Cell and Tissue Biology

A Textbook of Histology

Edited by

Leon Weiss, M.D.

Sixth Edition

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With 35 contributors

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Preface

The biomedical sciences have become important beyond precedent in understanding the nature of life and in the practice of medicine. Our fundamental knowledge, biotechnology, and relevance to the clinic allow us to deal effectively with scientific and medical matters that previously eluded us, and, rather surprisingly, with the profound mysteries of life that are traditionally the province of philosophers and other humanists. Understanding of the endocrine and immune systems is expanding, encompassing many tissues, the tight regulation of many processes by distant messengers (hormones), local paracrine factors (cytokines) and cell-to-cell associations (membranous junctional complexes). From studies of cell homing and the recognition of foreign antigens, the idea of *self* and *non-self* – where *I* stop and *they* begin – emerges, providing a profound and compelling view of the tissues of the body that verge into such philosophic and societal concerns as the limits of the individual and the dynamics of social behavior. The heart can no longer be regarded simply as a mechanical pump, but, in addition, it is viewed as a sensitive endocrine organ regulating fluid volume, blood pressure, and kidney function.

In a past so recent that its influence lingers, the applicability of basic medical research to the clinic appeared irrelevant, to the point that basic scientists and clinicians followed different modes of thought and action. In their pursuit of fundamental knowledge, basic scientists viewed clinicians as expedient and superficial and shuddered at the notion of clinical relevance. Clinicians viewed basic scientists as stiff-necked dwellers in ivory towers, unmoved by suffering, playing a glass-bead game devoid of practical value. As a result, many medical students graduated estranged from the basic medical sciences, and went out to practice in the community with inadequate knowledge of the sciences, unable and unwilling to champion the intellectual, investigative, and experimental life. As anti-intellectualism grows and assaults on animal experimentation multiply, the community physician, long a supporter of science, has in recent years, too often been uncertain of the value of basic research in medicine.

But at the very time that the basic biomedical sciences are contributing fundamental knowledge at an exponential rate, they are needed in the treatment of patients. Non-invasive techniques such as magnetic resonance are being applied to disease and treatment, as they garner new basic knowledge. Subtypes of lymphocytes are regularly defined in the management of leukemia, the results determining the type of treatment. The bank of reagent monoclonal antibodies is larger for human than for animal tissues, and is being used not only for diagnosis and treatment but to mark new cell types and systems. Gene therapy is on the threshold of the clinic. The old stand-off between clinician and basic scientist is disappearing. We are all in the same camp now. On moral, let alone practical grounds, scientists have become increasingly concerned with the sick and are impelled to make their discoveries applicable to disease. The process is humanizing and brings us into the company of caring physicians.

Molecular biology is a dominant discipline in this great age of biomedical research. Its focus has widened from the analysis of components of a cell and the reactions of single cells or cells of one type to the analysis of the interactions of diverse cells and the means by which cells communicate with one another. Regulation of the functions of a tissue, inherent in such interactions, is studied in cell and tissue biology. For example, while analysis of antibody secretion by plasma cells can be studied in isolated systems such as tissue culture, lymph nodes, bone marrow, thymus, spleen, related tissues and their interactions must also be studied, if we are to understand how the production of antibody is regulated and how this ties into inflammation, coagulation, and other systems affecting infectious disease. This tissue-derived knowledge is needed both in comprehending the immune response and in the effective management of disease.

Tissues, therefore, are hardly arbitrary assemblages of cells. A tissue, studied microscopically, is the morphological expression of the complex regulated functions that make up the body. Tissues are societies of cells, governed by rules, able to deal – at least to a degree – with cells that break those rules. Perhaps, because we are human, held on a short leash by the structure and functions of our brains, our perception of the nature and regulation of the society of cells that make up our body bears remarkable parallels with our perception of the nature and rules of the society we live in as individuals.

