to acantholysis.

Last, it should be noted that there are no truly subcorneal blisters, either as a consequence of substances immunologic or of effects enzymatic. The blisters actually develop in the uppermost part of the spinous zone or in the granular zone and by extension upward soon result in what seems to be a subcorneal blister. In reality, acantholytic cells may come into being above the basal layer (which is viable), as well as in the spinous and granular layer (which also are viable), but they cannot come into existence immediately beneath the cornified layer (which is nonviable). (SEE ACANTHOLYTIC DYSKERATOSIS)

ACANTHOLYTIC: pertaining to acantholysis

ACANTHOLYTIC CELL: an epithelial cell that has separated from other epithelial cells and in the process becomes round.

ACANTHOLYTIC DYSKERATOSIS: a condition marked histopathologically by changes in foci or in confluence of surface and/or infundibular epidermis marked by a supra-basal cleft above which cells in the spinous and granular layers have cornified prematurely (dyskeratosis) and become separated from one another (acantholysis), in conjunction often with parakeratotic cells that also have become acantholytic.

Acantholytic dyskeratosis is a distinctive pattern of changes identifiable with specificity histopathologically, it being a “reaction” only in the sense that all patterns histopathologic observed in skin and in other organs are a reaction to something; what exactly is not known with surety except for those patterns that result unquestionably from an infectious agent, such as infection by papillomavirus, the virus of molluscum contagiosum, and herpes virus. Although the denominators in common for acantholytic dyskeratosis are a suprabasal cleft and above it the presence of cells that are acantholytic and dyskeratotic, concurrently, the findings may be met with not only in foci of surface epidermis, such as in Darier’s disease, Grover’s disease, and one type of epidermal nevus, but also in a manner confluent dramatically in infundibular epidermis as occurs in the lining of a cyst known colloquially as warty dyskeratoma. Acantholytic dyskeratotic cells in epidermal epithelium are not, in themselves, sufficient to qualify as “acantholytic dyskeratosis.” For example, once a blister forms, no matter whether as a papulovesicle of Grover’s disease or as a bulla of Hailey-Hailey disease, both of those conditions being typified by the presence of acantholytic dyskeratotic cells, the findings no longer

are considered to be mere “acantholytic dyskeratosis.” Once serum has entered such a space, a cleft in an epidermis that houses acantholytic dyskeratotic cells is transformed into a blister, no matter how subtle or tiny, such as in a papulovesicle of Grover’s disease. When a lesion is a papulovesicle and no longer a papule, the term “acantholytic dyskeratosis” is not appropriate because the acantholytic dyskeratotic cells are joined by serum.

“Foci of acantholytic dyskeratosis” (focal acantholytic dyskeratosis) occur in surface epidermis of Darier’s disease and Grover’s disease, both of which are typified by lesions widespread. Parenthetically, in most patients with Grover’s disease, called originally by Grover “transient acantholytic dermatosis,” the condition is not evanescent, but persistent. Darier’s disease always consists of keratotic papules, that is, rough surfaced ones, and that is the case, too, for that expression of Grover’s disease in which each focus of acantholytic dyskeratosis is topped by a short column of parakeratosis; often, however, papules of Grover’s disease have a smooth surface because no zone of parakeratosis sits atop a focus in the epidermis in which acantholytic cells are accompanied by a tad of serum and/or a hint of spongiosis. Both the systematized and palmoplantar expressions of focal acantholytic dyskeratosis are representative of a type of epidermal nevus. In contradistinction to focal acantholytic dyskeratosis is confluent acantholytic dyskeratosis, which manifests itself usually in so-called warty dyskeratoma, which is not a neoplasm (tumor) but a particular type of infundibular cyst, the lining of which shows acantholytic dyskeratosis in confluence. Because parakeratotic contents of the cyst are housed wholly within the lining enveloping of it, the papule or small nodule neither is seen nor felt to be keratotic clinically. Much more common than warty dyskeratoma, which usually presents itself as a solitary lesion, is a keratotic papule typified histopathologically by foci of acantholytic dyskeratosis, to wit, acantholytic dyskeratotic acanthoma. Although that keratotic papule tends to appear as a solitary lesion on a trunk, it may present itself rarely as several lesions.

The acantholytic dyskeratotic changes that occur in a solar keratosis are those of very superficial expression of pseudoglandular squamous-cell carcinoma and are not those of acantholytic dyskeratosis. The two processes are different clinically, histopathologically, cytopathologically, biologically, and conceptually, although both have in common acantholytic dyskeratotic cells. In focal and in confluent acantholytic dyskeratosis, nuclei are not crowded and atypical, and acantholytic dyskeratotic cells are present in the spinous and granular zones (so-called corps ronds) and acantholytic parakeratotic cells (so-called grains) are present, too. In contrast, nuclei of cells in “pseudoglandular” solar keratosis are crowded and atypical, and acantholytic dyskeratotic cells are confined to the spinous zone; never are they found

in the granular zone or in the cornified layer. The term “pseudoglandular” derives from the gland-like appearance, at least to the eye of some histopathologists of days gone by, created by suprabasal clefts that form consequent to acantholysis in aggregations of squamous-cell carcinoma; acantholytic dyskeratotic cells with an atypical nucleus reside in those clefts. (SEE ACANTHOLYSIS)

ACANTHOMA: a benign proliferation characterized by a circumscribed increase in thickness of the epidermis consequent to an increase in thickness of the spinous (prickle, malpighian) zone secondary usually to an increase in number of cells, but episodically, to an increase in size of cells or, rarely, both of them together.

In practice the designation has been given to such disparate conditions as pale-cell acanthoma, in which the morphologic findings are of pale-cell acanthosis, and to acantholytic dyskeratotic acanthoma, in which they are of focal acantholytic dyskeratosis. In short, the term “acanthoma” is not specific and has been applied to various conditions characterized by proliferation of spinous cells.

An “acanthoma” is a tumor only in the sense of a benign proliferation, not in the sense of a swelling; most true acanthomas are but slightly elevated above the surface of the skin, e.g., pale-cell acanthoma (Degos-Civatte) and acantholytic dyskeratotic acanthoma. A so-called pilar sheath acanthoma is a proliferation with follicular differentiation, to wit, toward the isthmus, and to a much lesser extent with sebaceous ductal differentiation. The term acanthoma should not be used for tumor, unmodified, not only because no acanthoma is a tumor in the sense of a mass greater than 2.0 cm in greatest diameter, but also because no acanthoma is malignant. In the jargon of general pathology, tumor usually denotes a malignant proliferation. Neither should the term acanthoma be used for a benign proliferation with follicular differentiation nor for a hyperplasia.

“Acanthoma” is used for such disparate processes pathologic as hypertrophy (increase in size of cells) and hyperplasia (increase in number of cells) that comes into being by way of persistent mechanical trauma, as in acanthoma fissuratum, for a benign neoplasm such as large-cell acanthoma (a solar lentigo in which nuclei of keratocytes are larger than usual for that incipient reticulated seborrheic keratosis), melanoacanthoma (a pigmented seborrheic keratosis peppered by melanocytes with striking dendrites), and clear (pale)-cell acanthoma (a benign neoplasm of epidermal keratocytes that spares acrosyringia), and for lesions that are malignant, such as keratoacanthoma which is a type (keratoacanthomatous) of squamous-cell carcinoma, irrespective of whether that neoplasm arises in skin that bears hair follicles or that does not subungual. Keratoacanthoma does not qualify as a true acanthoma, because it is a malignant proliferation. (SEE TUMOR)

ACANTHOTIC: pertaining to acanthosis.

ACANTHOSIS: an increase in thickness of the epidermis consequent to an increase in thickness of the spinous (prickle, malpighian) zone secondary usually to an increase in number of cells (hyperplasia), but, episodically, to an increase in size of cells (hypertrophy) or, rarely, both of them together.

There is a difference between hyperplasia and hypertrophy of cells of the spinous zone; hyperplasia pertains to an increase in number of cells, whereas hypertrophy refers to increase in size of cells. Both hyperplasia and hypertrophy of spinous cells result in a thickened epidermis. Hyperplasia of spinous cells occurs, for example, in psoriasis, whereas hypertrophy of spinous cells appears in lichen planus. Both hyperplasia and hypertrophy of spinous cells are seen in some examples of lichen simplex chronicus.

Because the term acanthosis nearly always is employed conventionally as a synonym for thickening of the epidermis as a result of an increased number of spinous cells, it is incorrect to use it for proliferations, whether benign, such as seborrheic keratosis, or malignant, such as Bowen’s disease.

The word acanthosis also is applied aptly to hyperplasias like those of psoriasis, long-standing lesions of allergic contact dermatitis and of nummular dermatitis, and pityriasis rubra pilaris. It is imprecise to refer to acanthosis as “irregular” and “regular”; neither of those modifiers ever has been defined in a crisp, lucid way. It is proper, however, to describe epidermal rete ridges as being of even or uneven length, having a smooth or jagged outline, and being thin or thick. Each of those descriptors is definable in fashion comprehensible. In sum, although acanthosis may come into being as a consequence of hypertrophy of spinous cells, when the word acanthosis is employed in dermatopathology, it is usually meant to designate hyperplasia, not hypertrophy.

ACHROMIA: state of being devoid of color. Syn. achromatosis.

Achromia means a state of complete absence of color of the skin, but no cutaneous condition ever is devoid wholly of color, and that is why the term has no place in the language of dermatology.

The phrase so popular today among dermatologists, namely, “skin of color,” which is meant to apply only to the integument of Africans (and especially African-Americans), Asians (and especially Asian-Americans), and Hispanics (and especially Hispanic-Americans) has no basis scientific whatsoever; all peoples, including Caucasians, have “skin of color.” It is dangerous to mix political agendas suffused with financial incentives and the profession of medicine.

Often the word achromia is defined as absence of natural pigmentation, which means pigmentation by melanin, but, in fact, the skin is colored not only by melanin but by effects of the organization of corneocytes of the stratum corneum

and of the thickness of that cornified layer (i.e., it may have a slightly yellow cast when corneocytes are arranged compactly in a markedly thickened stratum corneum), degree of perfusion by blood (i.e., it may be red or livid), or by deposits of various kinds, i.e., of hemosiderin (which may cause the skin to assume a brown hue).

A reduction in pigmentation by virtue of a decrease in melanin in the epidermis may be designated specifically hypomelanosis, which is manifested clinically as hypopigmentation, and amelanosis, which is expressed clinically as depigmentation. Hypomelanosis is seen in post-inflammatory hypopigmentation, and amelanosis is observed in the depigmentation of vitiligo. Changes in color of skin may be indicative of a specific disease, for example, absence of perfusion of blood in vessels of nevus anemicus resulting in whiteness of that flat lesion. In short, a term that has applicability clinically, such as depigmentation, is preferable to one that does not, such as achromia.

What is called "achromia" in the common parlance of dermatology is not really characterized by absence of color. Achromia parasitica, achromic nevus, and incontinentia pigmenti achromians of Ito, as well as lesions in yaws or onchocercosis, are typified by hypopigmentation, not by depigmentation; the latter being a condition that results from absence total of melanin from the epidermis. A distinction must be made in this regard, therefore, between hypopigmentation in which the amount of melanin from the epidermis is reduced considerably, as is the case in one expression morphologic of the patch stage of mycosis fungoides (hypopigmented mycosis fungoides), and absence entirely of melanin from the epidermis, as is the situation in lesions developed fully of vitiligo.

ACHROMIC: pertaining to achromia, not colored.

ACID MUCOSUBSTANCE: is a histochemical term for a polymer of glucose and amino groups (mucopolysaccharides, glycosaminoglycans) that takes a stain in an acid medium.

ACINAR: pertaining to an acinus.

ACINOUS: resembling an acinus

ACINUS: a group of berry-shaped secretory parts (alveoli) of a gland that are continuous with a single duct; the smallest lobule of a lobulated gland.

The term acinus, fundamentally, has denotations morphologic, but it is defined differently in regard to different organs, such as the liver, pancreas, lung, and skin. The situation is even more confusing in regard to the relationship between acinus and alveolus. In Latin, acinus means a berry or a grape, whereas alveolus refers to a small sac. It is not correct, therefore, to define acinus as a small saclike dilatation; that is the correct definition for alveolus. In the lung, acinus

and alveolus clearly are different from one another, alveolus being a tiny air containing sac, in contrast to acinus which is made up of numerous alveoli that enter the same terminal bronchiole. In regard to the pancreas and the parotid gland, the term acinus designates the terminal part of the gland that consists of secretory cells positioned around a minute hollow opening to a duct. In dermatopathology, the words alveolus and acinus pertain to structures glandular, and they often are used interchangeably in the sense of denoting the smallest part of a gland. It would be more precise, however, to define those words in a manner terminologic analogous to that for structures anatomic in the lung, employing the term alveolus for the smallest portion of a gland and acinus for the terminal lobule of a gland that consists of a group of alveoli continuous with a single duct. (SEE ALVEOLUS)

The sebaceous gland is characterized by holocrine secretion, meaning that individual cells of the gland rupture with material released from those cells becoming the secretion itself. It is not correct to state, however that the acini degenerate. The situation is just the reverse of "degeneration"; cells of an acinus mature and the product of that maturation is sebaceous secretion. In dermatopathology, the word acinus is used either to designate the terminal part of a gland (including the eccrine and apocrine glands) or to describe the lobulated architecture of a gland like a sebaceous one. Terms like uniacinar and multiacinar derive from the second use of the word acinus. The eccrine gland and the apocrine gland are both tubular and have no lobules. Use of acinus in regard to an eccrine gland or an apocrine gland refers to the definition of that word as the terminal part of a gland.

ACNEIFORM: resembling clinically different expressions morphologic of acne vulgaris. Syn. acneform, acneiforme, acneforme.

The statement "resembling acne" does not convey a sense for how, precisely, a particular condition actually resembles acne. In reality, there are many manifestations morphologic of acne vulgaris, among them comedones, papules, papulopustules, pustules, cysts, rounded masses (conglobate), draining sinuses, and scars. Each of those lesions has an appearance distinctive, clinically and histopathologically. Acneiform lesions as they present themselves clinically, in the sense of resembling grossly those of acne vulgaris, may be encountered in conditions as disparate as halogenoderma, secondary syphilis, rosacea, tropical acne, and steroid acne, each of which is a process pathologic different from acne vulgaris.

Acneiform lesions as they express themselves histopathologically, in the sense of resembling those of acne vulgaris, may be met with in conditions as unrelated as occupational acne (in which insoluble cutting oils, to wit, machine oils, induce formation of comedones), pomade acne (in which a perfumed ointment, particularly in a scented dressing for hair,

causes comedones to come into being), and Favre-Racouchot syndrome (in which extensive exposure long standing to sunlight, as evidenced by an extraordinary amount of solar elastosis brings about comedones).

