Geriatric Dermatology

G. W. KORTING

TRANSLATED BY
WILLIAM CURTH
AND
HELEN OLLENDORFF CURTH

Geriatric Dermatology

by

G.W. KORTING, M.D.

Director, Department of Dermatology,
University Hospital, Mainz, Germany

translated by

WILLIAM CURTH, M.D.

Associate in Dermatology (Retired),
College of Physicians and Surgeons, Columbia University;
Assistant Clinical Professor, New York Medical College

and

HELEN OLLENDORFF CURTH, M.D.

Assistant Clinical Professor of Dermatology (Retired),
Special Lecturer in Dermatology, College of Physicians and Surgeons,
Columbia University

1980

W. B. SAUNDERS COMPANY
Philadelphia • London • Toronto

II

W. B. Saunders Company: West Washington Square
Philadelphia, PA 19105

1 St. Anne's Road
Eastbourne, East Sussex BN21 3UN, England

1 Goldthorne Avenue
Toronto M8Z 5T9, Ontario, Canada

Library of Congress Cataloging in Publication Data

Korting, Günter Waldemar.

Geriatric dermatology.

Translation of Die Haut im Alter und ihre Krankheiten.

Bibliography; p.

Includes index.

1. Geriatric dermatology. I. Title.

RL73.A35K6713 618.97'65 80-5204

ISBN 0-7216-5495-9

Original German Edition: DIE HAUT IM ALTER UND IHRE KRANKHEITEN
Eine Altersdermatologie für die Praxis

English Edition published 1980 by W. B. Saunders Company, Philadelphia, London, Toronto

Geriatric Dermatology ISBN 0-7216-5495-9

Last digit is the print number: 9 8 7 6 5 4 3 2 1

PREFACE

At this time, when so much has been accomplished in West Germany, people are demanding perfect health and believe themselves entitled to prophylactic medical care. This, along with today's affluence, creates the additional belief in the efficacy of gerontological efforts and even in gerontal prophylactic care. Undoubtedly, the skin is an organ that is especially exposed to the environment, and after completion of its normal development relentlessly begins to age in concert with the rest of the body. This organ occupies the center of these demands.

Each practicing physician, therefore, has an obligation to become thoroughly versed in the natural course and phenomena involved in the aging of the skin.

Going beyond the knowledge of the specific character and signs of aging skin, and regardless of the added significance of the patient's history or laboratory findings, the diagnostic classification of a specific cutaneous disease can be made more certain by the knowledge that this dermatosis "fits" or does not fit in with the time of life of the patient (e.g., "juvenile acne vulgaris," "senile" purpura, etc.). When diagnosing a senile dermatosis knowledge of the *"age distribution"* of some skin diseases gives valuable hints. Examples are the increase of leukoses with immature cells in childhood and the chronic lymphadenoses of old age, in which specific cutaneous lesions usually appear 10 years after the hematologic deviations. Another example is seborrheic eczema, which appears in two maximal variants, either as Leiner's erythroderma desquamativa or as so-called senile seborrheic erythroderma. It is also relevant that the paraproteinoses (plasmacytoma, macroglobulinemia) most often develop at an advanced age, whereas the reticulogranulomatoses (mycosis fungoides, morbus Paltauf-Sternberg) lack a preference for the later periods of life.

Noteworthy also is the enormous age divergence between true chronic pemphigus and "pemphigus of old age." The fact that there exists a typical age distribution of some dermatoses is not changed by the observation that among the *frequent* senile skin diseases only the epitheliomas present a genuine

increase during old age. Disorders that are typical for senile skin are (in addition to the skin cancers) senile atrophy, keratoma senile, seborrheic warts, and senile angiomas, among others.

The intention of this compilation is to introduce to the practicing physician the phenomena of the skin and its diseases in old age. Numerous color plates will illustrate the information in the text.

The author wishes to give cordial thanks to the diligent Mrs. Gabriele Moritz, presently retired, for her great help, to Mr. Faber, the photographer at my Institute, and to the publisher, Professor Doctor P. Matis and the publisher's director, Mr. P. Reeg.