Morphology has never been more valuable in understanding physiology and pathology. But our presentation of morphology must include biochemistry, physiology, molecular biology, and pathological and clinical correlations to justify our claim on the time of today's student of biology and medicine. Years ago the morphological field of *Cytology* was integrated into other basic medical sciences and evolved into *Cell Biology*. *Histology* has assimilated cell and molecular biology, physiology, biochemistry, pharmacology, and pathology. Therefore we, as scientists, teachers, and authors of this text, consider it appropriate to recognize the evolution of *Histology* into *Cell and Tissue Biology*, which is reflected in the title of this major revision. We wish to provide our readers with the knowledge of contemporary, microscopic, and morphologic science needed in the experi-

mental laboratory and the clinic – set out clearly and succinctly enough to fit into crowded academic schedules. We dedicate this text to our readers who, by their scientific work in the laboratory and in the clinic, will be making invaluable contributions to the public good.

I am grateful to our contributors for their professionalism and their success in setting an excellent record of discipline for our students. Twenty-nine scholars, veteran contributors to the text, are here contributing to its sixth edition. It is a privilege to welcome Joseph Sanger, Jean McGilvray Sanger, Albert I. Farbman, Michael D. Gershon, Eladio A. Nunez, and Virginia A. Black as new contributors. We are delighted that this edition is published by Urban & Schwarzenberg and look forward to a vital, extended relationship. We have been supported and encouraged by our editor, Charles Mitchell, and his father Braxton. I thank Michael Urban and the Mitchells for their trust. I am grateful to Barbara Rowe, working in my office, for her patience and care, and Mary Hsieh Woodman, for copyediting. Thanks go also to Mary Kidd for proofreading and Susan Lohmeyer for indexing. I am grateful to Jill Morris who, arriving late in the day, brought an invaluable indefatigable intelligence and professionalism to making this book. I am happy to acknowledge a great debt to the editors and compositors in Munich, known to me only by their splendid work and their readiness to oblige. I thank Yale Altman, editor at Elsevier, for his goodwill and helpfulness in arranging our move from Elsevier to Urban & Schwarzenberg. I appreciate the continued concern of Robert McGraw of McGraw Hill for this text. It is a privilege to enjoy, for almost forty years, the company of Roy Greep, founder and first editor of this book.

I am sad to record the death of three colleagues since our last edition. John Lawrence, our editor at Elsevier, died as the fifth edition of *Histology* was being completed. His commitment saw us through the fifth edition and set the stage for this one. Lois Tice contributed the thyroid chapter to two editions, a splendid example of clear scientific exposition, and was ready to update it when she died. John Long contributed the chapter on the adrenal glands; we enjoyed and admired his contribution and regret that his expanding career in medical education has been cut short. John Lawrence, Lois Tice, and John Long were our good companions and we mourn their deaths.

This year initiates the second century of the National Institutes of Health (NIH). This marvelous institution has through imaginative support of science, the development of peer review, and a rare ability to bring the federal government, universities, and the private sector into harmonious working relationships, been responsible for the growth of the scientific knowledge we chronicle. On behalf of the contributors of this book, I offer this volume with great respect and deep gratitude to the National Institutes of Health.