The word “acne” never should be used unmodified, any more than should the words “lupus” and “nevus.” Each of them must be employed always with a modifier, for example, acne vulgaris, acne keloidalis, and acne cosmetica, lupus vulgaris, lupus pernio, and lupus profundus, and melanocytic nevus, nevus sebaceous, and nevus unius lateris.

Just as a reader is confounded by the definition of “acneiform eruptions resembling acne lesions” because the statement does not make clear whether by “acne lesions” is meant lesions of acne vulgaris and, if that is what is intended which of the many kinds of lesions of acne vulgaris is meant. In times past rosacea was referred to by dermatologists, universally, as acne rosacea, but that practice, confusing as it was, is much less in evidence today. Rosacea is a disease different from acne vulgaris, even though both conditions have a predilection for the face, tendency to consist mostly of papules and pustules, and inclination to infundibulocentricity. It also must be emphasized that by acneiform is meant resemblance, clinically and/or histopathologically, to individual lesions of acne vulgaris, and not the disease acne vulgaris per se. Acneiform lesions in diseases other than acne vulgaris may be found at sites anatomic very different from those favored by lesions of acne vulgaris.

ACRAL: pertaining to the parts most distal of the body, referring particularly to the extremities, that is, the hands and feet, and especially the parts most distal of them, namely, fingers and toes, but also, conceptually, to other distal parts, such as the nose, the ears, and penis.

The word acral is used in the language of dermatology as a synonym for hand(s) and/or foot (feet), and especially, the fingers and toes, that is, the parts most distal of the limbs. In theory, however, acral pertains to all parts distal, such as the nose and the ears and even to the penis, but, in practice that is not the way the term is used. And that is why all the conditions whose name is prefixed by acro-, such as acrodermatitis continua (Hallopeau), acrodermatitis chronica atrophicans, and acrosclerosis, nearly always affect hands and/or feet especially and sometimes, but not always, the extremities proximal to those parts most distal.

The use of the word “acral” for hands and feet in general and for fingers and toes in particular is proper, but what is confusing is not the application of the term acral to certain sites anatomic, but the use of it to modify terms like “fibrokeratoma,” “nevi,” and “microlivedo,” instead of referring specifically to fibrokeratoma on a site acral, melanocytic nevi on a site acral, and to microlivedo (whatever that means) on a site acral.

ACRAL PARTS OF THE BODY: the distal parts, such as hands, feet, and even the penis but especially the fingers and toes, nose, and ears.

ACRAL PARTS OF THE SKIN: refers to the distal parts, especially the skin of the fingers and toes, but also the nose, ears, and even the penis.

ACRO-: a prefix meaning the furthest point and in dermatology referring to the part most distal of the extremities, especially to hands and feet, and, more particularly, to fingers and toes.

The prefix acro- usually is not employed by dermatologists as a synonym for the extremities per se, but for the distal part of the extremities, to wit, hands and feet, and, in particular, fingers and toes.

Although acro-, like acral, often pertains to the part of extremities most distal, as in the terms acrodermatitis continua (which is but one of many manifestations clinical of pustular psoriasis), acrokeratosis verruciformis (which are keratotic warty papules of Darier’s disease that occur on the dorsa of hands), and acrodermatitis chronica atrophicans (a particular manifestation of Borreliosis that affects especially the legs and feet, and culminates in broad patches of atrophy), it also is applied to conditions that affect the face as well as the distal part of extremities, for example, acrogeria (which conveys an impression of severe premature aging), acromegaly (which gives the appearance of progressive enlargement of parts affected), and acrosclerosis (which causes the forearms and hands, legs and feet, and face to look and feel tight severely) as well as to conditions characterized by considerable length or height, such as acrochordon (which is a long, cordlike polyp or papilloma known colloquially as a skin tag).

ACROSPIROMA: refers to a benign proliferation composed of cells thought to resemble those of the distal segment of eccrine ducts, i.e., acrosyringia. The term encompasses conditions of histopathologic characteristics very different from one another, among them being eccrine poroma, dermal duct tumor, poroid hidradenoma, and apocrine hidradenoma. Because the term acrospiroma is not indicative of a specific proliferation, it is best avoided in favor of a designation that actually conveys specificity.

ACROSYRINGEAL: pertaining to an acrosyringium, especially of an eccrine duct.

ACROSYRINGIUM: in general, the distal intraepithelial end portion of a duct of an adnexal gland (sebaceous, apocrine, and eccrine), but in particular and in parlance common of an eccrine duct.

Acrosyringium designates the distal intraepithelial end portion of a duct of an adnexal gland and, therefore, is not limited to the eccrine gland, the duct of which spirals

through surface epidermis. The apocrine duct, too, has a distal intraepithelial part, namely, that portion of the duct which spirals through infundibular epidermis at about mid-way through the course of it. The sebaceous duct also enters the infundibulum at the junction of it and the isthmus of a hair follicle. Acrosyringia of eccrine glands and of apocrine glands spiral in the same manner as they pass through surface epidermis and infundibular epidermis respectively. The sebaceous duct passes through the junction of infundibulum and isthmus in a straight line.

For purposes practical, the term acrosyringium is used as a synonym for the part of the eccrine duct that spirals through surface epidermis, but the apocrine duct also spirals through epidermis, albeit the infundibular component of it. That being so, a distinction clear should be made between eccrine acrosyringia and apocrine acrosyringia. None of those who employ the word “acrosyringium” in articles and texts make that distinction. It is sufficient to refer to the duct of the sebaceous gland as the sebaceous duct. When authors write of a neoplasm being derived from an acrosyringium, they mean to say that it differentiates toward cells of an eccrine duct. It cannot be stated with surety from whence cells of hidrocantoma simplex, so-called eccrine poromas, dermal duct tumor, and syringoma originate truly, and that being the case, it is best to speak and write of “differentiation toward” rather than “origin from.” In the case of syringoma, differentiation seems to be apocrine, rather than eccrine, in character, whereas for poromas, it can be eccrine or apocrine.

The cells of acrosyringia not only are different from epidermal keratocytes biologically (“separate symbionts in the epidermis”), but morphologically, too. The two, types of cells, acrosyringial and epidermal keratocytic, can be identified for what they are by microscopy conventional especially in the cornified layer of normal volar skin, where corneocytes of acrosyringia assume the appearance of a corkscrew as the duct spirals through that nonviable portion of surface epidermis. Moreover, acrosyringia of eccrine ducts largely are spared in the benign proliferations of epidermal keratocytes known as pale-cell acanthoma and in the malignant proliferation of epidermal keratocytes (a squamous-cell carcinoma) called solar (actinic) keratosis.

Last, an acrosyringium does not “extend deep into the upper dermis”; by definition it is the intraepithelial part of the duct of an adnexal gland.

ACROTRICHIAL: pertaining to an acrotrichium.

ACROTRICHIIUM: the distal segment of infundibular epidermis that traverses surface epidermis.

The definition of acrotrichium was coined by H. Pinkus as the analogue follicular of the acrosyringium, by which terms he meant to identify the distal intraepidermal segment of the follicle for the former and the distal intraepidermal

segment of the eccrine duct for the latter. Pinkus considered the funnel-shaped infundibulum to be the distal part of a follicle and he thought the acrotrichium was synonymous with infundibulum, it being the part that passes through the epidermis. In reality, however, the infundibulum is epidermis, it being infundibular epidermis in contrast to surface epidermis, and not at all a component of the follicle (the latter consisting of bulb, stem, and isthmus). It should be noted that being the case, the concept of acrotrichium is flawed irreparably, not the least of those limitations being that the word “acrotrichium,” from two Greek elements, means the distal part of the hair, whereas what Pinkus intended by it was the distal part of the follicle which, in actuality, is the isthmus, not the infundibulum as he supposed, that structure actually being epidermis.

Although Pinkus seems to have been the only one to define the term acrotrichium,” it nonetheless is used widely by authors of textbooks of dermatology and dermatopathology, although none of them states precisely what they mean by it. The confusion about the use of “acrotrichium” and “acrotrichial” derives from the notion erroneous that they are analogues of acrosyringium and acrosyringial; they are not. The acrosyringium refers specifically to the intraepidermal part of the eccrine duct, but there is no intraepidermal part of a hair follicle. The follicle ends at the infundibulum, which is a part of the epidermis, the major component of which is surface epidermis. The proximal part of a follicle is the bulb, the distal part of the isthmus, and what lies between is the stem. In brief, there is no place right for the term “acrotrichium” in the lexicon of cutaneous histology or of dermatopathology.

ACTINIC: meaning caused by radiant energy, especially by ultraviolet light.

In dermatology and dermatopathology, the adjective “actinic” designates conditions in general and changes morphologic in particular that are caused by exposure to radiant energy and especially by ultraviolet light. The term is not restricted to effects chemical of those rays, but in most instances refers to changes pathophysiological induced by them. Actinic, in the original meaning of it, refers to any kind of ray, not only to rays of ultraviolet light. Therefore changes induced by x-rays, like those responsible for radiation dermatitis, may be called properly “actinic,” but not “solar,” which refers specifically to rays from the sun. In short, actinic is a generic term that applies equally to radiation keratosis and to solar keratosis, whereas solar is more specific and is applicable, in particular, to that alteration of dermal connective tissue (solar elastosis) and to superficial squamous cell carcinoma of one type (solar keratosis) brought into being by rays of the sun.

The word “actinic” often is used synonymously with “solar” to designate changes morphologic that come into being after exposure longstanding to sunlight, i.e., solar (actinic) keratosis, which is a very superficial expression of the most common type of squamous-cell carcinoma. Whereas solar pertains only to exposure to sunlight, actinic designates any kind of ray that causes changes in tissue. Actinic (solar) keratoses and actinic (solar) elastosis nearly always are caused by ultraviolet light and, therefore, it is more precise to designate them solar keratosis and solar elastosis. The changes histopathologic in a keratosis induced by longstanding exposure to x-rays are indistinguishable from those in a keratosis brought about by chronic exposure to sunlight. Solar elastosis, however, is very different morphologically from radiation sclerosis, the former being fibrillar and the latter not at all fibrillary (being termed badly “homogenization” of collagen). The type of porokeratosis named “disseminated superficial actinic” is a consequence of exposure for many years to rays of the sun in a patient susceptible, presumably genetically.

ACTINIC GRANULOMA: a term that should not be used, because it is a misnomer as a consequence of the misperception that the condition being referred to is something other than granuloma annulare on skin damaged severely by sunlight with resultant elastophagocytosis.

ACTINO-: prefix meaning related to or caused by radiant energy, especially by ultraviolet light

ACTIVATED: not definable morphologically.

The word “activated” usually is not defined in dictionaries or in textbooks of dermatology and dermatopathology. Only the verb “to activate” is defined in general dictionaries and in some medical dictionaries, and “activated” is mentioned as the past tense of that verb. “Activate” is defined in Collins Concise Dictionary, 2001 as “to make active or capable of action,” in Merriam-Webster’s Collegiate Dictionary, 2001 as “to make active or more active,” in Taber’s Cyclopedic Medical Dictionary, 2001 as “to make active,” and in Dorland’s Illustrated Medical Dictionary, 2000 and Stedman’s Medical Dictionary, 2000 as “to render active.” Even were “activated” understood as “being made more active,” it is not clear what is meant by “active” in regard to findings in sections of tissue in which no action is apparent in the sense of anything being in motion. Lesions as they are observed histopathologically are static and, therefore, cannot be described rightly as active.

Because the term “activated” is not defined in any dictionary or: textbook of dermatology or dermatopathology, it is not clear what is meant by “activated” lesions of mycosis fungoides, and “activated” lymphocytes or T cells. It is clear, however, that none of these “activations” can be identified morphologically, either by inspection gross (clinically) or

by microscopy (histopathologically). Even if the term “activated” could be defined meaningfully, for one example, as “being stimulated by cytokines,” changes induced by cytokines may not be visualizable through a microscope conventional and those changes that are visualizable may not be caused necessarily by effects of cytokines. Moreover, if lesions clinical or changes in sections of tissue are designated “activated,” the implication is that a stage of the same process properly should be considered “inactivated.” But no such definition is to be found in any textbook of dermatology or dermatopathology, the reason being that no such concept has yet to be set forth. Therefore, there is no place in either dermatology or pathology, including dermatopathology, for the terms “active” or “activated.” Testimony to the assertion just made is the fate of what was called in the late 1940s by Arthur Allen “junctional activity” and “active” or activated “junctional nevus.” Those terms were as beloved by general pathologists and dermatologists interested in neoplasms of melanocytes as is the word “dysplasia” by them today. But, whereas “melanocytic dysplasia” (and derivatives of the concept, namely “dysplastic nevus” and “dysplastic nevus syndrome”) has had a relatively long run, i.e., nearly 30 years with the promise of several decades more, “junctional activity” and “activated junctional nevus” became passé after but 20 years, now being employed never-and with good reason. What Allen pictured in every one of many photomicrographs published was melanoma in situ.

ACTIVE: i.e., tending to act. Not definable morphologically.

In the realm of assessment morphologic in pathology, gross and microscopic, no action is observable in the sense of anything being in motion. Lesions as they are perused, clinically and histopathologically, are static. That being the case, they cannot be described rightly as being “active.”

Because no movement is discernible in lesions as they are viewed clinically and by conventional microscopy in sections of tissue, it is inaccurate to describe changes noted in them as being active. Of course there is activity, in vivo, in all lesions in the skin throughout the entire course chronologic of them, both in evolution and devolution; even a scar is associated invariably with subtle changes of remodeling, a type of activity. The rim erythematous of lesions annular, such as those of erythema multiforme, erythema annulare centrifugum, dermatophytosis, lichen planus, sarcoidosis, and syphilis, often is referred to by clinicians as the “active border.” The proper designation simply is the “border,” which can be modified by words descriptive such as erythematous, violaceous, and scaly. The periphery of lesions of all inflammatory diseases of the skin, not only those that are arcuate, annular, and polycyclic, is the zone that, in vivo, is associated with changes that are especially dynamic, and where histopathologically the infiltrate of inflammatory cells is seen to be most dense.

