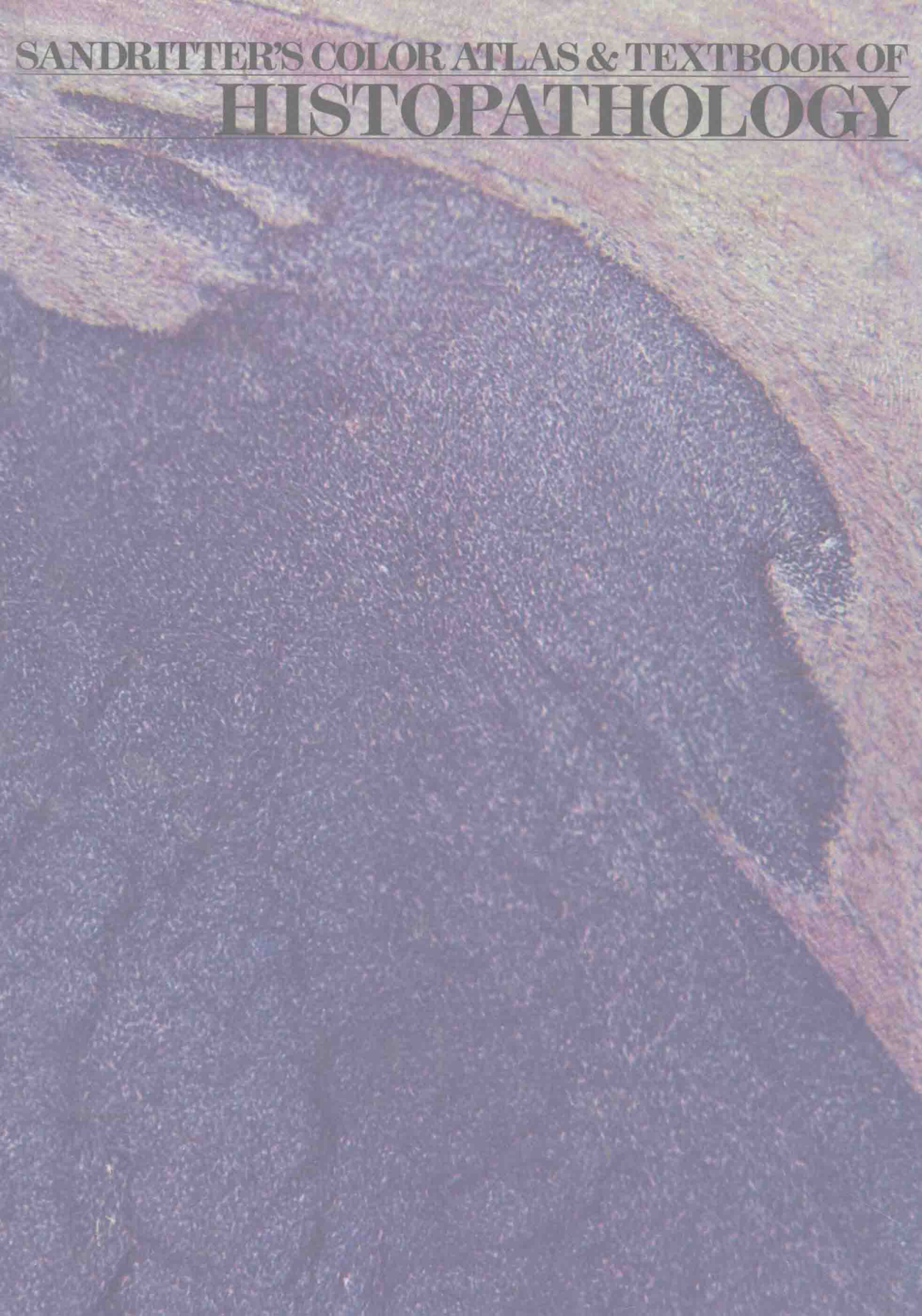
# SANDRITTER'S COLOR ATLAS & TEXTBOOK OF HISTOPATHOLOGY



# Sandritter's
# Color Atlas and Textbook of
# HISTOPATHOLOGY

## Seventh English Edition

Carlos Thomas, Dr. med.
Professor of Pathology and Director of
the Center for Pathology, University Marburg