G. W. KORTING

Mainz

TABLE OF CONTENTS

General Observations

The discomforts of old age have always occupied the attention of mankind. They belong, so to speak, to the autobiographical history of the individual. This is even more valid today because life expectancy for the past hundred years has increased from 35 years to about 70 years; therefore, the percentage of older people within the total population has increased and so has our concern with the weaknesses and infirmities of old age. As early as 1909 the expression geriatrics was coined in the USA as an antonym to pediatrics. If health, according to classical French medicine, means the "silence of organs," then there is no doubt that complaints of organ dysfunction (Latin: aegritudines) in old age are increasing.

Independent of the previous statement is the fact that some diseases of old age (osteoporosis, senile arthritis deformans, etc.) have increased, apparently not only because of an increase in life expectancy. On the other hand, there is no proof for the so-called physiological death due to old age — that is, death due to senile debility (ASCHOFF, FRANKE, BRACHARZ, LAAS and MOLL). However, almost all organs present similar signs of old age, such as atrophy, pigmentations, appearance of fat within cells, or definite phenomena of repression of the collagenous and elastic connective tissues (for details, see SCHALLOCK).

The process of aging can be brought down to a common denominator by stating that man is as old as his vascular and connective tissue system (ASCHOFF states: "as old as his vascular system"; GRASSMAN: "as old as his connective tissue"). However, there are phenomena of aging in organs that do not contain "collagen" (H. J. CURTIS). In addition, the amount of collagen in tissue in general is the result of synthesizing or decomposing processes. Independent of such general indicative signs of aging, specific diseases such as arteriosclerosis and diabetes, infections, and the special pathomorphic courses of certain diseases are of determining importance in old age. For example, malignant tumors in the aged have a slower tendency to grow, to spread, and to metastasize; there is also a definite pattern of distribution of malignant tumors with regard to age, sex, and location. Cancers of the female genitals and breasts have an earlier peak; the cancer incidence in the female is higher up to the age of 50, while there is considerable preponderance of malignant tumors in the male after the age of 55 (bronchial, gastrointestinal, and prostate cancers [SEIFERT]). There is also a gerontological change of occurrence; for example, the frequency of tuberculosis in the aged has decreased in favor of bronchial cancer (R. SCHUBERT).

Typically unchanged, however, is the incidence of leukemia in the aged; such as chronic lymphadenosis between the ages of 50 and 70; other diseases that remain unchanged in their frequency are the typical blood diseases of old age, the paraproteinoses (plasmacytoma, macroglobulinemia). Generally quite typical for chronically ill and old patients is the multimorbidity (SCHUBERT, FALCK), perhaps also the polyetiology, and the multifactorial genesis of these diseases. Isolated

stigmas such as neurological changes of old age — for instance, the senile tremor or the senile walk with small, tripping steps — are well known to the laity (for details see MUMENTHALER). It seems important to mention the *immunological aspects* of aging such as the so-called *senile amyloidosis* (present in the heart in 90 per cent of people over 70, as amyloid of the vessel walls of the brain in 65 per cent of people over 80, and also in Alzheimer's glands (WRIGHT et al., 1969).

Surprisingly, the immunological aspects of aging can be found also in vertebrates (WALFORD) in the second half of life. Apparently, beginning with the fifth decade, the cellular immunity and the humoral infectious immunity decrease while gammaglobulin G and A increase and small amounts of paraprotein M (RICKEN) are found. (Many details cannot be discussed here, such as the relative constancy of the production of cortisol.) On the other hand, there is a linear decrease in the formation of male testosterone after the fortieth year of life (WELLER). The serum transaminase level increases parallel with the age level (LANG), and there are quantitative changes of the free serum amino acids in old age (THEIMER). By way of an example, the pathological-anatomical aspect of disturbed healing of wounds is obviously dependent on age.

The course of healing of wounds is generally considered to *depend on age* (DOBERAUER, ORENTREICH, and SELMANOWITZ). There is a delay caused by age in all stages of healing; it starts with the first inflammatory phase in the healing of a wound to the retardation of the reparative formation of new cells and to cicatrization. Therefore, at an advanced age healing of a wound is definitely retarded (JORNS). However, cutaneous wounds, as opposed to laparotomy wounds, do not present a striking dependency on age (ENGELHARDT, STRUCK, and HERNANDEZ-RICHTER). Some local inflammatory irritants (e.g., croton oil, carragheenin — derived from red marine algae — Translator) at times may bring about opposite effects (SELYE, et al.). In any case, the ripening process of the collagen may have special importance in the healing of wounds; in animals it takes place faster in later life (BENEKE and SCHMITT).

