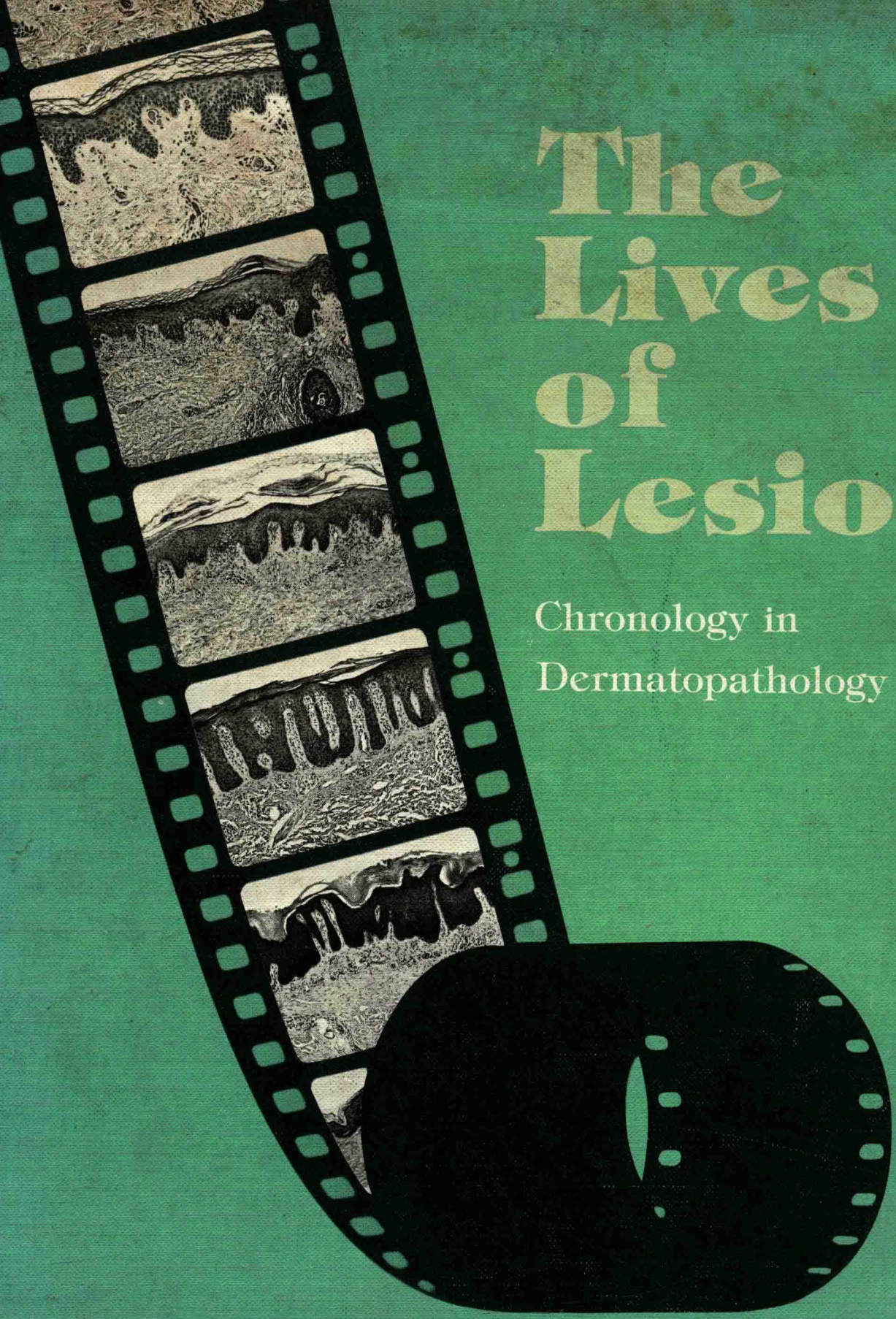

The Lives of Lesions

Chronology in Dermatopathology

A. Bernard Ackerman
Anna Ragaz

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CHRONOLOGY IN DERMATOPATHOLOGY