*With the assistance of*

C. P. Adler, N. Bohm, N. Freudenberg, M. Hagedorn,
Ch. Mittermayer, U. N. Riede, R. Rohrbach and K. Salfelder

*Translated and edited by*

GOETZ W. RICHTER, M.D.
Professor of Pathology
University of Rochester

*Previous English editions by*

William B. Wartman, M.D.
Formerly Professor of Pathology
Northwestern University and
University of Virginia

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# SANDRITTER'S COLOR ATLAS AND TEXTBOOK OF HISTOPATHOLOGY

Seventh English Edition

*To the memory of*

WALTER SANDRITTER
1920–1980
Professor and Director of the Institute of
Pathology, University of Freiburg in Breisgau
1967–1980

# From the Preface to the Ninth German Edition

THE STUDY OF DISEASE includes general, special, and experimental pathology. A subdivision into histopathology and macropathology is undoubtedly a compromise, justified only because of the existence of courses in general and special pathology that include gross and microscopic pathology. In this sense the organization of the two books, *Histopathology* and *Macropathology,* has proved itself. This preface is to serve for both volumes in order to emphasize that they are complementary and are meant to form a single unit.

Instruction in pathology is central to human medicine. The sense of responsibility toward medical students and young physicians, who are relying on the experience of others, must be appropriate. The rapid increase of knowledge confronting us daily imposes excessive demands, not only on students, but also on their teachers. For this reason we need to bear in mind a proven experience: to transmit that factual knowledge which is likely to be valid 5 years hence. In each new edition the author should see to it that the book does not become too big. This was the goal in revising the various chapters (e.g., those on the heart, lung, liver, and kidney). At the same time, new methods of examination, such as immunohistochemistry, and also the international guidelines for nomenclature, classification, and diagnosis of diseases—especially of tumors—were kept in mind. The macroscopic or microscopic picture should, as far as possible, contain the necessary information and be supplemented by a brief text. But in this respect an atlas has its limits, and neither *Histopathology* nor *Macropathology* can replace conventional textbooks of pathology. It is in those books that the reader must look for references to the relevant literature. In this edition we have omitted references, which in the past were mainly in German. The two atlases are meant not only to prepare students for examinations, but, preferably, to help provide practical training for students and young physicians.

Books like these can originate and prove themselves only through the joint efforts of a sizable group. It is a pleasant duty of the principal author to thank all collaborators, both in his own group and at Schattauer Verlag. I owe special thanks to Prof. Dr. h. c. P. Matis, Mr. H. Schwer, Managing Director, Mr. Bergemann (Druckerei Mayr), and Mr. Haub (Grafische Kunstanstalt Brend'amour). The drawings were made by Mr. Tschorner. Finally, I ask readers to remember the name of Walter Sandritter, who created both *Histopathology* and *Macropathology* (first published, respectively, in 1965 and 1970). My task is to develop his concepts further.

CARLOS THOMAS
MARBURG, GERMANY

# Preface to the Seventh English Edition

SHORTLY BEFORE HIS SUDDEN and untimely death in 1980, Professor Walter Sandritter suggested that I take over the job of preparing a new English edition of *Histopathologie* on the retirement of Professor William Wartman, the translator and editor of all previous English editions. With the agreement and at the urging of Year Book Medical Publishers, I accepted this challenge.

The seventh English edition is based on the ninth edition of the German text, prepared under the leadership of Professor Carlos Thomas, Director of the Medical Center for Pathology at the University of Marburg, and published in 1983 by F. K. Schattauer Verlag, Stuttgart. Professor Thomas also was co-author of previous German editions.

*Color Atlas and Textbook of Histopathology* has, in earlier editions, found favor with medical students and aspiring pathologists throughout the world. It has been translated into Spanish, French, Italian, and Japanese as well as into English. Undoubtedly, its success is in considerable measure due to the excellence and relevance of the illustrations, but the simple, direct manner in which the authors have described and explained histopathologic and cellular alterations associated with diseases is also impressive.

This new English edition reflects many changes in the German text. The chapter on diseases of bones and joints has been entirely rewritten. Illustrations and descriptions of neoplasms have been integrated with both General and Systemic Pathology rather than presented in a separate chapter toward the end of the book, as was done before. Other changes are scattered throughout. Many brief references to macroscopic alterations have been retained. However, in the absence of an updated English version of Sandritter's and Thomas' *Makropathologie,* no further attempts at correlation were made in this English edition.