What Allen in the 1940s, '50s, and '60s called "active junctional nevus" was, in actuality, melanoma in situ, that being evident in photomicrograph after photomicrograph published by him. In brief, "active junctional nevus" was wrong on all three counts: the proliferations were not active as judged morphologically, the melanocytes were not confined to the dermoepidermal junction, and the "nevus" was a melanoma. It was Allen who also introduced the flawed notion of "junctional activity" of melanocytes. Not only was activity of melanocytes not discernible by microscopy conventional, but Allen never defined the term itself in crisp, comprehensible, lucid fashion; in fact, for nearly 50 years Allen insisted stubbornly that melanocytes sprung directly from epidermal histiocytes. Both "active junctional nevus" and "junctional activity" are now relics historical, just as will be the case one day for dysplasia, including melanocytic dysplasia, dysplastic nevus, and the dysplastic nevus syndrome.

ACTIVE JUNCTIONAL NEVUS: is a term that should not be employed because it has never been defined in a comprehensible way. Arthur Allen, in the late 1940's, introduced that phrase, but what he pictured in numerous photomicrographs published as stereotypical of the condition was indubitable melanoma in situ, he not acknowledging the reality of malignancy of it. Moreover, "active" indicates a dynamic that cannot be identified by microscopy; everything seen in sections of tissue is static.

ACCUMINATE: pointed.

In dermatology, the word "accuminate" usually is applied to a papule whose surface is gently, rather than sharply, pointed. Never is a papule characterized by a slender point; and that being the case the term acuminate is inaccurate as a description for any papules.

Accuminate in regard to a papule pertains to a lesion whose shape is domed to a variable degree. An examining finger can feel the hint of the gently pointed surface of the papule better than the naked eye can visualize it. Nearly always a papule comes to be pointed subtly because of spongiosis in a discrete focus of the epidermis, as is the case for an id reaction and for miliaria rubra. A keratotic plug is not pointed and, therefore does not qualify as being acuminate. So called condyloma acuminatum, an infection of mucocutaneous regions by papillomavirus, is not at all pointed, clinically or histopathologically, but is characterized by gentle papillations. Neither is so called giant condyloma acuminatum of Buschke and Lowenstein pointed, the surface of that verrucous expression of squamous cell carcinoma usually is papillated, i.e., like nipples, rather than pointed.

ADAMANTINOID: resembling morphologically adamantinoma (ameloblastoma) of the bone or oral cavity; designating aggregations of cells in a neoplasm, which are columnar

and arranged in a palisade at the periphery and separated from one another by prominent spaces in the center. This pattern is known as stellate reticulum.

The adjective adamantinoid is not defined in any dictionary in use commonly or in any textbook of dermatology or dermatopathology. The etymologic basis of it is misleading, the reason being that adamantinoid in histopathology does not pertain to resembling the hardest metal or diamond. It refers to adamantine; the designation for enamel of teeth, which indeed is very hard. Proliferations derived from cells of the enamel organ (ameloblasts) are called ameloblastomas or adamantinomas.

"Adamantinoid" is used in dermatopathology and in dermatology, for both a benign and malignant neoplasm of trichoblasts, each of which bears similarities histopathologically to adamantinoma (ameloblastoma) found in bones of the jaw. The benign proliferation is known as adamantinoid trichoblastoma and to the malignant one as adamantinoid trichoblastic (basal cell) carcinoma. The word adamantinoid as a modifier in both instances refers to the appearance histopathologically of abnormal trichoblasts in aggregations whose cells at the periphery are columnar and arranged in a palisaded, and whose cells in the rest of them are separated from another by prominent spaces that are transversed by elongated intercellular bridges. The pattern just described, known as stellate reticulum, is found stereotypically in adamantinoma, but in cutaneous pathology it is encountered in adamantinoid trichoblastoma and adamantinoid trichoblastic carcinoma. The neoplastic cells in adamantinoma are ameloblasts, not trichoblasts.

ADAMSON'S FRINGE (A-FRINGE): marks the boundary between a follicular bulb and stem, and is the site at which cells of Huxley's layer of the inner sheath lose their trichohyalin granules and becomes compactly arranged blue-gray corneocytes. Above Adamson's fringe, cornification of the inner sheath and of the hair itself is complete (i.e., orthokeratotic). Nuclei are present in cells of the inner sheath and hair in the bulb below Adamson's fringe, but no nuclei are detectable in cells in the stem above it. Dermatophytes that infect hair are able to descend to the level of Adamson's fringe, but not below it, because those fungi are able to live only in cells that have cornified completely, such as those in the stratum corneum and nail plate.

ADENO-: prefix meaning pertaining to a gland

ADENOCARCINOMA: is a malignant proliferation of epithelial cells that shows tubular (glandular and/or ductal) differentiation or consists of glandular or ductal cells. When adenocarcinoma is primary in skin, the neoplastic cells differentiate toward sebaceous glands, apocrine glands, or eccrine glands (or the ducts of those glands), or tubules of

it are lined by neoplastic cells like those glands or ducts. In a well-differentiated adenocarcinoma, some cells resemble those of normal glands, i.e., vacuolated cells of sebaceous glands or cells with secretion typical of apocrine glands. In a poorly differentiated adenocarcinoma few cells are identifiable with exactness as being sebaceous or apocrine, as two examples. Of course, any hint of “differentiation” can be helpful in the diagnosis. Some adenocarcinomas in skin are not primary there; they are metastases.

ADENOID: having the appearance of a gland or duct of a gland.

In dermatopathology, adenoid pertains to looking like a cutaneous gland or the duct of a gland as evidenced usually by formation of tubules, those structures being apocrine glands or ducts of them, and eccrine glands and the ducts of them. The term “adenoid” is not applied to an appearance like that of the third gland in the skin, to wit, the sebaceous gland, which consists of lobules rather than of tubules, or to the duct of it which, although it is tubular, cornifies, and, as a consequence, has an appearance entirely different histologically from that of the duct of an apocrine or an eccrine gland. At times, cells with glandular differentiation can be identified in a proliferation even in the absence of a single tubular structure, such as the clear cells of apocrine hidradenoma, known also as solid-cystic hidradenoma, clear-cell hidradenoma, and pale-cell hidradenoma. Neoplastic epithelial cells in an apocrine mixed tumor can be recognized for what they are by their shapes polygonal and plasmacytoid. In cutaneous pathology, unlike pathology in general, the word adenoid never is applicable to lymphoid tissue.

The designation “adenoid” for a type of seborrheic keratosis is inaccurate because the cords of pigmented epidermal cells in no way resemble glands themselves or ducts of glands, the synonym for the adenoid type of seborrheic keratosis being “reticulated,” which means netlike, as truly is the situation in time for that benign keratosis. The acantholytic type of solar (actinic) keratosis is but a stage in the evolution of what later is termed, conventionally, pseudoglandular squamous-cell carcinoma, the gland like structures coming into being as a consequence of the formation of acantholytic, dyskeratotic spinous cells above suprabasal spaces of aggregations that make up the malignant proliferation. In actuality, the suprabasal clefts bear no resemblance truly to any gland. The term adenoid cystic carcinoma is correct only partly, that particular expression of apocrine carcinoma being adenoid in the sense that those aggregations are punctuated by round spaces that house mucin, thereby conveying the impression vaguely of gland like structures in cribriform pattern. Those spaces, however, are not truly glandular and, moreover, are small, which disqualifies them as being cystic.

ADENOID CYSTIC: refers to a distinctive sieve like pattern in an epithelial neoplasm in which discrete aggregations of cells house spaces that are relatively equidistant from one another, are relatively uniform in size and shape, and contain mucin. The term is derived from the appearance of a type of carcinoma of salivary glands, but it has come to designate other malignant proliferations with a similar pattern in other sites, such as types of apocrine carcinoma and trichoblastic (basal-cell carcinoma).

The designations adenoid cystic carcinoma, adenocystic carcinoma, adenocystic basal cell carcinoma, and adenoid basal cell carcinoma are confusing. In brief, adenoid cystic carcinoma is a specific type of carcinoma that may develop in skin, where it shows signs of apocrine differentiation, in salivary glands, and in the breast, an organ whose mammary glands exhibit apocrine secretion. Adenocystic carcinoma is also a specific neoplasm that, in the skin, is better known as mucinous carcinoma, and in the breast, as colloid carcinoma. Like adenoid cystic carcinoma, adenocystic carcinoma sometimes shows evidence of apocrine secretion. In contrast, the so called adenocystic and adenoid types of trichoblastic (basal cell carcinoma), although devoid of signs of follicular differentiation (i.e., germs and papillae, bulbs and papillae, trichohyalin granules, and blue-grey corneocytes arranged compactly), are composed of follicular germinative cells and are all nodular trichoblastic (basal cell carcinomas). They do not show evidence of apocrine differentiation. It would be preferable to call these three neoplasms adenoid cystic carcinoma, mucinous carcinoma, and trichoblastic (basal cell carcinoma), because their morphologic variations are so numerous and, with rare exceptions those variations do not influence biologic behavior. Adenoid cystic carcinoma and mucinous carcinoma may arise in the skin and in the breast where their morphologic features are maintained.

ADENOMA: a benign proliferation made up of glandular or ductal cells that often assume the forms of tubules, an indication of glandular or ductal differentiation.

An adenoma is a tumor only in the sense that it is a proliferation; it may present itself clinically as a papule, nodule, tumor, or plaque. The malignant counterpart of adenoma is adenocarcinoma. An adenoma consists of cells of glandular or ductal character, and although it usually shows signs of glandular or ductal differentiation, it may not necessarily do that, i.e., some apocrine mixed tumors are entirely solid, being composed wholly of polygonal and plasmacytoid myoepithelial cells and most examples of clear-cell syringoma are devoid entirely of tubules, as are numerous examples of the poromas.

Origin is irrelevant to characterization of adenoma; it matters not from whence an adenoma derives (i.e., there is no clue to the origin of adenomas known as in cylindroma, spiradenoma, apocrine hidradenoma, and poroid hidrad-

enoma, no connection being apparent to preexisting normal glandular or ductal epithelium), only that the cells that make it up are glandular or ductal in nature and they are given to glandular or ductal differentiation as evidenced by formation of structures that resemble glands or ducts.

What is termed, conventionally, sebaceous adenoma, really is a superficial sebaceous carcinoma (this is a matter of debate) analogous to superficial basal cell carcinoma and to solar keratosis, the latter being a superficial squamous-cell carcinoma of one type. So-called cystic sebaceous adenoma is a histopathologic variant of sebaceous carcinoma and, nearly always, indicates that the patient who bears it has Muir-Torre syndrome. Adenoma sebaceum is a misnomer on both counts, namely, it is not a benign neoplasm of glandular or ductal cells and it is unrelated to sebaceous epithelium. In actuality, it is a type of follicular hamartoma joined by angiofibromatous elements and often by an increase in number of melanocytes at the dermo-epidermal junction. The findings morphologically of adenoma sebaceum are identical to those of fibrous papule of the face. Trichoadenoma is also incorrectly used. It is basically infundibular and is therefore, epidermal not glandular. Apocrine papillary cystoadenoma and tubular adenoma are correct uses.

ADENOMATOID: resembling an adenoma.

ADENOMATOUS: relating to an adenoma.

ADIPO-: prefix meaning relating to fat.

ADIPOBLASTS: known also as lipoblasts, are mesenchyme-derived presumptive fat cells that are present normally only in embryonal and fetal fat.

ADIPOCYTE: a non-epithelial cell that manufactures lipid and, when mature, sports cytoplasm consisting entirely of a large vacuole of fat. syn. lipocyte, adipose cell.

An adipocyte is not “a fat cell,” the cell is not fat, it is filled with fat! Moreover, the definition “fat cell” does not distinguish an adipocyte from a lipophage, which is a macrophage that houses fat or an adipocyte from a mature sebocyte, which is an epithelial cell that produces fat. An adipocyte makes fat which then is stored in its cytoplasm. An immature adipocyte does not display a large vacuole of fat within its cytoplasm; it may exhibit only a tiny vacuole, if one at all.

Adipocytes in the subcutaneous fat, each one of which is encircled by a capillary, are arranged in lobules intersected by fibrous septa that by virtue of the intersections create an appearance of a fenestrated pattern. An adipocyte has a cell membrane, not a cell wall. Mature fat is designated white because of the cast of it grossly, whereas immature fat in an embryo is termed brown because of its hue macroscopically. In contrast to white fat, brown fat contains many small

vacuoles in its cytoplasm, thereby earning its nickname mulberry cell.

ADIPOSE: pertaining to or consisting of adipose tissue.

ADIPOSE TISSUE: connective tissue made up of adipocytes and arranged usually in lobules separated from one another by fibrous septa.

Adipose tissue is not simply fat, like triglycerides and cholesterol, and it is not just fatty tissue because sebaceous lobules also could be said to be fatty tissue. Adipose tissue is that kind of connective tissue which consists mainly of adipocytes, i.e., nonepithelial cells that manufacture lipid and, when mature, consists almost entirely of a large vacuole of fat, the nucleus being so inconspicuous at the very periphery of the cell that it often is bypassed by the knife of a microtome. A definition of adipocytes being a cell distended by droplets of fat is not precise because that definition applies equally to mature sebocytes and to lipophages. Adipose tissue not only consists of adipocytes, but of those cells arrayed characteristically in lobules that come into being by virtue of struts in the form of fibrous septa that house blood vessels and nerves.

The subcutaneous fat consists of adipose tissue arranged in lobules that are created by intersections of fibrous septa. There are two kinds of adipose tissue in the human body, namely, brown and white, the latter being the constituent of the subcutaneous fat. Some neoplasms, such as lipomas and variations on the theme of them, such as angioliipomas and fibrolipomas, are made up of adipocytes arranged in lobules, but that does not qualify as adipose tissue because the arrangement of cells in conjunction with fibrous septa does not approximate that which is characteristic of normal adipose tissue, such as is met with in the panniculus adiposus.

Distinction between mature and immature adipose tissue turns on the presence or absence of immature adipocytes (adipoblasts). Subcutaneous tissue does not always consist of mature adipose tissue, e.g., that in an embryo.

ADNEXA: sing. adnexus; In general, structures that are adjacent or subordinate to more major anatomic parts and in dermatology in particular, structures accessory to the two major components of the skin, namely, the epidermis and the dermis, including both ones that are epithelial (the folliculo-sebaceous-apocrine unit, the eccrine unit, and the nail unit) and ones that are nonepithelial (the nerves, smooth muscles, and blood and lymphatic vessels.)