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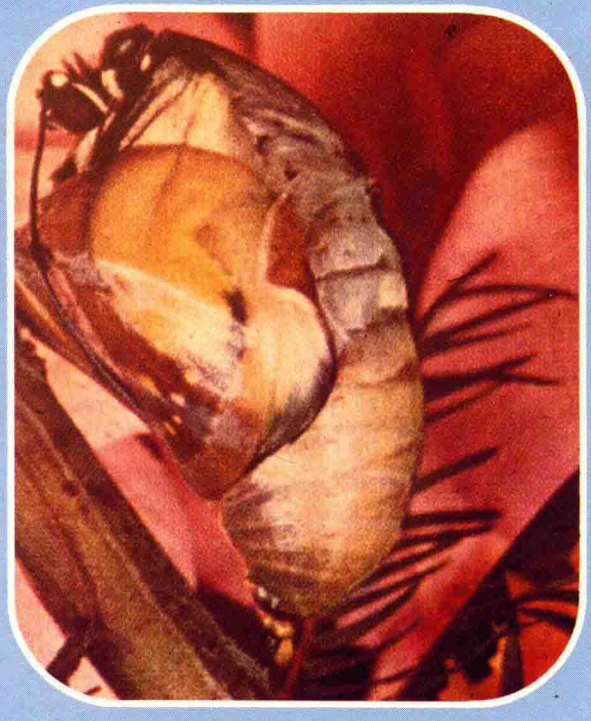

The Lives

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# of Lesions

Chronology in Dermatopathology

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Masson Publishing USA, Inc • New York • Paris • Berlin • Milan • Barcelona • Mexico City • Rio de Janeiro

for

ARKADI M. RYWLIN

a mentor in the science of pathology and in the art of living and a wise, generous, devoted friend

Preface

"For what a man adds up to must develop in stages, but no stage explains the man."

ERIK H. ERIKSON

ALMOST every textbook of pathology, including those on dermatopathology, pictures the histopathology of diseases at but a moment in their courses, as if they were static phenomena rather than dynamic processes. Sometimes for some conditions, there is no photomicrograph at all, and in many instances of other conditions, photomicrographs of merely single biopsy specimens are displayed as if they were representative of entire disease processes. Consequently, the student of pathology starts out with a warped view of pathologic progression; he gets an impression of histopathology as a "snap-shot", rather than as a slow-motion "moving picture".

This book came into being in an attempt to encourage students of general pathology and of special pathology of every organ to conceive of pathologic processes in time lapse, as indeed those processes play themselves out in actuality. This notion first occurred to me as I was preparing a book, now long in print, on "Histologic Diagnosis of Inflammatory Skin Diseases: A Method by Pattern Analysis." There I developed a theme that a disease like psoriasis, for example, could not be classified morphologically as simply a psoriasiform dermatitis, but must also be shown as a spongiotic dermatitis, a spongiform pustular dermatitis, and a subcorneal or intracorneal pustular dermatitis, each depending upon what stage in the life history of a psoriatic lesion was interrupted when the biopsy specimen was taken. The idea evolved that lesions have lives, just as human beings have; that lesions look very different at different times in their lives just as human beings do; and that stages in the lives of lesions can be described and depicted roughly as early, fully developed, and late, just as human beings can be described and depicted as infantile, mature, and old. This concept was explored in a symposium at New York University School of Medicine in October, 1979, under the title: "The Lives of Lesions: Chronology in Dermatopathology". The principal faculty for that symposium consisted of Dr. Anna Ragaz,

Preface

my collaborator in this work; Dr. Arkadi Rywlin, my mentor and friend; and me. That symposium was so well received and was so instructive to us who conducted it that Dr. Ragaz and I decided to offer its substance to a wider audience in the form of a book.

So here now is "Lives of Lesions: Chronology in Dermatopathology" in which chronological sequence in the pathology of 25 important cutaneous diseases are described and depicted. Like the lives of living things, the durations of some lesions are short (e.g., those of urticaria and erythema multiforme), whereas others are long (e.g., mycosis fungoides and scleroderma), but every life, brief or long, is better understood when viewed over its entire time rather than at points of time.