I have revised and adjusted the nomenclature in keeping with current Anglo-American usage but have not made a fetish of such changes. The authors used mainly World Health Organization nomenclature, and this has been retained where doing so seemed advantageous. Drastic changes, such as adoption of SNOMED, would have disturbed the authors' purposes and reduced the didactic value of the book.

My functions as translator and editor have precluded insertion of original material other than a few clarifying statements or revisions. Wherever possible, I have retained the previous translator's text.

The bibliography was dropped in the latest German edition. Accordingly, I have not attempted to provide what could at best only be a highly eclectic list of articles. It seems to me that students should first consult textbooks in which diseases are described more extensively.

Like its predecessors, this new version of "Sandritter-Thomas" bears the stamp of the late Professor Sandritter's highly practical educational and professional ideas, which have now been carried forward by Professor Thomas. May the book be useful and helpful to its readers!

G. W. RICHTER
ROCHESTER, NEW YORK

# Contents

# Introduction—General Pathology

A certain amount of practical knowledge and skill, particularly with respect to use of the light microscope, is desirable on the part of the reader if he is to get the most good from reading a textbook of histopathology. Profitable use of the microscope requires knowledge of its construction and of the interrelations of its individual parts. Furthermore, it is only possible to interpret a histologic slide after one is informed as to how the tissue has been prepared for cutting and how it has been stained. A solid foundation in normal histology and general pathology goes without saying, for the principles of general pathology are used constantly in special pathology.

## Preliminary Technical Remarks

### Use of the Microscope

The light source, the lens system with its diaphragms, and the eye must all be correctly aligned with one another in order to obtain optimal information from a histologic section. Artificial light, which consists predominantly of yellowish red light rays, can be corrected by a blue filter so that it will approximate daylight. Köhler's principle is commonly used to adjust the light source, since by using this principle it is possible to illuminate only the object area that is to be examined and that entirely uniformly. With a microscope with a built-in light source, swing in the front lens of the condenser. Focus the microscope on the specimen and stop down the *field diaphragm*. Rack up the condenser as far as possible and then lower it slowly, thus focusing the field diaphragm within the specimen area.

Center the condenser with the two centering screws if necessary (the condensers of many student microscopes are permanently centered so that this step may be omitted). Open the field diaphragm until its shadow disappears from the field of view. The field diaphragm should always be adjusted so that its image just disappears behind the edge of the eyepiece stop. Adjust image contrast and, if necessary, sharpness—but not image brightness—with the *condenser (aperture) diaphragm* by opening it entirely and then closing it down just far enough to remove glare from the specimen. Unstained objects can be seen best when the condenser diaphragm is closed as far as possible or with a phase contrast microscope. With blurred images, reducing the condenser diaphragm will increase the contrast.

The microscopic image is produced by diffraction of the light by the structures in the histologic preparation in the focal plane at the back of the objective (primary image). The secondary image, which is the one observed in the ocular, arises from magnification of the primary image.

Objective and ocular must be properly matched. In usual histologic practice, an ocular with a 10× magnification is used with the following objectives, in which the first number gives the magnification and the second the numerical aperture of the objective, which is a measure of its resolving power:

1. *Scanning lens:* objective 2.5/0.08—magnification 25×.
2. *Low magnification:* objective 10/0.25—magnification 100×.
3. *High dry magnification:* objective 40/0.65—magnification 400×.

For still higher magnification, especially for examination of smears of cells (blood, lymph node), oil immersion objectives (100/1.25) are available with a magnification of 1,000× or 1,250× (with 12.5× ocular).

*When using the microscope, the following suggestions will prove helpful.* With a monocular microscope, always keep both eyes open, since the adjustment for distance obtained in this way prevents rapid eye fatigue due to constant accommodation.

The lowest magnification should always be used before going to the other objectives because it is easier to orient the various structures under low magnification.

If the image is blurred, you should think of the possibility of the slide being upside down with the cover-slip resting on the stage of the microscope.