In general the term “adnexa” refers to parts that are in continuity with or in close proximity to a more major organ, such as the ovaries and fallopian tubes in regard to the uterus. In the skin, however, the word “adnexa” does not designate adjunctive parts, but rather authentic components of it, namely, those that are epithelial, such as the folliculo-

sebaceous-apocrine and eccrine units and the nail unit, and those that are nonepithelial, like nerves, smooth muscles, and vessels. In contradistinction to the adnexal (accessory) epithelial and non-epithelial components of the skin, the appendages, that is hair shaft and nail plate, are structures produced by epithelial components of the skin, to wit, cells in the matrix of a hair follicle and cells in the matrix of a nail unit, respectively. In sum, adnexa and appendages of the skin are not synonyms; appendages represent the products generative of adnexa.

The term “adnexa” is employed by most authors only for certain epithelial structures in the skin, namely, folliculosebaceous-apocrine and eccrine ones, but it is not used by them for the nail unit. Moreover, the term “adnexa” refers rightly not only to epithelial structures, but to nonepithelial ones like nerves, smooth muscles, and vessels. A part of an adnexal epithelial structure may be rooted in the subcutaneous fat, not simply in the dermis. For example, glands of apocrine and eccrine units and the bulb of follicles situated on the scalp are lodged in the subcutaneous fat. The same statement can be made about some nonepithelial adnexal structures, such as blood vessels and nerves in the subcutaneous fat that are in continuity with those same structures in the dermis. Philosophically and practically, nonepithelial structures in contiguity with epithelial structures of adnexa, for example, a follicular papilla and a perifollicular sheath juxtaposed to a follicle, are, in actuality, adnexal structures, too.

ADNEXAL: pertaining or relating to adnexa

ADNEXOCENTRIC: describes the orientation of cells particularly melanocytes of a congenital nevus around epithelial and nonepithelial structures of adnexa. These are some similar uses with “angiocentric.” However this arrangement is an expected finding in this type of nevus and therefore is redundant (as is angiocentric). (SEE ANGIOCENTRIC)

ADVENTITIA: the outermost connective tissue of an organ, vessel, or structure, but, not applied usually to organs covered by a serosa.

Often “adventitia” is said to be a synonym for “tunica externa” of the arteries and venules, which consists of connective tissue. More correctly, the term adventitia is defined as the outermost layer of any organ, except for those covered by serosa.

The term “adventitia” is employed rarely in regard to the skin and then usually in reference to the combination of papillary dermis and periadnexal dermis, those two parts of similar composition being termed the “adventitial dermis.” The word adventitial is applied, too, to the outermost part of arteries and veins, the adventitia of arteries being made up of collagen and elastic fibers, and containing tiny nerves and capillaries, the “vasa vasorum.” Sometimes, elastic fibers form an “external elastic membrane” in vessels muscular, but

it is always less prominent than the internal elastic membrane, which typically is present in arteries and not in veins.

ADVENTITIAL: pertaining to adventitia and in the skin to the combination of papillary dermis and periadnexal dermis, the two, together being designated the adventitial dermis.

AGGREGATION: collection of discrete units of the same kind of cells or fibers.

An aggregation is not a clumped mass of material, but a collection or a cluster of units of the same kind of cells or of fibers that remain discrete and separate from one another. The word derives from the Latin *grex* meaning collection or herd. In the realm of histopathology, aggregations may consist of cells or of fibers. In the sphere of clinical dermatology, when lesions of the same kind are aggregated, the phenomenon is referred to as agminated.

Aggregation and aggregate are used synonymously to designate collections of structures of the same kind, such as epithelioid cells, smooth muscle cells, blood or lymph vessels, or filaments of amyloid.

AGGREGATED: characterized by aggregation.

AGMINATED: lesions in a cluster, each of which is discrete and of a single kind.

The term agminated refers to lesions that are clustered, aggregated, or grouped, but it implies, too, that individual lesions in the group are discrete and of the same kind, like sheep in a herd. An agminated Spitz nevus would be an example of this.

The word aggregated, which sometimes is employed as a synonym for agminated, conveys the sense that the lesions are similar to one another, but it does not communicate that those lesions are discrete, i.e., not given to confluence, as is the case for ones that are agminated.

Lesions in the skin may be grouped in a fashion other than agminated, among those being herpetiform, zosteriform, corymbiform, segmental, linear, and annular. Agminated is the proper designation for discrete lesions of a single kind that are grouped together at a particular site, but that are not arranged in special distribution, such as along a dermatome, in a line, or in a ring. For that reason, lesions that are segmental and confluent are not truly agminated. Moreover, the designation “agminate folliculitis” is incorrect; the pustules, by themselves, could be considered correctly to be agminated because they are similar to one another and are present in a cluster, but the fact that they are present within “erythematous plaques” excludes them from being designated rightly agminated.

ALOPECIA: loss of hair, either physiologically or pathologically, or absence of hair from birth on.

Alopecia should refer not only to absence of hair, but also to loss of hair, in fashion either diffuse or localized. The

term alopecia conveys nothing about either the extent of loss of hair in terms of geographic range or amount of actual loss at a particular site, nor does the word communicate anything about the anatomic site or sites at which the loss of hair occurs. Loss of hair can appear only at sites where hair is present normally, i.e., the scalp as opposed to palms and soles, no hair being present normally at those latter sites. Neither is the cause of a particular loss of hair transmitted by the term alopecia, which in the vast majority of instances is not a result of an inflammatory process but of one wholly physiologic, namely, the effect of androgens on common (androgenetic) baldness. In short, alopecia is a general designation for all kinds of hair loss and for absence of hair irrespective of cause, site, or kind of follicles (terminal or vellus) affected. It may be consequent to destruction of follicles themselves, but also to inability of terminal follicles to maintain their structure, they, in the course of time, becoming vellus (as is the situation in androgenetic [common] baldness) or to severing of hair shafts of follicles whose viable epithelium remains intact (as is the situation in trichotillomania).

The term alopecia is generic for loss or absence of hair and conveys nothing about the specific kind of hair loss. No pattern typical for alopecia exists, the range being extraordinary, from a macule of alopecia areata to alopecia universalis (these two being different extents of a single pathologic process mediated by lymphocytes) and from a subtle retraction of the hair line anteriorly to near total baldness in androgenetic alopecia (those two being different extents of a single pathologic process devoid of infiltrates of inflammatory cells and mediated by the effects of hormones). Loss of hair can be caused also by factors as diverse as congenital absence of follicles (aplasia cutis), follicles rendered defective because of the effects of a drug administered systemically (i.e., cyclosporin), an inflammatory process that sends follicles in anagen into telogen (i.e., “moth-eaten” alopecia of secondary syphilis), destruction of hair shafts by an infectious agent (i.e., a dermatophyte), and a congenital abnormality of hair shafts (i.e., monilethrix). It is not correct that alopecias are inflammatory in most instances; “common baldness” is so designated because it is the most common type of alopecia far and away, being entirely physiologic and unassociated with an infiltrate of inflammatory cells. It is wrong equally to use aberrant hair growth as a synonym for alopecia, because hypertrichosis as it occurs in hirsutism also could be deemed properly a kind of abnormal hair growth. Congenital alopecia can result from absence of follicles or lack of growth of hair and, therefore, alopecia cannot be defined only as loss of hair, because it may be a consequence of absence of hair from birth.

ALOPECIC: pertaining to total loss of hair physiologically or pathologically, or to have an absence of hair from birth.

ALVEOLUS: in dermatopathology, the words alveolus and acinus pertain to structures glandular, and they often are used interchangeably in the sense of denoting the smallest part of a gland. It would be more precise, however, to define those words in a manner terminologic analogous to that for structures anatomic in the lung, employing the term alveolus for the smallest portion of a gland and acinus for the terminal lobule of a gland that consists of a group of alveoli continuous with a single duct.

AMELANOTIC: pertaining to absence of melanin.

The adjective amelanotic designates the complete absence of melanin in tissue in general and in melanocytes in particular. Absence of melanin does not mean that a tissue is necessarily nonpigmented because other pigments may be deposited in the skin, among them, hemosiderin, hematoïdin, and exogenous pigments, such as those of tattoos. The adjective amelanotic is not restricted to findings in the skin because an amelanotic metastasis of melanoma may be present in any organ.

Amelanotic is used most often to designate a type of melanoma, i.e., one that is red or skin-colored clinically devoid of melanin histopathologically. When studied by electron microscopy, the neoplastic melanocytes in an amelanotic melanoma house melanosomes, some of which may be melanized partially. A condition termed amelanosis is typified not only by absence of melanin from melanocytes, but from keratocytes, too, consequent to a defect determined genetically in the metabolism of melanin. (SEE ACHROMIA)

AMIANACEA: like asbestos. Syn. amiantaceous.

In general, amiantaceous or amiantacea means like asbestos in particular in dermatology those adjectives designate a lesion that clinically is covered by a scale so thick and hard that it resembles asbestos.

Tinea (pityriasis) amiantacea is not a disease per se, but a description of scales so thick and hard that they are reminiscent of the structure of asbestos. Some authors employ the term tinea amiantacea for a manifestation of psoriasis on the scalp, others for seborrheic dermatitis complicated by infection with bacteria, still others for an expression on the scalp of nonbullous congenital ichthyosiform erythroderma, and some as a disease unto itself. In actuality, amiantaceous as a descriptive term is analogous to ostraceous, furfuraceous, and rupial. These latter terms and amiantaceous are not used often in dermatology anymore.

AMORPHOUS: having no distinctive shape; without characteristic shape.

The adjective amorphous describes something that has no readily recognizable characteristic shape. However, all structures in the skin (and in every other organ) have an identifiable shape; none are amorphous. The word “amor-

phous,” therefore, has no place in description of gross or microscopic attributes of any organ.

In every instance in dermatology and dermatopathology in which the word “amorphous” is employed, the structure in point is not shapeless; it has a definite shape. For example, amyloid in the papillary dermis in macular and papular (lichenoid) expressions of amyloidosis is globular, as is the secretion “pinched off” by cells of an apocrine gland. Mucin is finely granular, crystals of gout are spicular, and colloid is homogeneous. Never, therefore, can the term “amorphous” be applied properly to any component of the skin.

AMPHOPHILIC: stained with both acid and basic stains, (i.e., with hematoxylin and eosin), as is expressed in a color that combines pink and blue. Syn. amphophil, amphophile, amphophilous.

Amphophilic refers to capability to stain with both acid and basic dyes as is the case for the abundant cytoplasm of abnormal melanocytes of polygonal shape in “classic” Spitz’s nevi that with hematoxylin and eosin are colored pink and blue together.

The word “amphophilic” is used correctly in current text books of dermatology and dermatopathology.

AMYLOID: homogeneous, amphophilic, often globular material (consisting of glycoproteins) that may be deposited in a variety of tissues, among them being those of the skin and subcutaneous fat, when stained by crystal violet appears red, and when examined through an electron microscope is seen to be composed of straight fibrils 7.5-10mm in diameter.

Amyloid, in the original meaning of the word, designates substances that when stained with iodine respond in a manner similar to that of starch. In the skin, however, amyloid is not a complex of mucopolysaccharides, but consists of various substances that have in common a similar appearance in sections of tissue stained with hematoxylin and eosin and with crystal violet (Congo red is ineffective for demonstrating amyloid with repeatability in the skin), as well as a consistent appearance in sections of tissue examined by electron microscopy. Biochemically, the various substances known as amyloid share the basic structure of glycoproteins, but they are very different from one another in origin and in other details. In the skin, amyloid derived from necrotic epidermal keratocytes as a consequence of animated scratching is present in the papillary dermis (at first as macular amyloidosis and, if that condition is then rubbed persistently for long, as papular [lichenoid] amyloidosis), whereas amyloid that comes into being from a systemic disease, e.g., a dyscrasia of plasma cells, may be deposited in the wall of vessels, leading thereby to leakage of erythrocytes and even to hemorrhage. Under no circumstance should amyloid be characterized as being “hyaline in appearance” because “hyaline” is a term generic that lacks specificity.

In short, the definition of amyloid as a substance morphologic does not turn at all on the origin of it or on the biochemical character of it, but on the appearance of it as assessed by conventional microscopy and electron microscopy. The term amyloid lacks specificity because there are so many different kinds of amyloid. Where as amyloid derived from necrotic epidermal keratocytes may contain melanin and be stained by anticytokeratin antibody, amyloid that comes from immunoglobulins secreted by plasma cells lacks those characteristics. Not only does some material designated amyloid come from sources other than immunocytes, but, as a rule, amyloid in the skin is made up mostly of necrotic keratocytes. Different kinds of amyloid deserve to be given different appellations. Were that practice employed, the term “amyloid,” unmodified, would become as passé as lentigo, unmodified, nevus, unmodified, and parapsoriasis, unmodified.

ANAGEN: that part of a follicular cycle during which a hair grows because the bulb is formed fully and matrical cells in the center of it mature to become the hair shaft.

Anagen is not the “growing phase of a hair follicle” and not the “growth stage of hair development,” but that part of the follicular cycle during which a hair grows. It is not the follicle that is growing, but the hair shaft that is growing as a consequence of maturation of matrical cells, just as is the case also for the inner sheath and the outer sheath, both of which represent maturation of matrical cells situated off center in the bulb. Moreover, it is not necessary to designate that part of the follicular cycle as “active” because growth of hair is never passive. Last, it is the follicle that cycles, not the hair, the stages in that cycle being known as anagen (during which the bulb of a fully formed follicle produces a growing hair), catagen (during which the bulb and stem of a follicle involutes), and telogen (during which only the isthmus of the follicle and, continuous with it, a tiny remnant of the inferior segment, i.e., bulb and stem remain, “resting” prior to the onset of anagen anew).

Vellus hair follicles proceed through the same cycle as do terminal follicles, but the former are situated more superficially, always rooted in the upper part of the reticular dermis, in contrast to terminal follicles that are based in the lower part of the reticular dermis and, sometimes, in the subcutaneous fat, such as is invariable on a normal scalp with pelage prominent. Every time reference is made to “hair cycle,” it is “follicular cycle” that is meant; the follicle cycles, not the hair.

ANALYSIS: denotes separation of elements into component parts, such as the act of assessing the components of histopathology patterns in order to come to a specific diagnosis, as of an inflammatory or neoplastic skin disease. Pattern analysis is part of the method by which the diagnoses of inflammatory and proliferative skin diseases are made. Using

pattern analysis, silhouette, algorithms, and other tools the diagnoses of such diseases can be made in an orderly way.

ANAPLASIA: a characteristic particular of a neoplasm, usually a malignant one, in which all of the cells constituent are immature, that is, nuclei of them are crowded, pleomorphic, and heterochromatic, and cytoplasm is scant, thereby precluding identification with specificity either of the cells themselves or of the proliferation itself; immaturity of cells of a neoplasm.