General Changes of Aged Skin

MAX BUERGER, in his monograph "Aging and Disease" (1947), emphasized as basic phenomena of aging the role of the reduction of the water content of tissue and its corresponding solidification. Nowadays, consideration of the physiological cutaneous changes of old age should start with the fact that the epidermis has the capacity to regenerate until death, but that the *duration of life of epidermal cells is diminished* from about 100 days in childhood to about 46 days in old age (WAGNER, KIMMIG). It is to be expected that if the rate of mitosis is reduced, it will result in gradual thinning of the epidermis or that this rate will remain constant with an increased rate of desquamation. As a rule, the basal layer remains unchanged and the thinning takes place to a large extent in the stratum spinosum (MASSHOFF). For example, Egyptian mummies (4000 B.C.) showed a cutis presenting the highest degree of histological preservation (connective tissue is preserved to a large extent), and regressive changes occur largely in the epidermis (GIACO-

METTI and CHIARELLI). (The decisive factors in the preservation of skin structures have been found to be not the embalming procedures but the dry climate —Translator.)

The clinical impression of the *transparency of senile skin,* depends on the quantitative and qualitative changes of the collagen in the dermis (MCCONKAY, K. W. WALTON, et al.). Together with the *decrease in the thickness of the epidermis* that occurs with increasing age, the borderline between epidermis and corium, which before presented a sawtooth-like appearance, flattens out and may present an almost straight line. Perhaps there is also reduced junctional cohesion (increased tendency to blister formation in old age?); in addition, there are certain functional losses, for instance the diminution of the capacity of the stratum corneum to withstand the influence of alkali (BLAER). However, there is no definite difference in the DNA content of the epidermis between young and old men, while the RNA content is 80 per cent lower in old people than in young people (PIETRZYKOWSKA and KONECKI). The fat content of the cutaneous surface shows the highest amount of free cholesterin in the very old age group (G. PETER and R. PETER). As a rule, old age presents increased cutaneous *pigmentation;* larger patches of hyperpigmented skin occur, especially in the genital and anal regions but also at the areolas (GARN and FRENCH). (Small pigmented areas will be discussed in the clinical chapter.)

In senile skin, *electron microscopy* shows to some extent foci of epidermal cells presenting deviations of size and form; for example, splitting of the basal membrane, vacuolization of cells, degeneration of melanocytes, and irregularities of pigmentation (NAGY and JÄNNER).

The senile *corium* shows *shrinkage of the collagen bundles,* while the elastic fibers degenerate but do not decrease in numbers; moreover, the mast cells also diminish in number in old age (HELLSTROEM and HOLMGREN; STEIGLEDER). It should be emphasized at this time that with increasing age the collagen bundles become arranged more parallel to the cutaneous surface rather than having the usual reticular interlacing arrangement present during youth — thus they show a behavior similar to some "presenile" skin diseases, especially scleroderma (KORTING and HOLZMANN).

Depending on age and localization, however, investigations of the quantitative behavior of collagen and noncollagenous albumin in human skin apparently show that *some cutaneous regions are not always coordinated during the process of aging* (MORSCHES, HOLZMANN, and KORTING). By physical testing, another measure of the qualities of collagenous materials can determine that the skin of rats shows a slow *decrease of its tensile strength* with increasing age (VOGEL, KOBELT, KORTING and HOLZMANN). In a similar fashion, by using skin of human cadavers one can confirm a decrease in the mechanical qualities of such skin with increasing age (HOLZMANN, KORTING, KOBELT, and VOGEL). Presumably, such collagen changes increase with old age and are caused by intra- and intermolecular reticulation of the collagen molecule. The solubility of animal collagen decreases with age, a fact that points in the same direction (RUSTEMEYER). Therefore, under physiological and pathological conditions, collagen goes through a time-dependent process of maturing (scleroderma is an example). This process results in less solubility of the collagen (VERZÁR and WALLENEGGER; BENEKE and SCHMITT; RASMUSSEN, KHALIL, G. WAKIM, and WINKELMANN).

