Skin Problems in the Elderly

Edited by

Lionel Fry

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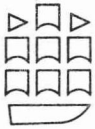

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Introduction

To a great extent the medicine of today and tomorrow is the medicine of old age. In every hospital in the Western World old patients predominate. In the past it was too readily assumed that either the medicine of old age was confined to degenerative disease and was uninfluenced by diagnosis and treatment; or that it was identical to the medicine of young and middle age and required no special study. Neither view is correct. It is now becoming clear that the diseases which strike old people, the symptoms and the signs which are induced, and the response to treatment are distinctive. Years of growth, maturation and decline alter the response of the host to disease and to its management in ways which require special study. As this fact has been grasped medical science and research-minded clinicians have embarked on the study of the diseases of late life and have documented their characteristic features. Progress has been slow, partly because of an initial lack of sense of urgency, and difficulty in attracting research workers and funds; partly because of the complexities of defining normal values in old age and of attributing deviations from the normal to any one cause. Methodological and statistical problems have compounded the difficulties. But over the years there has been a very real and impressive growth of knowledge of the medicine of late life.

Some years ago the idea was conceived of collecting this new knowledge, system by system, in a series of volumes to be entitled 'Medicine in Old Age'. These books were addressed to physicians in all Western countries and in all medical disciplines who dealt with elderly patients. The contributors included physiologists, pathologists, epidemiologists and community physicians, as well as general internal physicians, geriatricians, psychiatrists and specialists in the various systems of the body. The response accorded to the first few volumes in the series was most encouraging, and the publishers are continuing and expanding the series.

This enterprise is supervised by an Editorial Board composed of practising clinicians and academics on both sides of the Atlantic. The Board selects the topics and appoints the guest editors for each volume and has been fortunate in its choice as editors of leaders in each field. These have been able in turn to attract contributions of high merit from many countries, thus putting into

the hands of the reader a series of highly authoritative volumes. These bring together a wealth of knowledge and the best of modern practice in the care of elderly patients, retaining the critical spirit in the evaluation of the data which is characteristic of medicine in all age groups. The volumes are intended to stand mid-way between the immediacy of the scientific journal and the urbanity of the standard text book, combining freshness with authority. It is hoped that the profession will find them of value.

Birmingham 1984 Bernard Isaacs

Preface

Unlike many other specialties, there is not an increasing preponderance of elderly persons in dermatological clinics and wards. Many of the commonest skin disorders have their highest incidence in childhood and adolescence. However, because people are living longer, dermatologists are seeing an increase in a number of conditions associated with the ageing process. These include benign and malignant neoplasms, certain types of eczema, abnormalities of pigmentation and changes in the hair and nails. In addition there are changes in the skin simply due to ageing, of which the practising physician must be aware. There are some skin disorders such as psoriasis which may occur at any age, but the therapeutic approach may be different at different ages. Thus there is a need for a text which concentrates on those skin disorders most commonly seen in the elderly and for emphasis on the therapeutics of these diseases in old age. It is hoped the book will fulfil this need and be of value to the family physician, geriatrician and dermatologist.

I am grateful to my colleagues, many of whom are world experts in the subjects on which they have written, for contributing to this book.

London, 1985

Lionel Fry

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Biological aspects of ageing skin

> The true aim of science is the discovery of all operations and all possibilities of operations from immortality (if it were possible) to the meanest mechanical practice.
>
> Sir Francis Bacon

OVERVIEW

It is generally well appreciated that the skin and its appendages undergo characteristic changes with advancing age. In fact, these cutaneous clues are usually the first pieces of data one uses in appraising an individual's age. Unfortunately, we really know very little about the anatomical alterations, and even less about the physiological impairments which accompany these overt morphological changes.

Although certain aspects of cutaneous ageing have been examined, the ease by which these ageing changes can be observed poses a problem — it predisposes investigators to perfunctory estimations that are so readily accepted it makes experimental verification seem superfluous. These problems have been further compounded since many investigators have failed to differentiate changes which are due to the impact of environmental insults, whose effects may be cumulative and time dependent, from those resulting from innate ageing processes. Nor has it been generally appreciated that the skin is a very complex organ with pronounced regional differences in form and function as well as susceptibility to influences from both within and outside the organism. Thus, rather than having a solid body of knowledge concerning the dermatological aspects of gerontology and geriatrics, we have a rag-bag of cursory observations of dubious validity that are often inconclusive and sometimes contradictory

Ignorance to this extent is hazardous, for it opens up an area of intense human concern to conscienceless exploitation by charalatans and commercialists. Entrepreneurs have not failed to respond to how most people feel about the loss of their youthful appearance. Treatment emporia have sprung up all over the world and miracle rejuvenating cosmetics are readily available. Even well-meaning physicians have begun to recommend questionable and

insufficiently tested procedures for correcting defects of aged skin.

With this in mind, certain theoretical aspects of cutaneous ageing will be briefly considered. Then those age-associated changes that have been reported for various cutaneous compartments as well as integrated organ function will be surveyed. The chapter finishes by being concerned with the notion that these cutaneous clues might provide a useful measure of an individual's physiological age.