**Preparation and Staining of Histologic Sections**

*Sections* are prepared from blocks of tissue measuring about 2 × 2 cm. The selected tissue is usually hardened and fixed in Formol (ordinary 40% commercial Formalin diluted with water 1:9 so that the resulting solution is about 4%). The *hardening* results from coagulation and denaturation of protein, while the *fixation* arrests autolysis and bacterial decomposition. In order to prepare sections 5–10 μ thick, the tissue must have a consistency suitable for cutting. To obtain this, the tissue may either be frozen (at −20° C) with carbon dioxide snow and cut on the frozen-section microtome (this method is used particularly for demonstrating fat or for rapid diagnosis of biopsy specimens at the time of surgery) or the tissue can be processed through a series of alcohols (from 70% to 100%), methyl-benzoate, and benzol into paraffin with a melting point of 56° C. Liquid paraffin at 60° C penetrates the finest tissue spaces and produces a good cutting consistency. After cutting on a microtome, the sections are mounted on microscopic slides and stained, after first being deparaffinized with xylol.

*Note:* Frozen sections permit demonstration of neutral fat. In paraffin sections the fat is dissolved by alcohol and the droplets of fat appear as optically empty spaces in the tissue.

The methods used for *histologic staining* have been developed empirically and the physicochemical basis for them is not exactly known except in a few cases. Electrostatic binding, among other factors, plays a principal role. Negatively charged groups, for example, nucleic acids (phosphate groups) or proteins (−COOH groups) or the mucopolysaccharides (−COOH, $SO_4$), bind with the basic dye groups, which behave as cations. Acid dyes (e.g., eosin) with electronegative charges bind predominantly with positively charged protein groups ($NH_2$ groups). Excess and easily soluble dye in the tissue is removed after staining by differentiation in water, alcohol, or weak acid. Finally, the water is removed with 70% and 96% alcohol, the section immersed in a clearing agent (xylol), mounted in Permount or Canada balsam, and covered with a cover-slip.

*Histochemistry* deals with specific and sometimes quantitative identification of chemical substances in tissues, such as nucleic acids, certain proteins, carbohydrates, enzymes, etc.

*Artifacts* in histologic sections are caused chiefly by improper fixation, embedding (cracks or tears), or staining (transparent, unstained flaws, or dark spots).

Table 1 reviews the features of some commonly used stains. *Fluorescence microscopy,* in which tissues are stained with fluorescing dyes and examined under ultraviolet light, allows detection of dyes in low concentrations because the ultraviolet light rays (e.g., 350 nm) liberate secondary rays in the visible range. Some substances show autofluorescence, e.g., lipids, porphyrins, and elastic fibers.

TABLE 1.—STAINING METHODS

| METHOD | RESULTS | | REMARKS |
|---|---|---|---|
| **Hematoxylin-eosin** | **Blue**<br>*Hematoxylin*<br>Basophilic cytoplasm, nuclei, bacteria, calcium | **Red**<br>*Eosin*<br>Cytoplasm, connective, and all other tissues | Figs 1–15, 1–16 |
| **van Gieson's** | **Yellow**<br>*Picric Acid*<br>Cytoplasm, muscle, amyloid, fibrin, fibrinoid | **Red**<br>*Fuchsin*<br>Connective tissue, hyalin | **Black**<br>*Iron Hematoxylin*<br>Nuclei<br>Fig 1–6 |
| **Elastica stain** | **Black**<br>*Resorcin-fuchsin*<br>Elastic fibers | **Red**<br>*Nuclear fast red*<br>Nuclei | Fig 2–14 |
| **Elastica-van Gieson's** | Used in combination | | Fig 2–7 |
| **Azan** | **Red**<br>*Azocarmine*<br>Nuclei, erythrocytes, fibrin, fibrinoid, acidophilic cytoplasm, epithelial hyalin | **Blue**<br>*Aniline blue, Orange G*<br>Collagen fibers, basophilic cytoplasm, mucus | Fig 2–10 |
| **Silver stain** | **Black**<br>*Ammoniacal $AgNO_3$*<br>Reticulum fibers, nerve fibers | | Collagen fibers brown |
| **Fat stain** | **Red**<br>*Sudan III, Scarlet Red*<br>Neutral fat | **Blue**<br>*Hematoxylin*<br>Nuclei, cytoplasm | Fig 2–6 |
| **Congo red** | **Red**<br>*Congo red*<br>Amyloid | **Blue**<br>*Hematoxylin*<br>Nuclei | Fig 6–7 |
| **Weigert's fibrin stain** | **Blue**<br>*Lugol's solution, Crystal violet*<br>Fibrin, bacteria | **Red**<br>*Nuclear fast red*<br>Nuclei | Not specific for fibrin<br>Fig 3–29 |
| **Prussian blue reaction** | **Blue**<br>*Calcium ferrocyanide*<br>Hemosiderin $Fe^{III}$ | **Red**<br>*Nuclear fast red*<br>Nuclei | Fig 3–9 |
| **Giemsa (May-Grünwald-Giemsa)** | **Blue**<br>*Methyl violet*<br><br>Nuclei, all basophilic substances | **Red**<br>*Azur-eosin*<br><br>Eosinophils, cytoplasm and its granules, collagen fibers | Metachromatic: mast cells violet melanin green<br>Fig 16–2 |
| **Ladewig** | **Blue—gray-blue**<br>*Aniline blue*<br><br>Parenchyma<br>Mesenchyma | **Orange red**<br>*Acid fuchsin-gold-orange*<br>Muscle<br>Fibrin | **Black**<br>*Iron hematoxylin*<br><br>Nuclei |
| **Mason-Goldner** | **Orange-red**<br>*Azofuchsin*<br>Parenchyma<br>Fibrin | **Green**<br>*Light green*<br>Mesenchyma | **Black**<br>*Iron hematoxylin*<br>Nuclei<br>Fig 6–9 |