However, this process affecting the collagenous fibrils is not the only one dependent on age; the elimination of newly

formed collagen from the cells and the decrease of the extracellular formation of fibrils and filaments should also be noted. Increasing lack of the amino acids hydroxylysine and hydroxyproline is characteristic of old age. The behavior of connective tissue cells with increasing age shows a decrease in cellular division and an increase in the polyploidy of DNA in the nucleus of the cell. Some connective tissues show the presence of a so far incompletely defined non-collagen protein (BENEKE, 1971), if we disregard the aberration in immunoglobulin synthesis leading to senile amyloid mentioned above.

The collagen concentration increases only slightly up to the age of 41, but afterward shows a definite increase up to the age of 90 (CLAUSEN, 1971). Such changes of concentration are not all synchronous for the different species of animals. The collagen fractions soluble in sodium chloride at the time of the animal's most pronounced rate of growth show qualitative changes in later life, resulting in increased ability of extraction. With regard to collagen fractions soluble in citrate or acetic acid solutions, there are definite differences as to their amounts among animals and men depending on age (for details see BENEKE). Considering the whole picture, one must emphasize that the concentration of collagen in the skin increases with age and that during the time of growth one can obtain the largest amount of all soluble collagen fractions.

It is not known whether *the smooth muscles* of the skin are subject to change in old age. Sometimes, a pseudo-increase is simulated by atrophy of the surrounding tissue; hypertrophies and hyperplasias of the cutaneous musculature may form not too rarely, whether conditioned or by chance, in different skin diseases, for instance in elephantiasis or acrodermatitis chronica atrophicans (see KORTING, 1959), and in alopecia senilis.

The *xerodermic condition of senile skin* points to an atrophy of the *sebaceous glands* as part of the process of aging. The lipids of the sebaceous glands show changes in composition during aging, especially an increase of cholesterol and squaline in the highest age groups (G. PETER, RITTER, SCHRÖPL, and R. PETER).

In old age, vacuole formation is somewhat characteristic in *the eccrine sweat glands* while a decrease in the glycogen content of *the apocrine sweat glands* is fairly typical. It is somewhat peculiar that in persons over the age of 60 sweat secretion can no longer be stopped by application of aluminum chloride solutions (BRUN and HUNZIKER), while the disappearance of the typical caprylic odors of the armpits of old people ("sexual odors") points to nonfunction stigma of the apocrine glands. Sweating caused by strong heat definitely begins later in old than in young people, but once thermal perspiration has started the amount of sweat is the same in old people as in younger persons. In contrast, in middle life the seborrheic constitutional type will perspire quickly and profusely after thermic provocation, while the youthful endogenous eczematous patient will perspire later and to a lesser degree (KORTING: Oligobradyhidrosis of the endogenous eczematous person).

Changes of the hair are seen with increasing age as a decrease in grouped hair follicles (OBERSTE-LEHN and NOBIS); the daily rate of hair growth supposedly decreases after the 70th year (PELFINI, CERIMELE and PISANU). Clinically important are differences in the type of hairiness caused by aging; shortening of hairs in old age is seen more often in men than in women. *Phenomena of virilization,* however, can be quite obvious, such as growth of hair on cheeks and upper lip and climacteric or androgenetic alopecia. Men in old age will develop increased hair growth on