THEORETICAL ASPECTS OF CUTANEOUS AGEING

It should be mentioned at the outset that the numerous theories which have been proposed to explain ageing in general will not be examined in depth. Indeed, all existing theories are essentially variations of the same two general themes that were recognised nearly 70 years ago by C.M. Child (1915) in his classic book, *Senescence and Rejuvenescence*. According to one view, ageing results from some kind of 'wear and tear' such as the accumulation of toxic wastes, the depletion of critical, irreplaceable materials or the cumulative effects of more or less random events which damage the organism more than it can repair. The other view supposes that ageing is genetically programmed by some sort of pacemaker that might be the ultimate extension of differentiation or the expression of the gradual but inevitable breakdown of homeostatic mechanisms due to an ill-adapted genotype. In the terms proposed by Comfort (1956), these are the epiphenomenalist and fundamentalist views respectively; and the reader is referred to any one of several excellent texts (Strehler, 1962; Kohn, 1971; Comfort, 1972) for a more in depth treatment of these theoretical aspects.

As far as cutaneous ageing is concerned, it is quite clear when one compares the face of a farmer with that of an office worker of comparable age, that chronic exposure to environmental insults, such as sunlight, can greatly affect skin. The term 'photoageing' has been coined to describe this process. Of course, other types of physical, chemical and mechanical trauma also impinge continuously on the skin. Unless protective steps are taken, the ensuing damage will result in a decrease in the functional ability of the skin with the passage of time, in concordance with the epiphenomenalist view. On the other hand, the existence of several progeroid syndromes, such as Hutchinson-Gilford and Werner's, as well as other genodermatoses such as xeroderma pigmentosum, indicates that such hereditary diseases may result in the premature appearance of old age. This suggests, in agreement with the fundamentalist view, that their phenotypic manifestations of cutaneous ageing result from genotypic changes. Thus, rather than holding these two views to be mutually exclusive, there is good reason to treat them as interrelated, especially when considering age-associated changes in exposed areas. However, this does not mean that 'photoageing' merely represents an accentuation or acceleration of the intrinsic ageing process, in which the changes observed in the protected areas are qualitatively similar but of lesser magni-

tude, as has been suggested by several workers (Montagna & Carlisle, 1979; Lavker, 1979). Indeed, as recently reported by Braverman & Fonferko (1982a,b), qualitative differences between exposed and non-exposed sites exist as well. This means that one of the most important tasks that lies before us is to dissect out those age-associated changes in skin structure and function which are inevitable from those due to external assaults that may be avoidable.

SURVEY OF AGE-ASSOCIATED CHANGES IN SKIN STRUCTURE AND FUNCTION

The fact that skin is architecturally a very complex organ is often overlooked. Not only is it stratified horizontally into three compartments, the epidermis, the dermis and the sub cutis, but it is also perforated vertically by a variety of appendages which produce vastly different products such as sebum, hair, and eccrine and apocrine sweat. The epidermis alone is a mixture of several discrete cell types such as the keratinocyte which produces the cornified horny layer, the melanocyte which makes melanin and the Langerhans cell which serves in peripheral immune surveillance.

Inevitably, all these structures undergo regressive structural changes with advancing age. Although it is important to describe these involutions, anatomy itself provides little insight into those problems which most concern gerontologists and geriatricians. Our major thrust will be towards understanding how structural decadence affects functional attributes, and the extent to which the ensuing physiological decline predisposes to disease. We will not dwell on the clinical consequences, as these will be the centre of attention in other chapters of this volume. We also wish to refer the reader to the excellent monograph by Gilchrest (1984), entitled *Skin and Aging Processes*, which also deals with cutaneous gerontology and geriatrics.

With this is mind, we will not only review some of the age-associated changes which undeniably occur in various skin structures, but we will also focus upon the equally real, but as yet largely undefined, functional decrements. Since substantial knowledge is very limited, many of the statements proffered will be derived from our own currently ongoing experimental studies of cutaneous aging in normal, non-institutionalised volunteers (Grove et al, 1981).

The epidermis

Stratum corneum

The stratum corneum is the outermost region of the skin which provides a tough, flexible barrier that prevents excessive water loss and also provides protection from various environmental insults. It consists of layers of dead, cornified cells (corneocytes) with remarkably rugged cell envelopes. Neither

the number of cell layers nor the thickness of the stratum corneum show any significant changes with advancing age (Holbrook & Odland, 1974; Lavker, 1979; Grove & Kligman, 1983).

On the other hand, the corneocytes which comprise the horny cell layers of the stratum corneum are known to change with advancing age. Since these cells are continually being shed from the skin surface, it becomes a simple matter to collect them for subsequent analysis in a variety of ways. What is so exciting about these exfoliative procedures is that a great deal of information can be obtained without harm or inconvenience to the volunteer. By utilising such a non-intrusive approach, several different groups have demonstrated a decrease in the rate of desquamation (Leyden et al, 1978; Roberts & Marks, 1980) and an increase in cell size and heterogeneity (Plewig, 1970; Grove, 1979a; Corcuff, de Rigal & Leveque, 1982; Herrmann, Scheuber & Plewig, 1983) with advancing age. Such changes are thought to be an expression of the decreased proliferative activity of the underlying viable epidermis.