Reversion pertains to characteristics biologic, not to attributes morphologic; one cannot observe reversion through a microscope. For that reason, anaplasia, if it is to be employed at all, should refer to findings morphologic and not to function. Although the changes seen in anaplasia doubtlessly reflect abnormal growth of cells and possibly even reversion in growth of them, growth in any form is irrelevant to definition of the term. "Primitive" implies primary or original, and the abnormal cells of anaplasia surely cannot be judged to qualify as that. Individual cells of a proliferation may be described as immature, partially mature, or mature, but not as undifferentiated, poorly differentiated, or well differentiated; only numerous cells in proximity to one another can differentiate, that is, form structures that resemble those in normal tissues or organs of an embryo, a fetus, or an individual postnatal. If by dedifferentiation is meant "regression of a specialized cell or a tissue to simpler unspecialized form (Stedman's 2001), then despite the inadequacies of that definition, dedifferentiation is a synonym for anaplasia and should be defined, as we have proposed, in the same way, namely, morphologically, not biologically. Phrases like "loss of orientation [of cells] to one another and to their axial framework and blood vessels" and "nuclear contour angulation" are uninformative and, therefore, are unhelpful in defining "anaplasia."

The problems inherent in the definition by others of the term "anaplasia" become manifested in usage of the word. In short, there is no unanimity in regard to how anaplasia should be employed. Some authors utilize it to describe abnormalities cytopathologic that conventionally are designated "nuclear atypia," others for lack of features histopathologic they deem to be specific, and still others as a synonym for dysplasia, a term that is often invoked but has yet to be defined in a comprehensible, lucid, repeatable manner. In brief, failure to define anaplasia in a manner meaningful renders the word useless in the parlance of pathology in general and of dermatopathology in particular.

ANASTOMOSE: to connect or join structures by a union of parts in a fashion intercommunicating.

The verb to anastomose does not mean to connect structures that formerly were separated, but rather to link two structures, usually ones of the same kind. Those structures

not only can be vessels, but ones tubular such as those in apocrine cribriform carcinoma and those solid such as abnormal trichoblasts in cords and columns of fibroepitheliomatous trichoblastic (basal-cell) carcinoma. Those structures linked surely need not have been separated previously.

The verb "anastomose" is used rarely in dermatology and dermatopathology, whereas the word anastomosing, which is derived from it, is employed commonly as an adjective for a pattern, usually of a neoplasm, in which numerous communications are established among structures of similar composition. The term is not restricted to angiomatous or vascular structures as, for example, in sinusoidal hemangioma. "Anastomosing" also is employed as a synonym for a pattern of epithelium in which cords and columns are linked in a manner fenestrated.

ANASTOMOSED: characterized by anastomosis.

ANASTOMOSING: describes a pattern in which numerous communications are established among structures of similar composition, it being created by structures tubular housed in proliferations of epithelial cells that exhibit adnexal differentiation, such as apocrine mixed tumors and apocrine fibroadenomas. Fibroepithelial trichoblastic (basal cell) carcinoma (Pinkus) also is marked by anastomosis striking of cords and columns of neoplastic cells, the arrangement of elements epithelial and nonepithelial in that condition resembling windows separated by panes, i.e., fenestrated.

ANASTOMOSIS: the union of parts in a manner intercommunicating.

The word "connection" is not quite right to characterize anastomosis because it does not convey the sense that the two structures joined actually communicate with each other. A connection exists, too, between structures linked but without any communication of them. The word "union" describes better the continuity between structures in an anastomosis.

In dermatology, the word anastomosis is employed nearly exclusively to refer to communication between blood vessels. In general, the term also designates communications between structures tubular, such as between the gastrointestinal and the urogenital tracts, consequent to a union produced artifactually. In principal, the term anastomosis could be used for communication between solid structures of the same kind, i.e., trabeculae of neuroendocrine carcinoma, as well as between tubular structures of the same kind.

ANATOMY: is a science that pertains to the structure of the body of an animal (or plant) and to the relationship of the parts of the body to one another. It is predicated mostly on what is learned from dissection of a body. Gross (macroscopic) anatomy deals with structures that can be identified by a naked eye. In contrast, histologic (microscopic) anat-

omy deals with structures so minute that they can be seen only through a microscope.

ANEMICUS: anemic, bloodless.

In dermatology, the English word anemia hardly ever is encountered, where as the Latin word anemicus is used to designate pallor of lesions clinical, it's pertaining solely and specifically to reduction or absence of perfusion with blood of a tissue in a locus discrete.

The adjective anemicus, which is synonymous with anemic, appears in the term "nevus anemicus," a site of skin localized that lack the usual perfusion of blood. When erythema is induced in the skin adjacent to nevus anemicus, as is done conventionally by applying ice to the region, the lesion of nevus anemicus itself remains white, in contrast stark to the redness of the skin around it. No relationship exists between the appearance anemic of those nevi and anemia systemically. A reference to "anemic infarcts" is incorrect because the infarct is not anemic; the infarct causes pallor of tissue because the lumen of vessels that supply the site is obstructed. Moreover, in medicine in general, the word anemic is used principally to convey a relationship direct to anemia. In that sense, an anemic infarction would be an infarct caused by anemia, which certainly is not the case. In dermatology, anemic should be used only as a designation of lesions that lack perfusion with normal blood, the example consummate of that being nevus anemicus.

ANETODERMA: a kind of atrophy of the skin characterized clinically by a protrusion (colloquially referred to as an outpouching) in the form of a papule or a nodule that is so compressible it can be herniated readily and histopathologically by loss of elastic fibers especially, but also of collagen in the reticular dermis, usually in the upper half of it.

Anetoderma is a term generic for one type of cutaneous atrophy, namely, that characterized by slackness in the form of a pouch, an appearance that result from loss of elastic fibers especially, but also of collagen, in the upper half of the reticular dermis.

Atrophy results from loss of substance, and in the skin there are two basic types of it, to wit, that which involves loss of dermal papillae (with subsequent loss of epidermal rete ridges) and the other that involves loss of a rather circumscribed zone of reticular dermis. Almost always, those two expressions of atrophy are secondary to the effects of products of inflammatory cells, although the first type also may be consequent to the effects of neoplastic lymphocytes in mycosis fungoides (i.e., poikiloderma vasculare atrophicans). Loss of dermal papillae leads to atrophy clinical typified by skin that displays a shiny surface, loss of normal skin markings, and capability to wrinkle easily when it is compressed between the thumb and the forefinger. Sometimes wrinkling is apparent without intervention by the fingers

of an examiner. When there also are changes associated of hyper- and hypopigmentation, as well as telangiectases, the constellation of findings is known as poikiloderma. All forms of poikiloderma, irrespective of character fundamental, of which there are many, result from loss of dermal papillae and of epidermal rete ridges.

In contrast to attributes clinical of atrophy that develops consequent to loss of dermal papillae, atrophy clinical secondary to loss of reticular dermis is typified by a pouch that can be herniated with ease, a condition termed anetoderma. When anetoderma presents itself as papules, as occurs often on the back of young men with severe acne vulgaris, the condition is referred to wrongly as macular atrophy (it is papular, not macular). When anetoderma presents itself as a nodule (and, rarely, a tumor) of cause unknown, it was termed in times past "anetoderma of Schwenninger and Buzzi."

Atrophy may involve subcutaneous fat, as well as the skin. When huge numbers of adipocytes are lost, as they are in lipodystrophy, lupus profundus, and pancreatic panniculitis, a gully forms, which is just the opposite of what happens in anetoderma.

Anetoderma is a term generic for atrophy of the skin of one particular type. So called macular atrophy is one kind of anetoderma, but it is not a synonym for anetoderma. There are no "anetoderma-like skin changes"; there is either anetoderma or there is not. Neither is a distinction between "primary" and "secondary" anetoderma instructive; everything is secondary to something and that applies, too, to anetoderma. So-called primary anetoderma is merely anetoderma whose cause could not be determined (idiopathic). All anetodermas, therefore, are "secondary" and, that being so; stating "secondary" is redundant. Although loss of elastic tissue surely is responsible largely for the appearance of anetoderma (destruction of elastic fibers doubtlessly occurs by a process other than "elastolysis," whatever that may mean), collagen, to an extent much less, is lost along with it, as might be expected in a condition that usually comes into being as a result of the effect of large numbers of inflammatory cells whose products do not make a distinction in influence destructive between kinds of connective tissue, i.e., elastic and collagenous, although elastic tissue undergoes degeneration far more readily than does collagen.

ANEURYSM: saclike dilation of a vessel.

An aneurysm is a saclike widening of a vessel, but the word does not tell anything about the kind of vessel affected or the extent of the widening, either in terms of length or breadth of it. It may be an artery, a vein, an arteriole, a venule, a capillary or a lymphatic. The extent of dilation may be very long and broad, as in an aneurysm of the aorta, or short and rather narrow, as in a "thrombosed capillary aneurysm," which actually is a markedly ectatic venule. The aneurysm may be pulsatile if the vessel affected is one of the larger

arteries of the body, it can be filled with clotted blood if the vessel is a vein or an artery or arteriole in a person whose blood pressure is low, or it may be filled with lymph if the vessel affected is a lymphatic. The dilation may be the result of weakness acquired in the wall of the vessel, as is the case with aortic aneurysm in cardiovascular syphilis, but it also may be a consequence of an error in development embryologically, as in Marfan syndrome. In brief, the term aneurysm itself communicates nothing about the kind of vessel affected, the extent of the effect, or the cause of it.

When the term aneurysm is used without qualification further, authors who employ it usually intend it to mean a saclike dilation of arteries. Nevertheless, aneurysm also is used to designate widening of other kinds of vessels, such as veins or capillaries. It is a contradiction in terms to refer to a cirroid aneurysm because that proliferation of small blood vessels approximated closely is a hemangioma, which is not a saclike dilation of a single vessel. The term cutaneous aneurysm is imprecise because it is not the cutis which is aneurysmal, but an artery positioned in the skin. Dilation extraordinary of lymphatics in a type of “deep” lymphangioma is called, rightly, caverns and not aneurysms.

ANEURYSMAL: pertaining to an aneurysm

ANGIO-: prefix pertaining to a lymphatic or blood vessel

ANGIOCENTRIC: centered around a vessel, usually a venule, but sometimes around a larger vessel.

The word “angiocentric” is not defined in most dictionaries devoted to medical terms. Moreover, in major textbooks of dermatology and dermatopathology, no definition of “angiocentric” is provided.

Despite the fact that the word “angiocentric” is not defined in most medical dictionaries and not in any textbook of dermatology or dermatopathology, it is employed widely by authors of those textbooks. They use it to characterize lymphomas, inflammations, infiltrates, and “features” various. The term also is found in the name itself of diseases, like “angiocentric T-cell lymphoma.” Curiously, the meaning of all these designations is unclear, but that should not be surprising, given the reality that a definition of “angiocentric” never is provided. The word, literally, means “in the center of a vessel,” but that is not how the term is used by histopathologists, they seeking to describe a pattern of distribution of cells, usually of nature inflammatory, or various proliferations around vessels, those cells sometimes also being present in the wall and in the lumen of the vessels. Furthermore, it is truistic to speak of angiocentricity of cells of an inflammatory disease because in every instance the cells of such a process appear first in tissue in the vicinity adjacent immediately to venules. In regard to malignant neoplasms, angiocentric T-cell lymphoma surely is not the only lymphoma in which

T-lymphocytes are arrayed in fashion angiocentric; so, too, it is for mycosis fungoides, lymphomatoid papulosis, and adult T-cell lymphoma/leukemia, as but three examples. The arrangement of abnormal melanocytes of a congenital nevus in distribution angiocentric and adnexocentric is expected in that type of it referred to by us as “superficial and deep” The cliché “angioinvasion and angiodestruction” has become synonymous with changes reputed to be characteristic histopathologically of angiocentric T-cell lymphoma, but, as can be understood readily, that has no legitimacy because “angioinvasive” and “angiodestructive” are phenomena that can not be discerned morphologically; neither “invasion” nor “destruction” are identifiable in sections of tissue scrutinized through a microscope conventional.

ANGIOID: resembling a lymphatic or blood vessel

ANGIOMA: an increase in number of blood vessels or lymphatics, usually ones that are dilated, but not otherwise abnormal morphologically, in a lesion circumscribed.

Angioma is not necessarily a swelling or tumor; witness the tiny papule known as cherry hemangioma. Neither is it correct to designate an angioma; a tumor in the sense of neoplasm, because what is called an angioma by dermatologist and pathologists may be a hamartoma, a choristoma, or a malformation, not only a benign neoplasm, of course all are generically “proliferations” and this may be an easier term. For example, a pyogenic granuloma is a hyperplasia (it involutes in time in the absence of any treatment), a port wine stain is a malformation, a glomangioma is a hamartoma (it is composed of elements, namely, venules, glomus cells, and fascicles of smooth muscle, that are present normally in skin), and strawberry hemangioma is a benign neoplasm. Neither is a definition for angioma such as “an abnormal growth produced by the dilatation or new formation of blood vessels” precise, it being applicable equally to angiosarcoma.

The term “angioma,” either with or without the prefix lymph- or hem-, is employed usually for lesions benign of character vascular, including malformations vascular like nevus flammeus and hemangiomas like cherry angioma. When authors speak of angioma without specification further, they usually mean hemangioma, not lymphangioma. Nevus flammeus is a deformity of vessels that comes about during embryogenesis and qualifies, therefore, as a vascular malformation and not as a hemangioma. The same obtains for angioma serpiginosum; it, too, is a vascular malformation, not a hemangioma.

ANGIOMATOUS: pertaining to an angioma.

ANGIOTROPISM: not definable for purposes practical. Adj. angiotropic

A biological phenomenon that indicates growth or the turning movement of a cell or a collection of cells toward a vessel. In strictly morphologic sense, not definable.

The following terms should be used instead: 1. Intravascular: within the lumen of a vessel. 2. Perivascular: around a vessel. 3. Intramural: in the wall of a vessel.

In general, the suffix-tropism implies a movement.

In biology, tropism designates the movement of an organism in response to an external source of stimulus, usually toward or away from it.

In medicine, tropism is used to refer, i.e., to viruses and other pathogens that affect only a certain host (host tropism) or only one specific type of cell (cell tropism).

Neither the adjective “angiotropic” nor the noun “angiotropism” is defined in any of standard medical dictionary or in any textbook of dermatology or dermatopathology. Strictly speaking, the suffix-tropism implies a movement, the best example being the turning or bending phenomenon plants suffer in response to light as the environmental stimulus, called phototropism. Literally, angiotropism means a “turning towards a vessel or having an affinity for a vessel.”