the tip of the nose, vibrissae of the nasal entrance, bushy eyebrows, and hypertrichosis of the pinnae; these conditions may also have a genetic basis or be due to porphyria cutanea tarda (see VOGEL and DORN; H. PINKUS). In addition, the older man, less so the older woman, may experience a thinning of the axillary hair; absence of axillary hair in older persons cannot always be a clue to cirrhosis of the liver in the sense of Chvostek's phenomenon. In men graying of the hair takes place earlier and more often than in women; although in graying of the scalp hair, hereditary factors and hormonal situations as well as chronological age are of importance. If no other signs of aging are present, gray hair in itself can hardly be evaluated as an expression of presenescence. There are no single gray hairs; the impression of gray hair is caused by the simultaneous presence of white and pigmented hairs; moreover, black people turn gray on the average at the age of 43, about 10 years later than Europeans, who start graying at about 34 years. With regard to *pubic hair,* older women show more pronounced thinning than older men. As can be expected in older persons, the *hair roots* are fewer in the vertex region and less so at the occiput, and much of the hair is in the telogen (resting) phase (WITZEL, BRAUN-FALCO). As the individual grows older the hairshaft becomes thinner and shows a larger medullary space. Histologically, the white hair is interspersed with optically empty vacuoles and, as opposed to black hair of the same person, it presents only sporadically intact melanin granules and there are large amounts of pigmentary detritus (ORFANOS, RUSKA and MAHRLE). *Alopecia senilis* presents, when examined with the light microscope, clumping of the elastica and thickening of the walls of the arterioles in the foreground of the substrate (KADANOV and DSCHERVO). The *nails* grow more slowly in old age; and they become more and more dull and lusterless with a yellowish discoloration, which is apparently due to a certain thickening of the senile nailplate. Excessive thickening of the senile nail takes place more often on the toes than on the fingers. The toe nails may present onychogryphosis caused by deformed overgrowth, giving the nail a hooked or curved appearance. In Biblical times Daniel (4.33) stated that Nebuchadnezzar showed nails that were like bird's claws. Especially typical of the senile nail is a slow disappearance of the lunula and an increase of longitudinal ridges. Independent of the senile etiology of onychogryphosis of the big toe nails is the possible indication of a peripheral vascular disturbance. Similarly, in the animal kingdom the German red deer shows analogous de-

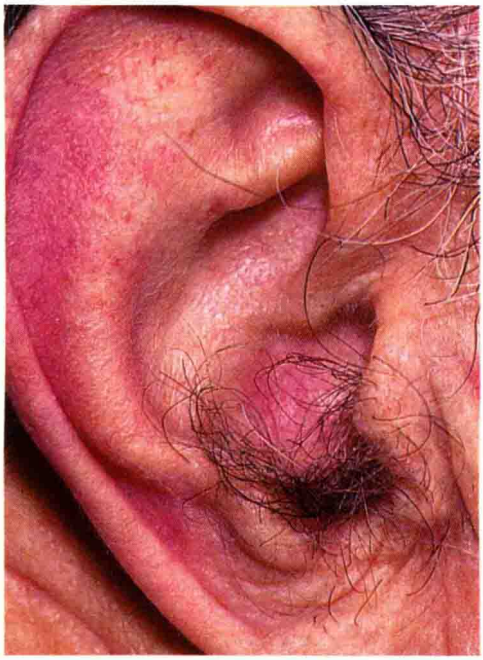

Figure 1. Hypertrichosis of ears.

Fig. a.

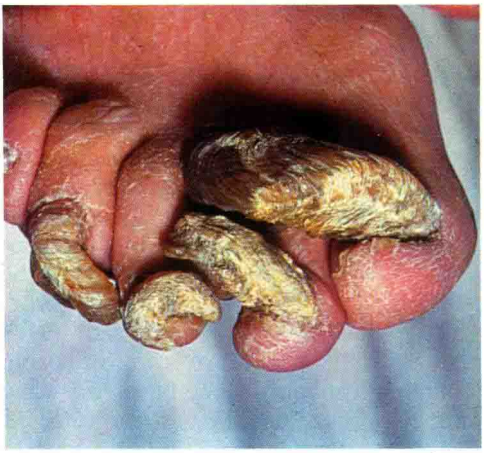

Fig. b.

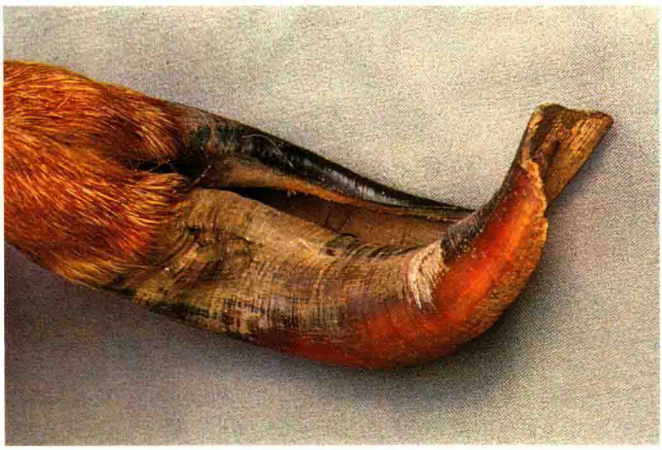

Fig. c.