It is a pleasure to acknowledge with thanks the following persons and indispensable services they rendered to us: William H. Atkinson for superb photomicrography; Howard N. King for nonpareil book design; Hans Kresbach, M.D. and Helmut Kerl, M.D. for some clinical photographs from the Department of Dermatology of the University of Graz; Daniel Baker, M.D. and Joseph Scherzer, M.D., for use of a clinical photograph; William Slue and Meryll Krolick of the Department of Dermatology at New York University School of Medicine for sharp clinical color photographs made all the sharper for color corrections by Atkinson;

To Morris Leider, M.D. for scrupulous editing; Ellen P. Walroth for effectively coordinating the editing; Diane Pitre for faultless typing; former residents and Fellows in the Section of Dermatopathology of The New York University School of Medicine who, hailing from many lands, stimulated us by their intellectual fervor, probing questions, and constructive criticisms; and the New York Public Library for the color photographs used on the end papers of the book.

Photomicrographs of the following conditions are reproduced here from The American Journal of Dermatopathology by permission of Masson Publishing USA, Inc.: Extramammary Paget's disease, 1:101–132, 1979. Psoriasis, 1:199–214, 1979. Lichen planus, 3:5–25, 1981.

A. Bernard Ackerman
New York City

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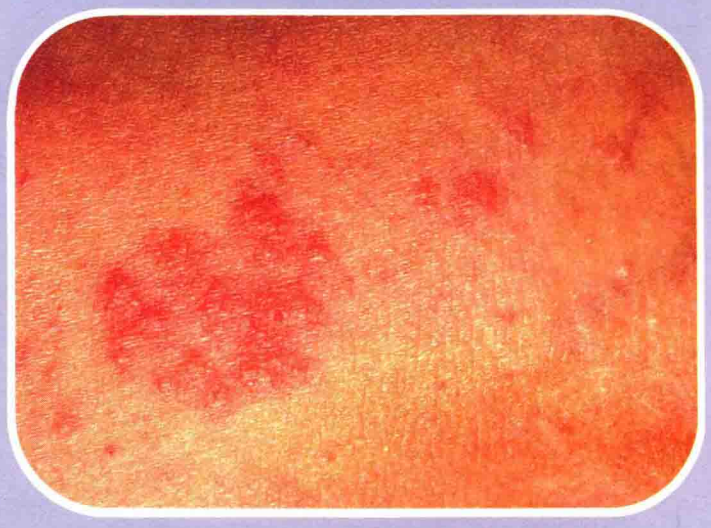
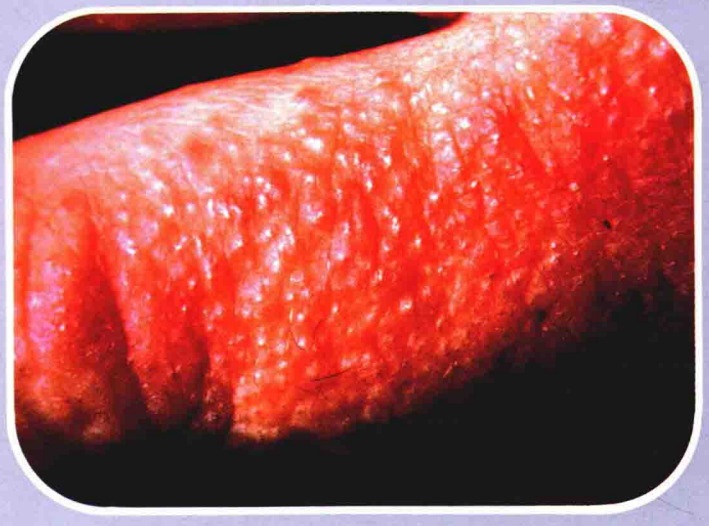
Papules and papulo-vesicles of early contact dermatitis