Even though a term should not be used before it is defined properly, angiotropism and angiotropic are used in a variety of different circumstances. They demonstrate that angiotropic and angiotropism are not employed consistently: When referring to melanoma, they are used for cells that “occupy a pericytic location,” and authors specifically emphasize that there should be no evidence of intravasation in that circumstance. When referring to a specific type of lymphoma, so called intravascular lymphoma or angiotropic lymphoma signals exactly the opposite, namely, confinement of the neoplastic cells of lymphoma to a vascular lumina. Some authors use angiotropic synonymously with the term “angio-invasive,” and they designate it by the presence of neoplastic cells within vessel walls. Yet other authors employ angiotropic for inflammatory cells being present around vessels, that being synonymous with “perivascular,” or for inflammatory cells being present within vessel walls, that being a normal finding in any kind of perivascular dermatitis when small vessels are concerned, whereas it is a sign of vasculitis when large vessels are concerned, such as is the case, i.e., in periarthritis nodosa. Meaningful definition of a term, however, should not be dependent on the diseases entity to which it is applied, but it should be clear and independent of the disease for which it is disposed.

Many questions remain unanswered, i.e., why is angiotropism not used for distribution of melanocytes in a congenital nevus? Why not for neutrophils in nodular vasculitis? Why not for proliferations of endothelial cells of Kaposi’s sarcoma, which usually surround a preexisting vessel in the “promontorium sign”? Why not fat deposits around vessels such as amyloid in systemic amyloidosis? And why not for

presence of melanoma cells within a vessel? It appears that authors make no clear distinctions between angiotropic and angiocentric; between angiotropic and angi destructive; between angiocentric and perivascular, or between angiotropic and intravascular.

Interestingly, the majority of usages of angiotropism in literature of dermatopathology refer to malignant conditions, such as melanoma and lymphoma, which are known to be capable of spreading through sanguineous or lymphatic vessels. That journey of malignant cells, however, can not be seen through the microscope. A pathologist can only identify the precise location of cells in relation to preexisting vascular structures. In fact, intravascular as well as perivascular and angiocentric location of cells is much more commonly encountered in benign lesions than in malignant ones. But in benign lesions, we usually do not attribute any prognostic meaning to these cells, whereas neoplastic cells in and around blood vessels are often considered to be synonymous with metastasis.

Dermatopathologists seem to use the word angiotropism only after they have come to the conclusion that a lesion is a malignant proliferation. Application of the term angiotropism implies invariably that a dermatopathologist expects a certain behavior of the cells. But dermatopathologists should limit themselves to describing accurately what they really see. For this reason, the word angiotropism should not be used in a description of microscopic findings in sections of tissue. The terms intravascular, perivascular, and intramural are purely descriptive and therefore more accurate as stated earlier.

ANLAGE: in an embryo, an aggregation of cells that represent progenitors of tissue or an organ.

An anlage is an aggregation of cells in an embryo that signifies a stage incipient in the development of a tissue or an organ. Anlage is synonymous with primordium. It is imprecise to define anlage as “foundation of a subsequent development” because it is not a foundation, but an initial stage in development (which is subsequent) and because development does not necessarily mean formation of a tissue or an organ. An anlage refers specifically to that aggregation of cells visualizable as representing the very first stage in development of a tissue or an organ. At about 10 weeks in the life of an embryo, two anlagen appear on the undersurface of surface ectoderm, namely, that which gives rise to the infundibulo-apocrine-sebaceous follicular unit on one hand and that which eventuates in the eccrine unit on the other.

The word anlage is employed mostly by authors European, and especially ones German-speaking. Americans tend to use the word primordium in its stead, but, in actuality, that is not done very often. In fact, most textbooks of dermatology and dermatopathology do not provide much informa-

tion about embryology of the skin in general or structures adnexal (epithelia and non-epithelial) in particular.

ANNULAR: in the shape of a ring, syn, annulare.

The term *annulus* or *annulus* is Latin for little ring or finger ring, the Latin word for ring being *anus*. An *annulus* is circular, but the word is not synonymous with a circle; in terms geometric, an *annulus* is the region between two concentric circles, whereas a circle is solid. A lesion is round but the center is different from the border. Thus, a round lesion is not necessarily annular. In dermatology, the word *annular* applies to a lesion that is circular in the shape of a ring, and the term *nummular* to one that is circular and solid.

The word *annular* is used to describe the shape of lesions clinical. It is true that *annular* and *round* are different from one another and, therefore, should not be used interchangeably; in a lesion *annular*, the center is different from the periphery, whereas in a *round* lesion the center and the periphery are more or less the same. Nevertheless, an *annular* lesion is *round*, which means that any part at the circumference of it is equidistant from the center of it. Most lesions that are said to be *annular* clinically are not truly *annular* geometrically. The term *annular* is employed widely and incorrectly to designate lesions whose contour is closed and that may be *round*, *oval*, or *polycyclic*. *Annular* is used, therefore, interchangeably and wrongly with *circular*, *circinate*, *arcuate*, and *polycyclic*, none of which are shaped like a ring. For this reason among others, the appellation “*annular erythemas*” is a misnomer; most lesions of diseases grouped under that title have configurations *arcuate* and *polycyclic*, as well as *annular*. The term “*granuloma annulare*” derives from but a single manifestation clinical of that disease, namely, a papule or plaque ring like because of an elevated border and a depressed center. Other presentations clinical of *granuloma annulare* are discrete papules, plaques, and nodules devoid of a shape like that of a ring, and papules and plaques whose configuration is *arcuate* or *oval*, but not *annular*.

ANONYCHIA: absence of nail, i.e., nail plate.

Anonychia is defined correctly in dictionaries of medicine as well as in textbooks of dermatology as absence of nails, meaning absence of nail plates. The term tells nothing about the cause of that absence, which includes diseases of character as different as *lichen planus*, *pemphigus vulgaris*, *erythema multiforme*, *epidermolysis bullosa dystrophica*, infection by fungi, or effects adverse of trauma and drugs and even aberrations in utero that result in congenital absence of nails. The term *anonychia* is analogous to *atrichia* which designates absence of hair. The word *anonychia* alone however does not allow any conclusion to be drawn about the nature of the defect, i.e., injury to the nail matrix, any more than the word *atrichia* communicates anything about

the nature of the defect responsible for it, i.e., injury to cells in the follicular matrix.

Anonychia is employed correctly when it designates conditions in which nail plates are absent. That applies to congenital absence of nails, as well as to absence of nails that follows on inflammatory diseases (or neoplastic ones like *mycosis fungoides*) that affect the nail matrix. When the nail is absent or destroyed, *anonychia* is likely to be permanent. If, however, matrical cells remain visible after injury to them, such as occurs after episodes febrile in the extreme, then *anonychia* is likely to be transient; the nail matrix begins to manufacture a nail plate anew.

APHTHA: small ulcer on a mucous membrane.

An *aphtha* is a small ulcer that occurs on a mucous membrane. It is not restricted to the oral mucosa. When *aphthae* appear concurrently in the mouth and on the genitalia the possibility of *Behcet's disease* should come to mind. An *aphtha* is a term for a clinical feature morphologic and is not synonymous with a disease, namely, *aphthosis*. An ulcer may be situated on skin or on mucous membrane, whereas an *aphtha*, by definition, is confined to a mucous membrane. An ulcer on a mucous membrane and on skin may be surrounded by a rim of redness and may be covered by a grayish or yellowish, exudate.

Defects in *aphthosis*, in primary infection by *herpes simplex*, as well as in lesions on mucous membranes of *Behcet's disease*, are called *aphthae*. The term *aphthous ulcer* is used interchangeably with *aphtha*, but that is redundant because an *aphtha* is an ulcer, i.e., one situated on a mucous membrane. The macule, vesicle, or pustule that precedes the *aphtha* in *Behcet's disease* or *aphthosis* is not called an *aphtha*, because an *aphtha* is a small ulcer, that is, a defect in both mucosal epithelium and lamina propria. The appearance clinical and histopathological of an *aphtha* does not allow differentiation between *Behcet's disease* and recurrent *aphthosis*, but the course clinical does, i.e., oral and genital manifestation of *aphtha* and/or associated symptoms of the eyes and of the brain signifies *Behcet's disease*.

APHTHOID: similar to *aphthae*

APHTHOUS: relating to or characterized by *aphthae*

APLASIA: lack of development of a tissue or an organ.

Although “*aplasia*” designates lack of development of a tissue or an organ, it is not just a failure to develop normally; *hypoplasia* also fulfills that definition. As far as development in an embryo is concerned, *aplasia* of a tissue or an organ may be present at birth, but *aplasia* of tissue that may develop during the course of a lifetime, like of the bone marrow, may manifest itself at any time, i.e., after chemotherapy for leukemia, *aplasia* of bone marrow may result. Even in hematology it is not correct to define *aplasia* as “incomplete, retarded, or

defective development, or cessation of the usual regenerative process,” because that definition includes hypoplasia.

“Aplasia” appears almost exclusively in dermatology and dermatopathology in the term aplasia cutis congenita, a group of rare diseases congenital characterized by either lack in a locus discrete of skin and/or subcutaneous tissue. Lesions of aplasia cutis congenita may present themselves clinically as an ulcer at birth, but usually by that time they have healed with a scar that comes to be covered by a thin epidermis. Aplasia also may be employed for lack of development of any kind, like that of a limb or of red blood cells.

APLASTIC: pertaining to aplasia.

APO-: prefix meaning off, away from.

APOCRINE: denoting a method of secretion whereby the product of a gland accumulates in the apical part of cells, which then detaches in a manner that has been likened to “decapitation,” “pinching off,” and “snouts.”

The notion of “losing cellular tissue in the process of secreting” is inadequate for definition of the term apocrine because that is what happens, too, in holocrine secretion, such as that of sebaceous glands in which the cell in its entirety becomes the product of secretion. Apocrine secretion is not simply a “pinching off” of the free end of the secretory cell, but is separation complete of the apical part from the rest of the cell. That part apical of the cell is different from the rest of it because products secretory accumulate there prior to the separation taking place. The apical portion of the cell is not incorporated in the secretion, but rather itself consists largely of the product secretory. Parenthetically, it is illogical to say that apocrine secretion occurs in apocrine glands and mammary glands, because mammary glands are apocrine glands.

The purpose of apocrine glands is to produce a secretion, known colloquially as “decapitation secretion,” the exact role of which is not understood. The function appreciated best is the secretion of cells of the mammary gland, which is an apocrine gland, specialized to produce colostrum. Apocrine glands are identifiable morphologically by the apical part of cytoplasm giving the appearance of being “pinched off” or “decapitated.” An “apocrine system” per se does not exist; apocrine glands at different sites anatomic are specialized to produce secretion as different from another as “mother-milk” and cerumen. The adjective apocrine is used not only to denote a component of the *gemisch* known as a type of secretion, but to convey a sense for “pertaining to or derived from apocrine glands.” Apocrine glands are situated normally in the axillae, perineum, periumbilical region, and eyelids, and to an extent lesser, on the face and scalp.

Other clues to apocrine glands or differentiation towards them although not as specific as decapitation secretion include fringe, vacuoles in a ring at the periphery of a lumen,

mucinous cells, signet ring cells, polygonal cells, plasmacytoid cells, large round granules in cytoplasm, lipofuscin, elongated tubules, papillations, continuity of tubules (other than sebaceous ductal ones) and infundibula, and the presence, in sections from the same biopsy specimen, of a hamartoma or neoplasm that shows follicular and/or sebaceous differentiation.

However, as noted earlier, the finding of unquestionable decapitation secretion in a proliferation, primary in the skin is virtually specific for apocrine differentiation.

APOPTOSIS: a type of necrosis in which cells characterized by pyknosis and karyorrhexis are removed by phagocytosis.

If apoptosis means “the death of cells which occurs as a normal part of an organism’s development,” then every keratocyte that matures to become a cornified cell of the stratum corneum of epidermis, of the hair shaft, in the process of dying, which is the case for every cell, and of the nail plate should rightly be considered apoptotic. The “death” of any keratocyte could be deemed “programmed,” that is, to end as a dead cell, a corneocyte in the stratum corneum, in the hair shaft, or in the nail plate. The life span of every cell in the body is limited genetically, but surely not all cells are designated apoptotic. It makes no sense to invoke “requirement of energy of the process” as a criterion for differentiating apoptosis from necrosis; that requirement cannot be identified morphologically. A “deletion of selected cells in both physiologic and pathologic processes” also cannot be the basis for criteria morphologic. Characterizing apoptotic cells as “single homogenous eosinophilic necrotic cells (Civatte or colloid-bodies),” “fragmentation into membrane bound particles,” or “marked by the shrinkage of the cell, condensation of chromatin, formation of cytoplasmic blebs, fragmentation of the cell into membrane-bound apoptotic bodies” hardly justifies the concept; each of those findings are possessed by cells that in classic pathology Virchowian qualify as necrotic.

Controversies should rage (but do not) about definition of the terms “apoptosis” and “apoptotic.” The fact that definition, at times, is constructed solely on the basis of findings morphologic at other times on the basis of a process physiological, and on the basis at other times pathologic, indicates how flawed irreparably is the concept of apoptosis. Morphologically, cells said to be apoptotic are typified by pyknosis and karyorrhexis, those criteria for more than 150 years having been signs agreed on for necrosis. In the short space of 30 years, the concept of apoptosis has become accepted universally; despite the fact that it has never been defined comprehensibly. “Programmed cell death” is not a definition, but a phrase that conveys ignorance of principles fundamental of physiology and of pathology. Curiously, the concept derived initially from observation of changes affiliated with a process physiologic (i.e., necrosis of individual cells of the

bulb and the stem of a hair follicle as it involutes in catagen), but soon was extended to findings associated with nearly every process pathologic in every organ, even being invoked as the key to pathogenesis of cancer. Moreover, apoptosis sometimes is used in regard to differentiation of a process and at other times to refer to loss of differentiation of a process. Some authors claim that irradiation causes apoptosis and that the “sunburn cell” is the example stereotypical of an apoptotic cell, whereas others acknowledge that irradiation and ultraviolet light may cause necrosis. No one has yet refuted the assertion that the attributes cytologic claimed to be identifying of so-called apoptosis, to wit, pyknosis and karyorrhexis are the very same ones requisite for recognition of necrosis. All of this renders it impossible to employ the term apoptosis in a fashion rational and meaningful, but apologists for the term make no effort whatsoever to respond to criticism of it but they proceed to expand the number of conditions claimed by them to be characterized by it!

APOPTOTIC: pertaining to or characterized by apoptosis.