TABLE 1.—STAINING METHODS (continued)

| METHOD | RESULTS | | REMARKS |
|---|---|---|---|
| **Spielmeyer's myelin stain** | **Blue-black**<br>*Iron-alum hematoxylin*<br>Myelin, erythrocytes | | Figs 14–14, 14–17 |
| **Ziehl-Neelsen** | **Red**<br>*Carbofuchsin*<br>Acid-fast rods, Tb bacilli, lepra bacilli | **Blue**<br>*Hemalum*<br>Nuclei | |
| **Periodic acid-Schiff reaction (PAS)** | **Red**<br>*Schiff reagent*<br>Adjacent hydroxyl groups and amino-alcohols | | Neutral and acid polysaccharides<br>Fig 4–28<br>Demonstration of fungi, parasites |
| **Levaditi** | **Black**<br>*$AgNO_3$-reduced Pyrogallic acid*<br>Spirocheta pallida<br>Listerella monocytogenes | | Fig 15–3<br><br><br>Fig 5–16 |
| **Thionine, toluidine blue** | **Blue**<br>Basophilic cytoplasm | **Blue**<br>Nuclei | Metachromasia (mucins, lipids) |
| **Staining of smears (Papanicolaou)** | **Blue-violet**<br>*Hematoxylin*<br>Nuclei, bacteria | **Orange-red**<br>*Orange-G*<br>Cellular glycogen, keratin<br>Blue-green/green/rose<br>*E.A. 36 dye mixture*<br>Cytoplasm of basophilic cells: (blue-green)<br>Cytoplasm of acidophil cells: (rose-colored)<br>Mucus: (green) | |

## Histologic Interpretation and Diagnosis

A famous physician (Franz Volhard) once said: *"The Gods have put diagnosis before therapy—man must put careful observation and interpretation before diagnosis." Analysis must precede synthesis* as it does in all other branches of knowledge. Analysis begins with the examination of the subject with a clear-cut objective in mind. *Careful observation* of similarities and dissimilarities, the separation of the typical and the atypical, the general from the special, all contribute to the desired knowledge of the subject. The arrangement, color, size, and form of the tissue elements and their relations to one another all help to determine the essential characteristics of the various structures under consideration. As such observations cannot be obtained without adequate preparation, a thorough theoretical grounding and a certain amount of experience become essential.

The beginning student will find it helpful in getting exact histologic details either to make drawings or to set down his observations in abbreviated outline form. The student is thus forced to emphasize the essential features and to deemphasize unessential ones.

*After first carefully making the necessary observations, it is then possible to take the second step, that is, to synthesize the observations and make a diagnosis.* On the other hand, hasty, careless examination will often lead to an incorrect opinion. In order to arrive at a *diagnosis,* the histologic observations need to be classified in some logical manner, usually one which has been reached through a compromise of experience and hypothesis. But, by its very nature, no diagnosis can be considered final, since it can change with the progress of scientific knowledge. Thus, it is understandable that an exact description retains its validity indefinitely, even when the interpretation and diagnosis of a section have already been revised.