Figure 2. Onychogryphosis: *a* and *b*, in man; *c*, probably analogous malformation in deer.

formities of the toes. (KORTING quotes several reports on such occurrences which appeared in German hunting journals — Translators.)

Lay people are aware of the senile change from the well vascularized "rosy" complexion of the infant to the frequently white facial skin of the senile man (*with sparse capillaries?*). But there is no definite knowledge of poor capillary supply of senile skin; PAUTRIER and WORINGER found reduction of capillaries in the papillary body in scleroderma. The general opinion, however, is that in view of the marked senile changes of the large vessels or of internal organs, *the blood vessels of the skin* lack marked changes with the exception of the vessels of the feet and legs (CRAMER, F. BÜCHNER). Old people do show a diminished functional blood supply because the speed of circulation is decreased (WALTER, KIRCHER, POLZIEN); however, sometimes younger patients suffering from endogenous eczema

show similar peripheral vascular changes (KORTING and HOLZMANN, 1960). The decrease in the circulation of the blood is accompanied by a slower capillary response to external stimuli; examples of the slowing of the circulation with age are the latency observed in pathological white dermographic response, the prolonged latency of erythema in myxedema, in ichthyosis, in endogenous eczema, or in typhus (for details see KORTING, 1954). Sphygmographic examinations show that the senile changes of peripheral arterial vessels (arcade-like flattening and decrease of amplitude of the pulse volume) are due primarily to a more or less pronounced loss of vascular elasticity accompanied sometimes by marked constriction of the lumina of the vessels (FORCK). Also, some senile phenomena of the oral mucosa — for instance, decreased salivation — are considered to be due to decreased vascular function (SHKLAR).

The increased shrinkage of the *subcutaneous fat tissue,* together with atrophy of the septa of connective tissue within the subcutaneous fat, is well known to lay persons as wrinkles and formation of *baggy eyelids;* however, age itself is only partly responsible for the typical facial expression of elderly people; the role of light and weather in the formation of the aging facial skin also must be considered. Beyond this the physician recognizes the *segmentary lipodystrophies* or the transformation of the fatty layers at the time of endocrine crises, and also the increased disposition to ruptures caused by shrinkage of the fatty tissue. However, histologic senile changes of the subcutaneous fatty tissue are denied (HILL and MONTGOMERY), except that occasionally some fat cell complexes move higher in the corium of older people (CRAMER), although the same phenomenon can be observed in younger patients with scleroderma or after cortisone treatment (KORTING, 1963). Such fatty heterotopies will never assume such dimensions as seen in nevus lipomatodes cutaneus superficialis or in focal dermal hypoplasia, the Goltz-Gorlin syndrome. In addition, in later life the borderlines between epidermis and cutis and between cutis and subcutaneous fat begin to flatten out; this may explain the before-mentioned formation of senile wrinkles. Changes of the fatty tissue are also of importance in the senile changes of the human *tongue.* The tongue of the young person possesses a uniformly dark red musculature; however, as observed by autopsy, later in life there occurs in the inner part of the tongue a striated yellow-gray design, and inspection shows *"lingua glabra senilis"* due to the leveling of the papillae. Typical of the tongue of old age is *physiological lipomatous atrophy* (BÄSSLER, BILDEN, and LANGE). The blood vessels on the underside of the tongue develop *caviar-like* grains or globules (MENDES DA COSTA); histologically, these blood vessels show dilatations caused by a decrease of tissue elasticity (KOCSARD et al.).

Other stigmata of old age will not be discussed here, such as *elongation of the lobule* of the ear (FLEISCHER), the ampullary sac formations of the apocrine ceruminal glands of the ear canal (HORSTMANN), the *degenerative senile changes* of the zonula zinnii of the human eye, etc. (GÄRTNER).

Only *the functional reaction of aging on the surface of the skin* will be discussed. Beginning in the seventh decade the pH value of light-exposed skin becomes increasingly acid, while the skin's *resistance to alkali* and *its capacity to neutralize acid* are decreased. Together with these there is an increase in the water soluble areas of the corium ("amino acid mantle"). Also the *sebaceous mantle* — soluble parts of scrapings of the corium (fatty acids, fats and waxes) — decreases with increasing