APPENDAGES: structures produced in the skin, emerge from the skin, and extend beyond the surface of the skin, namely, hairs and nails. Matrical cells of mature hairs are seated in a follicular bulb and matrical cells of nails are positioned in the matrix of a nail unit.

In general, appendage is a part attached to a main structure. The term does not convey anything, however, about the function of the part appended, i.e., whether it is subordinate or not. In dermatology, the term appendage designates structures that are manufactured in the skin and that extend beyond it, to wit, hair and nails. In contradistinction, the term adnexa designates epithelial (follicular-sebaceous-apocrine units, eccrine units, and matrix and bed of nail units) and nonepithelial (smooth muscles, nerves, vessels) structures that are housed entirely in the skin (and subcutaneous fat).

As is apparent readily, the term appendage often is employed when adnexal structures really are meant. The only true skin appendages are hair and nails, and those structures, per se, are not associated with any neoplasm; so-called skin appendage tumors are actually adnexal proliferations of the adnexa of skin (and/or subcutaneous fat); benign and malignant proliferations may differentiate toward epithelial and nonepithelial structures of adnexa.

ARBORIZATION: describes a tree-like shape that is a result of inward turning of epithelial structures of adnexa at the periphery of a structure such as a common wart, a seborrheic keratosis, or a pyogenic granuloma around which the change sometimes is called, inaccurately, an “epidermal collarette.” In truth, the elongated collars of epithelium are eccrine ducts and hair follicles, not epidermal rete ridges.

ARCHITECTURAL: pertaining to architecture.

ARCHITECTURAL ATYPIA AND ARCHITECTURAL DISORDER: clichés that are meaningless and, therefore, to be eschewed. For the term “architectural atypia” and “architectural disorder” to have meaning repeatable, there must be a definition agreed-on of “architectural atypia” and “architectural order,” but none exists. In fact, the standard for architectural atypia and architectural order of skin is normal skin itself and, therefore, any deviation from normal skin by any neoplasm, benign or malignant, nevus or melanoma, qualifies as architectural atypia and architectural disorder, both of which fail to inform in general and about diagnosis specific in particular.

ARCHITECTURE: in general, the design, in its entirety, of the structure of a thing; in dermatopathology, the design of an abnormal condition of the skin as assessed by visualization of the structure in its entirety and of its constituent parts.

Interestingly, neither the noun architecture nor the adjective architectural is defined in standard dictionaries devoted to medicine general or in any textbook of dermatology and dermatopathology. In the most recent edition of Dorland’s Illustrated Medical dictionary, architecture is defined tautologically as pertaining to architectural pattern; no definition of architectural pattern is provided there. In dermatopathology, architecture pertains to the overall structure of an abnormal condition as it is assessed at scanning magnification of a conventional microscope, with particular reference to each of the components, including distribution and arrangement of cells, as well as of noncellular constituents, such as fibers and deposits.

It is apparent from the quotations above that the words architecture and architectural are employed commonly in textbooks of dermatology and dermatopathology, although a precise definition of those terms cannot be found in any of them. What truly is meant by intrinsic epidermal architecture, architectural atypia, or architectural disorder is impossible to fathom; the terms are opaque. If such designations are meaningless, how can they possibly be helpful to those charged with responsibility for determining whether a particular neoplasm is benign or malignant? Ackerman never referred, ever, to “architectural atypia,” except to decry the use of it. He did introduce the concept of silhouette (architectural pattern) of a proliferation as being the representation morphologic of the behavior biologic of that particular neoplasm and, therefore, the surest route to deciding whether that neoplasm is benign or malignant. He also ridiculed repeatedly the concept of “architectural disorder,” asking rhetorically how that purported phenomenon could possibly be identified when no one had ever supplied a definition of “architectural order.” For Ackerman, the word atypia never should be used for architecture (i.e., “dysplastic nevus”) of a proliferation or for an unconventional presentation of a

common disease (i.e., “atypical pityriasis rosea”), but only for nuclear characteristics of cells, especially pleomorphism and heterochromasia (and much less so for large size and hyperchromasia).

ARCUATE: shaped like an arc or a bow. Syn. arciform.

In dermatology, the adjectives arcuate and arciform are used synonymously to describe lesions that clinically are shaped like either a bow or an arc. A lesion arcuate may come into being over time, like papules of granuloma annulare become confluent, or by growth continuous, such as a plaque of melanoma. It is imprecise to define the arcuate as being arranged in arches because that criterion is satisfied equally by the term polycyclic.

In textbooks and in articles given to dermatology and dermatopathology, the adjectives annular, gyrate, figurate, circinate, arcuate, and polycyclic often are used interchangeably or in conjunction with each other. In most instances, no definition is provided for any of them. An annulus is a ring or ring like structure, a gyrate lesion is circular, round, or spiraled, and a figurate lesion may assume any number of geometric shapes. Circinate means rounded, arcuate means formed like an arc, and polycyclic refers to a group of bow-shaped lesions that have become confluent to form a special design. Annular or gyrate lesions may evolve from ones arcuate or circinate (circular) or they may resolve asymmetrically, so that an arc or bow actually comes into existence as the lesion resolves. A single disease, such as urticaria, bullous pemphigoid in the urticarial stage of it, and mycosis fungoides in the plaque stage, may express itself in the form of all of the shapes of lesions just mentioned.

ARRECTOR: the muscle of hair erection. Syn. arrector muscle, arrector pili muscle.

Arrector has only one meaning in dermatology and dermatopathology, namely, erection of the smooth muscle affiliated with a hair follicle. A designation more correct than muscle of hair erection is muscle of follicular erection, but arrector pili muscle, arrector muscle, arrector pili, or simply arrector are synonyms for it. The arrector pili muscle is smooth muscle that originates from bulges of the isthmus of hair follicles and seems to insert at the base of epidermal rete ridges. When the muscle contracts, a papule ever so-subtle of normal skin comes into being in the form of what clinically is called “cutis anserine” by physicians and “goose bumps” by laypersons.

Some hamartomas and neoplasms may consist of smooth muscle of follicular erection, for example, leiomyoma of one type (the other type being composed of smooth muscle affiliated with blood vessels rather than with hair follicles). The so-called bulge is not single a structure but rather several bulges of isthmic epithelium. Although the exact site of insertion of the arrector pili muscle has to this day not been dem-

onstrated beyond doubt, almost certainly it is the base of epidermal rete ridges and not the dermal papillae. That is true in general for origin and insertion of muscles, to wit, from one type of structure to another of the same type.

ARTERIOLE: smallest of arteries that end as capillaries.

Arterioles sometimes are referred to as precapillary arteries, that is, the smallest arteries of the body which terminate in a network of capillaries. In contrast to capillaries themselves, the wall of arterioles contains smooth muscle. Arterioles are differentiated histologically from venules by possession of an internal elastic membrane.

Under normal circumstances, arterioles end in a network of capillaries, i.e., the superficial and deep plexuses of the reticular dermis, as well as in conditions pathologic, such as the hyperplasia known as pyogenic granuloma; the vascular malformation designated spider angioma and the benign neoplasm, referred to as cirroid aneurysm. The number of layers of smooth muscle is not relevant to establishing a vessel as an arteriole; what matters is that it contains some smooth muscle.

ARTERY: a vessel that conveys blood away from the heart and that consists of three layers termed intima (endothelium, connective tissue, internal elastic membrane), media (smooth muscle, and elastin), and externa (connective tissue). Syn. arteria.

Artery is a vessel that conveys blood away from the heart. Most arteries, therefore, carry oxygenated blood except for pulmonary and umbilical arteries in an embryo. The wall of arteries usually is thicker than those of corresponding veins and the lumen of them is smaller and rounder. The wall of an artery consists of three layers, namely, intima, media, and externa, the latter also named adventitia. The intima is made up of endothelium that covers connective tissue as well as a more or less prominent layer of smooth muscle that is surrounded by an internal elastic membrane. The media is fashioned entirely of smooth muscle in arteries of the so-called muscular type, but it also contains elastic fibers in those that are found close to the heart, e.g., the aorta. The adventitia consists of connective tissue (collagen and elastin), and contains nerves and, occasionally, blood vessels, the so called vasa vasorum. Sometimes, elastic fibers form an external elastic membrane, but that structure always is less prominent than the internal elastic membrane.

Artery is used correctly to designate vessels that convey oxygenated blood from the heart to the rest of the body. Arteries branch and become arterioles, which, in turn, become capillaries. A vascular plexus consists of arteries, capillaries, and veins, not of capillaries only. In the skin, the two plexuses in the dermis, the superficial one in the upper part of the reticular dermis and the deep one in the lower part of the reticular dermis, are connected to one another through

“intercommunicating” vessels. Arteritis means inflammation of an artery, so-called arteritis temporalis being associated commonly with arteries that may no longer be pulsating, because the lumen of the vessel has been obliterated consequent to the effects of the inflammatory process. Arteries in the skin and subcutaneous tissue always are of the muscular type, arteries of the elastic type being found only in the immediate vicinity of the heart. It is not necessary, therefore, to designate arteries in the skin “muscular.”

ARTIFACT: something that is not natural but made by human beings deliberately or by instruments technologic.

ARTIFACTUAL: not natural, made by human beings or by technology. Syn. artefactual, artificial, artifactitious, factitious, facticial.

The adjective artifactual is derived from the Latin word *arte*, meaning art, and the verb *facere*, meaning to make, it designating something that comes into being by manipulation of humans or by the effects of instruments of technology. That which is present in nature never is artifactual, whereas findings that are pathologic may come about as a consequence of a “natural” process, such as one infectious, immunologic, or metabolic, or as a result of manipulation external. That being the case, as far as changes in viable tissue are concerned, that which is artifactual also is pathologic, i.e., trichotillomania or factitious panniculitis.

Artifactual is used in dermatopathology in two different circumstances. First, it designates findings, clinically and histopathologically, that are caused by trauma inflicted by the person himself/herself or by another person. Trichotillomania and traction alopecia are examples of those phenomena. Second, all changes in sections of tissue that is introduced by defective technique of a physician in the course of biopsy, or of a machine, or of a histotech in a laboratory during the process of preparation of tissue are designated artifactual, i.e., crushing of cells during manual extraction of a specimen from the skin. Both usages are correct. The words artifactual, artificial, artifactitious, factitious, and facticial are used interchangeably. Contrary to popular belief facticial changes in the skin may be diagnosed for what they are by paying attention to the criteria for them.

ASTEATOSIS: condition of slight scaliness purported to be “dryness” secondary to decrease in normal quantity of lipids in the skin.

“Asteatosis” literally means a condition in which fat is lacking, but that is not what dermatologists intend when they use it. They mean fine scaling, usually on the extremities and particularly on the extensor aspect of the legs, a condition they call “dry skin,” which they deem to be common, and which they ascribe often to “bathing too frequently” or to “using harsh soaps.”

Some European authors use “asteatosis” synonymously with “sebostasis,” although there is no evidence at all that the fine scaling is attributable to a decrease in secretion sebaceous or in sebum. Combining the adjective “asteatotic” with the term “eczema” or “dermatitis” implies that an inflammatory disease of the skin is either caused by asteatosis (which is merely the presence of fine scales) or that the asteatosis results from the dermatitis, neither of those theses being compelling. In brief, there is no true asteatotic dermatitis or asteatotic eczema (see definition [actually absence of it] of “eczema”); asteatotic has to do only with subtle scaling and nothing at all to do with inflammation. The confusion is compounded by linking asteatotic eczema to eczema craquele, which has never been shown to be spongiotic dermatitis, and to nummular dermatitis, which is an authentic spongiotic dermatitis that may eventuate in spongiotic vesicles. In sum, if asteatotic is to be employed at all, it should refer only to paltry scaling that probably is physiologic rather than pathologic.

ASTEATOTIC: pertaining to asteatosis.

ASTEROID: in the shape of a star, in the shape of the flower aster.

“Asteroid” is derived from the Greek word *aster* (star) and designates structures that have the shape or appearance slightly of a star. In regard to appearance histopathologic, asteroid refers to a structure somewhat circular associated with elements linear oriented centrifugally, thereby simulating the points of a star. In reality, what is termed “asteroid,” conventionally, by histopathologists resembles only vaguely a star.

In dermatopathology, “asteroid” is used mostly for bodies found in macrophages, ones both mononuclear and multinucleate (histiocytic giant cell). The structures once were considered to be specific for sarcoidosis, but, in time, it became apparent that they were found in cells of granulomatous inflammation of all kinds. Moreover, “asteroid” is employed also for blastospores in sporotrichosis. In each of those instances, the term is utilized more or less correctly because “asteroid bodies,” as well as “sporothrix asteroid,” are reminiscent somewhat of a star.

ASYMMETRY: lack of symmetry; in dermatopathology, marked variation in structure of two halves of a lesion as it is assessed at scanning magnification of a microscope conventional and in dermatology uneven distribution and shape of lesions on halves of the body.

Symmetry and asymmetry in dermatopathology and dermatology usually refer to silhouette architectural of lesions histopathologically and distribution and shape of lesions clinically, the symmetry and asymmetry being assessed bilaterally. Bilateral symmetry means that a lesion can be divided

by a longitudinal plane into halves that are mirror images of one another. Never in biology, however, is a lesion as it is assessed clinically or histopathologically absolutely symmetrical. The terms symmetry and asymmetry, therefore, are relative, asymmetry usually referring to differences marked in the “halves” of a lesion than to true “absence of symmetry.” Interestingly, no definition of asymmetry can be found in any textbook of dermatopathology or of dermatology.

The terms asymmetry and symmetric are used to characterize distribution and shape of lesions clinically and silhouette of lesions histopathologically. Asymmetry is mentioned often as a criterion for malignancy, i.e., in the so-called ABCD rule (asymmetry, border irregularity, color variability, diameter > 6.00 mm) for diagnosis clinical of melanoma. Unfortunately, that simplistic mnemonic alone does not work for many “pigmented lesions” of the skin, no small number of benign conditions, such as many congenital nevi and Clark’s nevi, fulfilling the criteria, and of melanoma failing to fulfill them, chief among those melanomas being those that develop in prepubescent children, hardly any of those ever being diagnosed clinically as melanoma. Considering the fact that asymmetry is used as a criterion histopathologic for malignancy, it is surprising that no attempt has been made to define that word in any textbook of dermatopathology. In sections of tissue, only two dimensions of a lesion can be assessed. Therefore, a lesion that seems to be symmetrical in two dimensions may be asymmetrical in the third dimension, which may not be visualizable in a particular section of tissue, that reality not being uncommon in circumstance of melanoma arising in association with a melanocytic nevus and when the blade of a microtome cuts only through that part of the lesion consisting of nevus, the melanoma in conjunction with it not having been sampled.