The student will therefore be well advised to put his chief effort into a careful description of a microscopic section. In examinations, this is always graded higher than a diagnosis unsupported by accurate description.

In practice, the first step in examining a histologic preparation is to look at the section with the *unaided eye.* The shape and the various components of the tissue structures—easily recognized by differences in staining—often provide essential topographical information and have an important influence on the next step in the analysis of the section. *An inverted ocular used as a scanning lens* will provide an overall view of the tissue at very low magnification. Ordinary *low-power magnification* can then be used to examine in greater detail the structures already seen with the inverted ocular. In this way, a rough overall picture of the essential elements of the lesion is formed. Further details can also be distinguished with *low magnification,* such as the size and position of the nuclei and the structure of the cytoplasm. This magnification is probably the most useful of all, for at a magnification of about one hundred-fold, all the essential structures are well seen without losing the overall architectural relationships. A drawing at this stage of the examination will fix the typical findings firmly in mind. Practically all histologic preparations can be diagnosed with low magnification. *High magnification* is used only to clarify individual details, such as the shape and division of nuclear chromatin, mitoses, and so forth.

Such a methodical approach is an essential prerequisite for profitable observation and correct diagnosis. In studying histopathologic slides the student acquires the knowledge necessary to separate essential from nonessential observations—knowledge that a physician uses at the bedside.

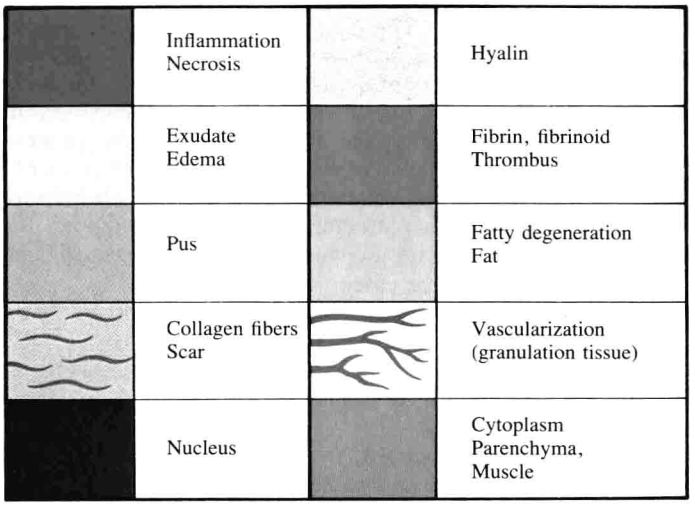


**Fig 1.**—Explanation of the colors and symbols used to depict lesion components and pathologic processes.

## Notes on General Pathology

These brief, almost stenographic, introductory remarks about general pathology are intended only as a means of making it easier to understand the complexities of special pathology. Reference to the appropriate illustrations of the book permits its use as a guide to the principles of general pathology.

In the schematic diagrams and tables the same symbols and colors are used for similar pathologic processes throughout the book (Fig 1). The individual components of a lesion or process are indicated by colors or symbols based, unless otherwise noted, on the gross (e.g., pus—greenish yellow) or microscopic appearance (e.g., collagen fibers—curly lines).

A knowledge of general pathology is an excellent foundation for the study of disease. The knowledge so obtained can be applied in nearly all special situations, since *the host in reacting to the many different pathologic stimuli that may affect it has only a limited number of possible responses available*. These originate essentially from either transient or permanent increase *(anabolism)* or decrease of metabolism *(catabolism)* or from *work failure*. In addition, complex tissue responses occur in *circulatory disturbances*, the various forms of *inflammation*, and in *tumors*.

In theory, pathologic stimuli can reach the cells and tissues in various ways (Fig 2):

(1) *directly* (e.g., trauma, radiant energy); (2) by way of the *bloodstream* or the *lymphatics* with resultant direct cell injury (e.g., toxins, alterations of the vascular contents as in thrombosis); (3) *indirectly*, when the stimulus acts on the *vessel walls*, a secondary circulatory disturbance then causing the cell injury (e.g., nervous derangement of permeability); (4) the pathologic stimulus can come from conduits such as the *alimentary tract*. Finally, primary (e.g., inborn) defects of metabolism may cause secondary cellular reactions.