Tense vesicles of fully developed contact dermatitis

Lichenification in late contact dermatitis

1

Allergic Contact and Nummular Dermatitis

Allergic contact and nummular dermatitis, like other spongiotic dermatitides from which they are indistinguishable such as dyshidrotic dermatitis and "id" reactions, proceed through an early "spongiotic" stage to a fully-developed "spongiotic psoriasiform" stage and to a late "psoriasiform" stage before resolution. Clinically, lesions of contact dermatitis and nummular dermatitis begin as reddish macules or patches that progress rapidly into papules or plaques and then into tense vesicles or bullae. Spongiotic vesicles may become so large and tense that they "explode," causing the initially intra-epidermal blisters to appear to have been subepidermal all along. The lesions then regress, less rapidly, to crusted papules or plaques and finally disappear leaving hyper- or hypopigmented macules or patches in their wake. Like all inflammatory processes in the skin, contact and nummular dermatitis may quickly regress, sometimes spontaneously and frequently under treatment, without chronicity being established. Because both contact and nummular dermatitis are persistently pruritic processes, the lesions are commonly scratched or rubbed. The trauma of scratching may promote impetiginization marked by purulence, crusts, erosions, and ulcerations. Longstanding vigorous rubbing may induce lichenification. When scratching and rubbing are complicating, the underlying process may be obscured histologically. One clue to the true nature of contact or nummular dermatitis may then sometimes be the presence of focal spongiosis within a lesion that otherwise shows evidences of erosions, ulcerations, or lichen simplex chronicus. Impetiginization secondary to scratching may cause intra-epidermal spongiotic vesicles to become vesiculopustules.

Early lesions (papules)

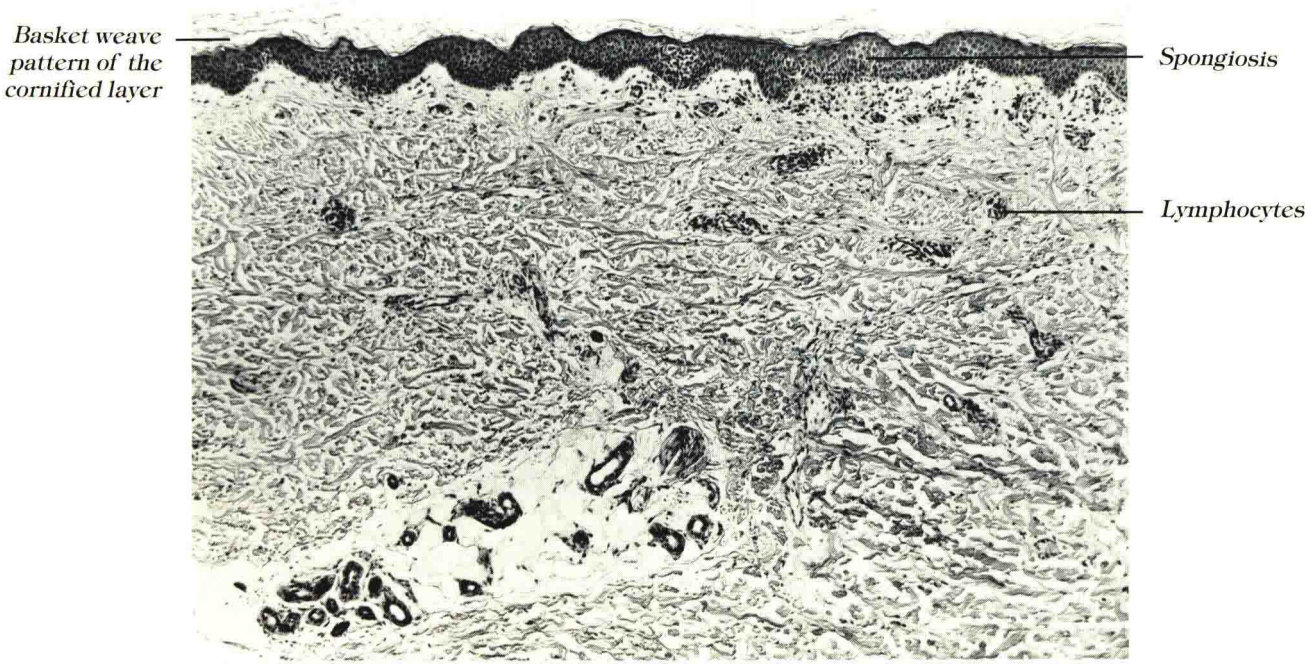
1. Cornified layer normal, i.e., in basket-weave configuration except on palms and soles where it is compact
2. Spongiotic foci in the lower portion of the epidermis; lymphocytes and occasionally eosinophils in those foci
3. Edema of the papillary dermis
4. Extravasated erythrocytes in variable numbers in the upper part of the dermis
5. Superficial perivascular infiltrate of lymphocytes, histiocytes, and often eosinophils