It should be noted that symmetry or lack thereof is only one criteria (but a major one) for the diagnosis of melanoma. A constellation of criteria must be used for melanoma and for all proliferations to determine benignancy versus malignancy. Most feel there is a hierarchy of criteria. Some being more important than others. (e.g., Ackerman feels that symmetry is more important than circumscription, etc.)

ATOPY: a genetic disposition toward development of allergic rhinitis, allergic asthma, and allergic urticaria. Pruritus is a common symptom, and response to it by furious rubbing and scratching produces the skin disease known as atopic dermatitis.

ATRICHIA: means absence of hair. Syn. atrichiosis, atrichosis.

Atrichia is defined correctly as absence of hair, but the term itself does not tell anything about the reason for that absence, which may be congenital or acquired and may occur in the presence of hair follicles or in the absence of them. The adjective atrichial, therefore, refers to absence of hair, and

not to absence of follicles. Although the word alopecia usually is used in common parlance of dermatology to identify a condition characterized by absence of hair, rarely does a definition of atrichia make clear the distinction between alopecia (loss of hair) and atrichia (absence of hair), the terms not being synonymous. “Atrichia can be understood well in the context of its derivation from a- (absence of) and trichos (hair), in contrast to alopecia, which comes from the Greek alopex (fox), the reference being to loss of the mane of a fox. Not all conditions that are termed alopecia are reminiscent of a fox’s mane clinically, but all of them are characterized by loss of hair to extent various.

Rarely is the word “atrachia” employed in Textbooks of dermatology and dermatopathology. Some authors use it only to designate absence congenital of hair. Others mention atrichia in regard to absence of hair shafts in the presence of follicles, in contradistinction to aplasia in which follicles are lacking entirely. In fact, the word atrichia conveys nothing other than absence of hair, the cause of that absence not being communicated, it hardly ever being identifiable clinically. Alopecia areata, traction alopecia, and alopecia mucinosa (the latter being one of many manifestations of mycosis fungoides) may present themselves as atrichia.

ATROPHIC: pertaining to atrophy. Syn. atrophied

ATROPHODERMA: atrophy of skin, that is, reduction in amount of tissue in the dermis, especially elastic tissue but also often of collagen. Syn. atrophia cutis, atrophoderma.

Atrophoderma is atrophy of skin, but the loss essential to what is seen clinically as atrophy pertains to loss of dermis, not of epidermis. If, however, there is loss of dermal papillae, it is inevitable that that will be followed by loss of epidermal rete ridges. The loss of epidermis in that circumstance is paltry in comparison to loss of volume of dermis, and it is the latter which is responsible for the signs clinical of atrophy. An atrophoderma can be localized or widespread, as is the case, too, for many other processes pathologic of skin. Although the term has been employed for conditions like atrophoderma ulerythematosum or atrophoderma of Pierini and Pasini, the word atrophoderma does not refer to any specific condition per se. Atrophy has different causes and assumes different appearances clinically. In the majority vast of instances, atrophy is not a result of “improper” development of the skin, but a consequence of the effects of products released from inflammatory cells, lymphocytes in particular, fibrocytes, which then manufacture connective tissue altered markedly. That is what happens in conditions like atrophic lichen planus and lichen sclerosus et atrophicus. But products of inflammatory cells, in particular neutrophils, can destroy fibers and hereby induce changes of atrophy in a condition such as cutis laxa. Anetoderma is not a synonym for atrophoderma, but rather is one kind of atrophoderma, characterized

by loss of both elastic fibers, and to a lesser extent collagen, in the upper half of the reticular dermis with resultant slackness in the form of a pouch. In contrast, atrophy of dermal papillae leads to skin that displays a shiny surface, loss of normal skin markings, and facility for wrinkling easily.

Atrophoderma is used most often in dermatology for the atrophoderma of Pasisni and Pierini, which in actuality, is nothing other than a late stage of morphea. The term follicular atrophoderma is a contradiction in the sense that it is infundibular epidermis that withers, not the dermis per se. Because the term “atrophoderma” is generic and does not refer specifically to anyone condition, and because it encompasses so many different manifestations morphologic of loss of dermis, it has little utility for parlance of dermatology and dermatopathology.

ATROPHY: loss of substance consequent to reduction in amount of tissue. Syn. atrophia

Atrophy is neither a condition in which there is failure to develop fully nor an alteration regressive, but one that results from a loss of substance. It is a term that refers to reduction, morphologically, in amount of tissue. The term implies nothing about cause, mechanism, or type of tissue lost. It can occur during embryogenesis, i.e., in incontinentia pigmenti, or after birth, in poikiloderma vasculare atrophicans (mycosis fungoides) and it can follow on a process inflammatory or one neoplastic. Atrophy can affect the epidermis, the dermis, or the subcutaneous tissue, or a combination of them. When there is loss of dermal papillae and, subsequently, of epidermal rete ridges, the skin appears shiny and crinkled (superficial atrophy); when there is loss of a rather discrete zone in the upper half of the reticular dermis, the skin pouches out (midatrophy); when there is loss of adipocytes in the subcutaneous fat, a gully forms (deep atrophy).

So-called macular atrophy is actually papular; it is a type of anetoderma that comes into being by virtue of loss of elastic tissue and also of collagen in the reticular dermis. Anetoderma is but one type of atrophy; it is not a synonym for atrophy. Atrophie blanche, the end stage of livedo vasculitis, may be atrophic, but it may be characterized only by post-inflammatory pigmentary changes without any signs overt of atrophy. The best example of superficial atrophy is lichen sclerosus et atrophicus, of midatrophy anetoderma, and of deep atrophy lipodystrophy.

ATYPIA: deviation from the normal; not conforming to type. In histopathology, nuclei of cells that are crowded, pleomorphic, and heterochromatic in comparison with the usual appearance of nuclei of that particular type of cell. Syn. atypism.

The definition of “atypia” as a state of being not typical or the condition of being irregular or not conforming to type is based on derivation of the word from the Greek

a- (not) and typos (type), but it does not inform much to a student of the subject. In pathology in general, the term atypia is used specifically to designate characteristics nuclear that are different from the appearance of nuclei of normal cells in regard to size, shape, and/or color, the cliché for that being nuclei that are “large, hyperchromatic, and pleomorphic.” Curiously, a definition of atypia is found but rarely in textbooks of dermatology, dermatopathology, and general pathology, although the identification of atypia is said to be of importance crucial for identification of proliferations as being malignant. Pleomorphic nuclei, hyperchromasia of nuclei, coarse nuclear membranes, clumping of chromatin, large nucleoli, an increase in ratio of nucleus/cytoplasm, and abnormal mitotic figures often are mentioned as attributes ancillary in definitions of the term atypia. Hardly ever cited, however, is crowding of nuclei, which is much more helpful in assisting a histopathologist in regard to coming to diagnosis of malignant proliferations than are large size and hyperchromasia of nuclei. As a matter of fact, large size alone is of no importance in the distinction of a benign from a malignant proliferation; large monomorphic nuclei are not regarded as being atypical.

Hyperchromasia of nuclei in itself is of no benefit in the differentiation of a benign from a malignant process. For example, normal lymphocytes are so deeply blue when stained by hematoxylin and eosin that they seem sometimes to be black, and, therefore, lymphocytes never can be judged to be hyperchromatic. In brief, the three signs most important of atypia in order descending are crowding of nuclei, pleomorphism (variation markedly in size and shape), and heterochromasia (variation markedly in the intensity of staining). There is no correlation direct between atypia nuclear and malignancy biological witness for example, the “monster” nuclei in some dermatofibromas and xanthogranulomas, inflammatory conditions both and in some benign neoplasms like “classic” Spitz’s nevus and ancient schwannoma. In reverse, some malignant neoplasms are devoid of cells that exhibit nuclear atypia, chief among them being lymphocytes in the patch stage of mycosis fungoides, mesenchymal cells at any stage of dermatofibrosarcoma protuberans, and neoplastic cells of a metastasis to skin of renal-cell carcinoma.

“Atypia” is not used exclusively to designate attributes pathologic of nuclei. Some authors employ the term “cytologic atypia” (linguistically, it would be more correct to speak of “atypia of nuclei” or nuclear atypia), others “architectural atypia” which is meaningless utterly because no definition has been proffered ever of “architectural typhia.” Equally opaque are designations like atypical “classic” Spitz’s nevus, atypical blue nevus, and atypical fibrous histiocytoma. Clinicians also have gotten into the act of “atypia” and, in the process, have muddled matters further. For example, dermatologists not only refer to “atypical pityriasis rosea” and

“atypical psoriasis” but to atypical (dysplastic) moles, better designated Clark’s nevi, (which are the most common by far of all types of melanocytic nevi), to atypical (dysplastic) mole syndrome (which is not a syndrome because it does not consist of a group of signs (and sometimes of symptoms) that together constitute a particular condition, but rather a single finding, namely a number of Clark’s nevi, and to atypical mycobacteria (which are atypical only because *Mycobacterium tuberculosis* was judged arbitrarily to be typical). It becomes apparent that recognition of atypia is reputed to be helpful for diagnosis of malignant neoplasms such as melanoma, yet a benign neoplasm of melanocytes, namely, “classic” Spitz’s nevus, may sport nuclei more strikingly atypical than in most melanomas. It is incorrect to use terms like “endothelial atypia,” because it is not the endothelium itself of angiosarcoma that is atypical but the cells that make it up. A term like atypicality is unsatisfactory because the noun pertaining to the adjective is atypia, not atypicality, and usage of the latter term for features of malignancy of a neoplasm adds nothing noteworthy. What is meant by “reactive atypia” is mere speculation, although it is used often to convey a sense for atypia of cells of an inflammatory process (when general pathologists invoke the word “reactive,” as they often do, they mean “inflammatory process”), and a big nucleolus is not in itself a sign of atypia, it being but one attribute of an atypical nucleus. In dermatopathology, atypia should be used only to designate nuclei of cells that possess certain characteristics repeatable that pertain to size, shape, and/or color in contrast with the appearance normal of cells of the same particular type and for nothing else.

In conclusion, some noted dermatopathologists believe there really is no need to use the word atypia or atypical in dermatology or pathology including dermatopathology. Even in regard to nuclei; those can be described simply as being “large,” “pleomorphic,” or “heterochromatic” without any reference at all to atypia or atypical. The point here is to “say what you see.”

ATYPICAL MELANOCYTIC HYPERPLASIA: a phrase that is wrong conceptually and linguistically. Literally, it denotes an increase in fashion abnormal of melanocytes, but those who use the term really mean an increase in number of abnormal melanocytes; the “hyperplasia” is not, what they mean to be atypical, but rather the melanocytes themselves. Furthermore, what is designated “hyperplasia” actually is neoplasia; (although this word is also poorly defined) the proliferation of melanocytes does not involute, an attribute essential to hyperplasia authentic. The phrase “atypical melanocytic hyperplasia” superseded “active junctional nevus” and preceded “pagetoid melanocytic proliferation” in the lexicon of evasions by histopathologists from a diagnosis of “nevus” or “melanoma,” it having been in parlance common from the mid 1960s to about 1980. Those three

terms unconstructive were replaced in the early 1980s by mild, moderate, and severe “dysplasia,” that hedge being the one utilized most often to this day for the purpose of avoiding issuance of a diagnosis direct of “nevus,” “melanoma” or “melanoma arising in an existing nevus” or “I do not know” and therefore the lesion must be removed completely. Of course, why one “does not know” is discussed.

AUTOECZEMATIZATION: not definable.

Not surprisingly the term “autoeczematization” rarely is defined in dictionaries or textbooks of dermatology and dermatopathology. Leider and Rosenblum assert that the word is synonymous with autosensitization, and that is the position largely of Dorland’s Illustrated Medical Dictionary in 2000. Its definition also is not helpful because “spread of lesions from a circumscribed focus of eczema” requires a definition lucid of eczema for the phrase to have meaning and no such definition exists. Criteria morphological for recognition of autosensitization are not provided in any of the definitions just quoted. In short, the word has no place in dermatology or dermatopathology.

“Autoeczematization” is a synonym for “autosensitization” and for “id reaction,” and is claimed by some authors to be characterized by a “widespread vesicular eruption” typified histopathologically by “spongiotic dermatitis.” Other authors emphasize that the condition comes into being secondary to an existing “localized infectious focus,” (“focus of infection”) a concept that was rife in the 1930s and 1940s when such a focus of infection in the teeth, tonsils, or gall bladder was sought for any patient who had what was designated “autosensitization,” “autoeczematization,” or for “Pustular Bacterial of Andrew” (which was simply pustular psoriasis on palms and soles). Needless to say, dermatologists today are loathe to recall that once a “focus of infection” was suspected, it was not unusual for the patient to be relieved of all teeth, the tonsils, and the gallbladder.

There are no criteria histopathologic for distinguishing “autoeczematization” from allergic contact dermatitis, nummular dermatitis, or dyshidrotic dermatitis, which may manifest itself first as a localized eruption and later as one widespread that is determined by microscopy conventional to be spongiotic dermatitis.

AUTOSENSITIZATION: not definable.

Rarely is the term “autosensitization” ever defined and when it has been, the definition provided is unenlightening, i.e., sensitization towards one’s own tissues” and “sensitivity to one’s own cells fluids, or tissues.” Those phrases are reminiscent vaguely of the concept current of so-called auto-immune diseases, but what is called “autosensitization” or “autoeczematization” has nothing whatsoever to do with lupus erythematosus and rheumatoid arthritis. Moreover, the term “auto-immunization” itself is opaque. That being

true, those “auto-” words are best eschewed. See also autoeczematization.

The word “autosensitization” is used as a synonym for “autoeczematization” and “id” reaction. But no author makes any effort, let alone one serious, to define or explain any of those terms. Some authors contradict others in regard to description of features clinical of what they call autosensitization. Equally unclear is what is meant by “dissemination often widespread and quick, of a previous localized eczematous’ process” on one hand and presentation as “vesicular and on the palms” on the other. No author of a textbook

of dermatology or dermatopathology provides criteria based on findings repeatable for identification morphologic, i.e., (clinically or histopathologically) of the condition they refer to as autosensitization, and no attempt is made to distinguish histopathologically that condition reported from allergic contact dermatitis, nummular dermatitis, and dyshidrotic dermatitis. In actuality, on grounds histopathologic alone, the four “entities” cannot be differentiated one from another. It is not an overstatement that the phenomenon autosensitization is “poorly understood and somewhat difficult to describe.” It may be that the phenomenon itself is ill-conceived.