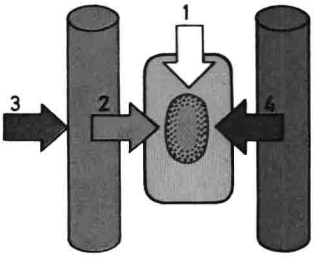


**Fig 2.**—See text for explanation.

The following diagram sets out the possible reactions of the organism to pathologic stimuli in simple fashion (Fig 3).

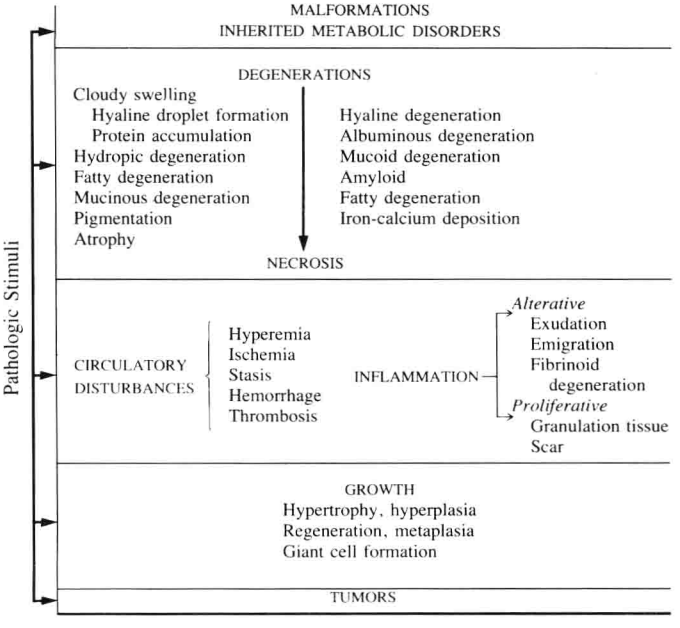


**Fig 3.**—Schematic survey of possible host reactions to pathologic stimuli.

### Malformations: Inherited Metabolic Disorders

*Malformations* or *metabolic disorders* can develop in the embryonal (up to the third month of pregnancy) and fetal periods (after the third month) either because of hereditary errors in the genetic material or of the action of pathologic stimuli (e.g., teratogens). These manifest themselves, for example, either in *agenesis* (absence of enzymes, e.g., galactosemia; defective organ formation) or *aplasia* or *hypoplasia* (faulty development of existing organs). A great number of different manifestations can be produced in this way.

### Degenerations

The different sorts of *degeneration* are morphological manifestations of metabolic disturbances either of cells (left-hand column of Fig 3) or of intercellular substances (right-hand column of Fig 3).

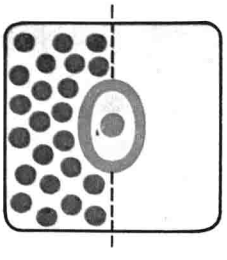

**Fig 4.**—Cloudy swelling *(left);* hydropic degeneration *(right).*

Cloudy swelling and hydropic degeneration (Fig 4) result from disturbance of the metabolic systems that maintain the ionic environment of cells (so-called ion pumps). When these regulatory mechanisms fail, then sodium and water flow into the cells and potassium leaves them. As a result, the mitochondria swell and the cytoplasm appears to be filled with fine "protein granules" *(cloudy swelling).* The resulting cloudiness is due to increased scattering of light *(Tyndall effect).* The mitochondria may also be transformed into water-filled vesicles *(hydropic transformation* of mitochondria). The water may accumulate in the cytosol or in the cisternae of the endoplasmic reticulum (hydropic degeneration). Compare Figs 6–1, 6–2 (light photomicrographs); Table 4, Fig 1–22 (electron micrographs).

Nuclei may also show swelling *(degenerative nuclear swelling).* This must be distinguished, however, from physiologic or *functional nuclear swelling,* which is often accompanied by enlargement of the nucleolus and is a reflection of increased metabolic activity.