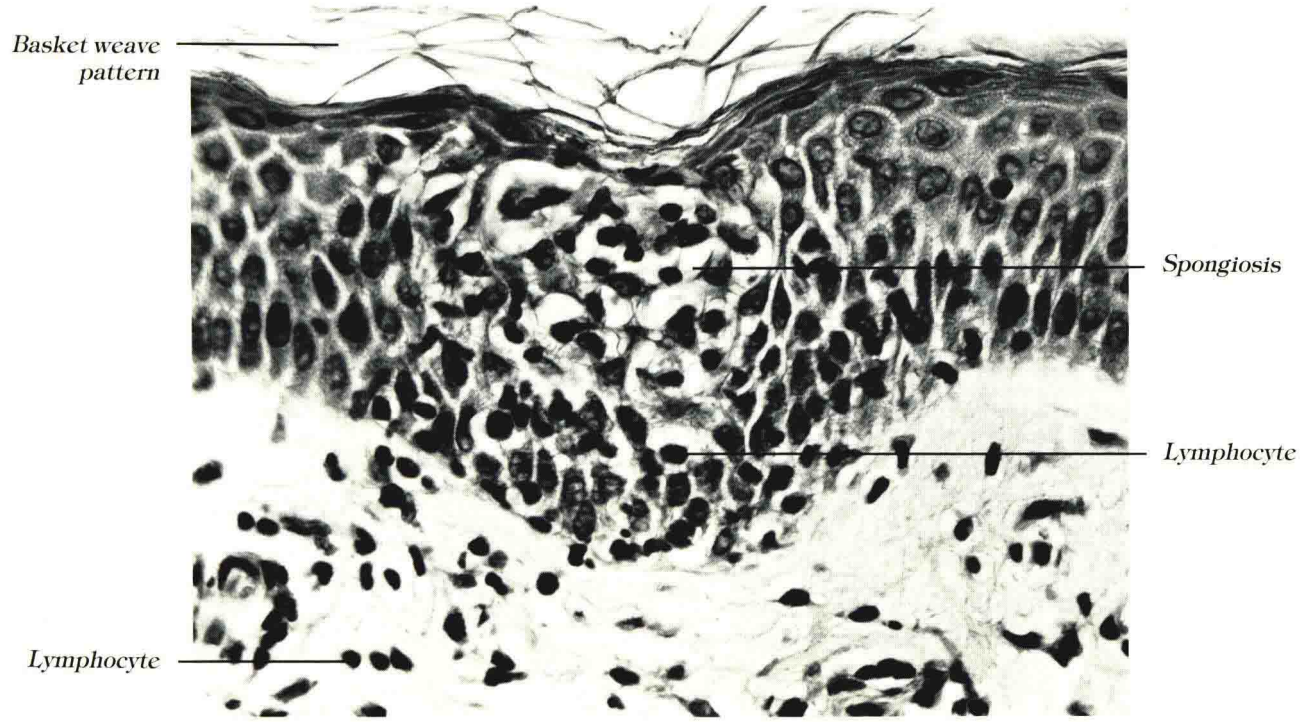
Fully developed lesions (vesicles and bullae)

1. Scales and crusts atop some vesicular or bullous lesions
2. Spongiotic microvesiculation and intracellular edema (ballooning)
3. Intra-epidermal vesiculation secondary to spongiosis and sometimes subepidermal vesiculation secondary to massive edema of the papillary dermis
4. Psoriasiform hyperplasia
5. Edema of the papillary dermis
6. Extravasated erythrocytes in the upper part of the dermis
7. Superficial perivascular infiltrate of lymphocytes, histiocytes, and eosinophils

Late lesions (psoriasiform plaques)

1. A picture of lichen simplex chronicus, namely, compact orthokeratosis, hypergranulosis, psoriasiform hyperplasia, and a papillary dermis thickened by coarse collagen fibers arranged in vertical streaks
2. Little, if any, spongiosis
3. Superficial perivascular lymphohistiocytic infiltrate
4. No edema of the papillary dermis
5. No extravasated erythrocytes





FIGS. 1-1a,b: *An early lesion of allergic contact dermatitis.* There is but a sparse infiltrate composed mostly of lymphocytes around vessels of the superficial plexus and there are several foci of spongiosis in the epidermis. Note the normal basket-weave appearance of the cornified layer, a sign that the process is still early. Histologic changes just like those shown here may be seen in early evolving lesions of nummular dermatitis.