It has also been observed that in sun-exposed areas, the cells become more pleomorphic due to such anomalies as the retention of nuclear remnants, loss of lines of overlap, roughening of the border edges, and changes in tintorial properties (Grove, 1979a). Although the exact clinical significance of these abnormal cell types is currently unknown, there is good reason to believe that they will be of diagnostic importance. Indeed, these specimens are akin to those being used in Papanicdaou (Pap) smear cytodiagnosis and it is anticipated that exfoliative cytology of aged human skin will be equally rewarding.

Viable epidermis

It is frequently stated that the epidermis thins and becomes atrophic in old age (Selmanowitz, Rizer & Orentreich, 1977). This is one of those generalisations that is all too easy to accept in view of clinical appearances, and which, under closer scrutiny turns out to be false. For example, Evans, Cowdry & Wielson, (1943) showed that epidermal thickness in routine histological sections was appreciably less in the aged. Still, they concluded that there was no age-associated change in epidermal thickness. They explained this paradox by showing that excised aged skin shrank slightly or not at all, while specimens from young adults were reduced in area by as much as 50%. In young specimens, the dermis contracts sharply after excision, causing the epidermal cells to crowd together. Whitton & Everall (1973) thoroughly appreciated this shrinkage artefact and avoided it by separating the excised epidermis in sodium bromide and then stretching it on a template to match its original area. They, too, felt that there was no age-associated change in epidermal thickness. Freeman (1971) also found that there was no significant change in epidermal thickness of unexposed skin of the buttocks in individuals ranging from ages 25 to 76 years.

Still, a cursory glance of histologic sections suggests that the area occupied by the viable epidermis diminishes with age. The reason for this is that the dermal papillae and their corresponding rete pegs disappear and the

epidermis flattens. Thus, there are fewer mitotically active basal cells per unit of surface area, which could possibly diminish the production of keratinising cells. Another possible consequence of this flattening is that the epidermal-dermal junction is more susceptible to shear forces, which may explain the age-associated differences in the onset of blister formation under suction, as observed by Comaish & McVittie (1973). The reduplication of the lamina densa anchoring fibril complex, first observed by Lavker (1979) and recently confirmed by Hull & Warfel (1983), is thought to represent an attempt to form a better bond at the epidermal–dermal junction to compensate for the reduced area available for interdigitation due to this flattening.

Although the electron microscope fails to reveal any major differences in fine structure between young and old subjects (Mitchell, 1969; Lavker, 1979), light microscopy has shown that even in areas not exposed to the sun there is an age-associated increase in disparity in size, shape and staining qualities of the keratinocytes (Montagna Formisano & Kligman 1964; Montagna, 1965; Montagna and Carlisle, 1979; Stableford, 1981). The orderly sequence from cuboidal basal cells to spheroid Malpighian cells and onto flattened granular cells is disrupted and polarity is lost in the elderly epidermis. No age-associated changes were observed in the activities of 14 enzymes, representative of the major metabolic pathways, which were found in the epidermis from the lower part of the abdomen (Yamasawa, Cerimele & Serri, 1972).

Changes in the other epidermal cell types have also been reported. The number of dopa-reactive epidermal melanocytes has been reported to decrease by roughly 10% of the surviving population each decade, in both covered and sun-exposed areas (Snell & Bischitz, 1963; Fitzpatrick, Szabo & Mitchell, 1965; Quevedo, Szabo & Virks, 1969; Gilchrest, Blog & Szabo, 1979). It is also known that although ultra-violet light can augment the number of dopa-positive cells, the number of melanocytes activated in this way decreases with age, suggesting a diminished reserve capacity. In spite of this overall loss of active melanocytes, the chronically sun-exposed areas of elderly individuals are often characterised by generalised hyperpigmentation interspersed with areas of hypopigmentation (Walsh, 1964; Papa, Carter & Kligman, 1970). Recently, Gilchrest, Blog & Szabo (1979) explained this paradox in terms of a greatest dopapositivity of the individual chronically irradiated melanocytes. That the melanocytes remaining in aged skin may be functionally hypertrophied was also suggested by the observation that they tend to be larger and more dendritic (Walsh, 1964; Fitzpatrick, Szabo & Mitchell et al, 1965). However, smaller and less dendritic melanocytes have also been reported to be characteristic of aged skin (Snell and Bischitz, 1963), while others have seen no discernable changes in size or dendriticity of this cell type (Gilchrest, Blog & Szabo, 1979).

Very little is known about age-associated changes in Langerhans cells except that Gilchrest, Murphy & Scotter (1982) have shown that the population density of these cells may be decreased by more than 50% in elderly subjects. Thiers et al (1984) have also shown that the number of Langerhans