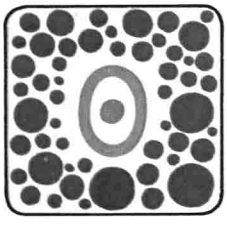

**Fig 5.**—Hyalin droplet regeneration (protein reabsorption).

Hyalin droplet degeneration (protein accumulation) should be distinguished from cloudy swelling (Fig 5). The microscopic appearances of the two conditions can be similar, but in hyalin droplet degeneration active work is performed by the cell (anabolism) with accumulation of protein in cytoplasmic organelles, for example, the reabsorption of protein in the renal tubules. Such reabsorption of material is accomplished by pinocytosis, in which small vesicles are formed by constriction of the cell membrane. Phagocytosis, on the other hand, is the process by which the cytoplasm takes up large, formed materials, such as bacteria (see Figs 10,A and 19 and 20).

*See also Figures 6–2 (light photomicrograph) and 6–12 (electron micrographs).*

The term *fatty degeneration* (perhaps better called fatty change or fatty metamorphosis) describes the appearance of microscopically visible fat, either in the form of fine (Fig 6, right) or large (Fig 6, left) droplets. The size of the droplets depends on the proportion of neutral fat to phospholipid (large droplets contain little phospholipid). Normally, fat is taken up in the form of fatty acids, which is accomplished through pinocytosis. The fatty acids are synthesized to triglycerides, bound to phospholipids and proteins, and delivered to the blood as lipoproteins. Any disproportion between the amounts of triglycerides (e.g., increased alimentary supply) and protein or of phospholipids (e.g., choline deficiency) or failure of energy coupling (oxygen or enzyme deficiencies) leads to accumulation of fat, i.e., to fatty degeneration.

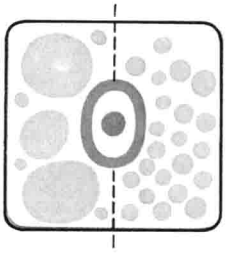

**Fig. 6.**—Fatty degeneration: *left*, large droplet form; *right*, fine droplet form.

*See Figure 1–8 (light photomicrograph) and Figure 5–48 (electron micrographs).*

*Fat phanerosis* may develop in *necrobiosis* (perceptible, slow cell death) in which structurally intact fat tissue is broken down into microscopically visible droplets.

In disordered carbohydrate metabolism, glycogen droplets may appear (e.g., in the kidney in diabetes), or mucinous degeneration may develop (production of mucopolysaccharides but without secretion) as, for example, in mucinous carcinoma (signet ring cells, Fig 7).

**Fig 7.**—Signet ring cell.

*See Fig 4–32*

Alteration of the character of mucin secretion may also result in obstruction of excretory ducts (e.g., cystic fibrosis of the pancreas).

*See Fig 4–51*

Glycogen storage disease is caused by an *inborn error* of carbohydrate metabolism.

Pigments are naturally colored materials that are laid down in either diffuse or granular fashion. They usually have as a chief component either *protein* (e.g., melanin), *lipid* (lipopigments, e.g., lipofuscin) or derivatives of *hemoglobin* (hemosiderin or siderin, hematoidin, bile pigment). In addition, a number of *exogenous pigments* may be seen.

*See the following:*

Lipofuscin Table 2, Fig 1–3
Melanin, Fig 9–24
Hemosiderin or siderin, Figs 3–9, 5–36
Hematoidin, Table 2, Fig 11–25
Bile pigment, Fig 5–4
Malaria pigment, Fig 5–3
Exogenous pigments, Table 2, Fig 11–27

Table 2 lists the most important differential characteristics of various pigments.

Atrophy of cells (Fig 8) results from inactivity or chronic malnutrition, is manifested by reduction in cell size *(simple atrophy)*, and eventually results in reduction of cell number *(numerical atrophy)*. Hypertrophy connotes enlargement of cells, with increased function.

Degeneration of the connective and supporting tissues affects chiefly the *ground substance*. It may result in unmasking of the fibers, infiltration of fat and mucin, and deposition of foreign substances.

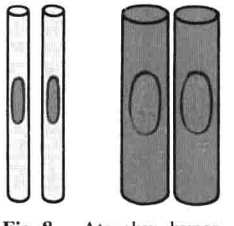

**Fig 8.**—Atrophy; hypertrophy.