Leon Weiss
Philadelphia
April 4, 1988

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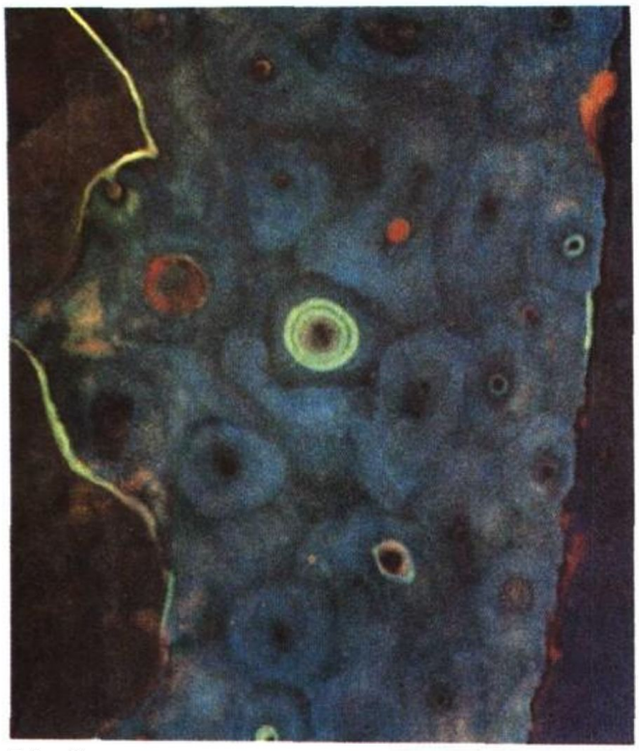
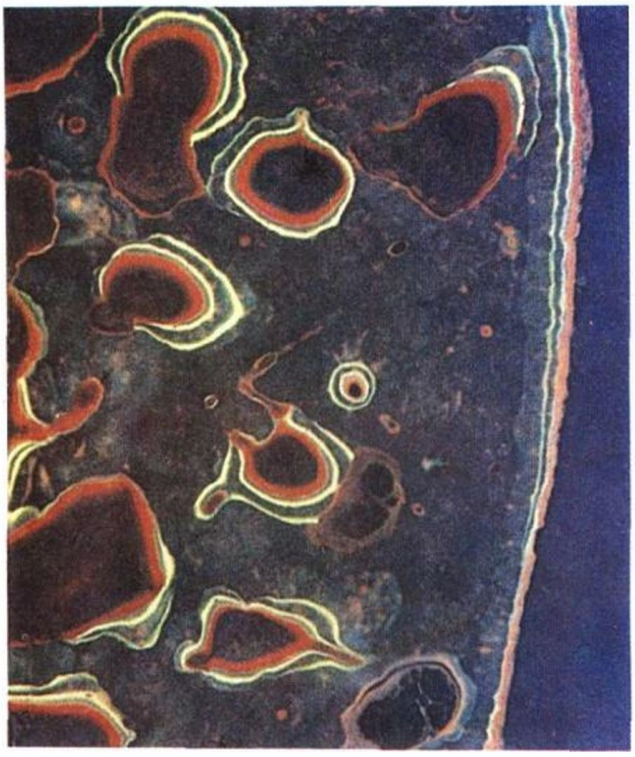
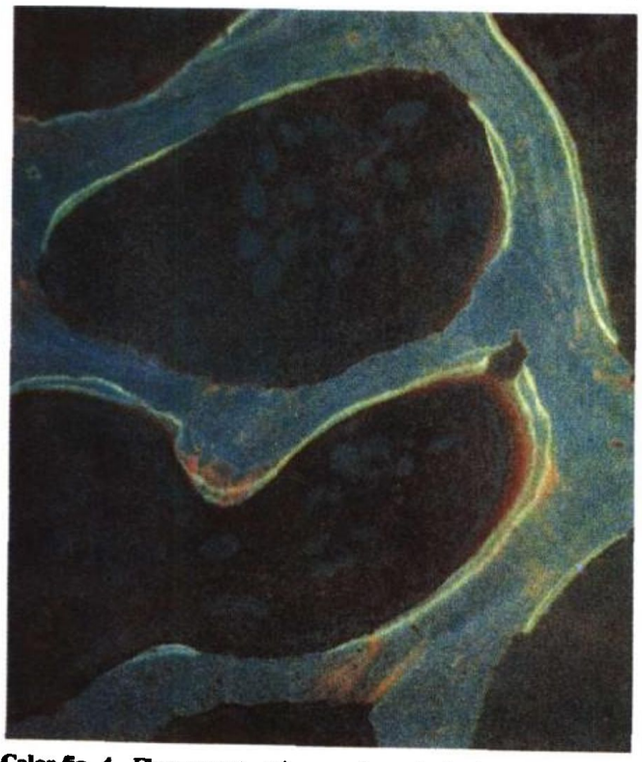
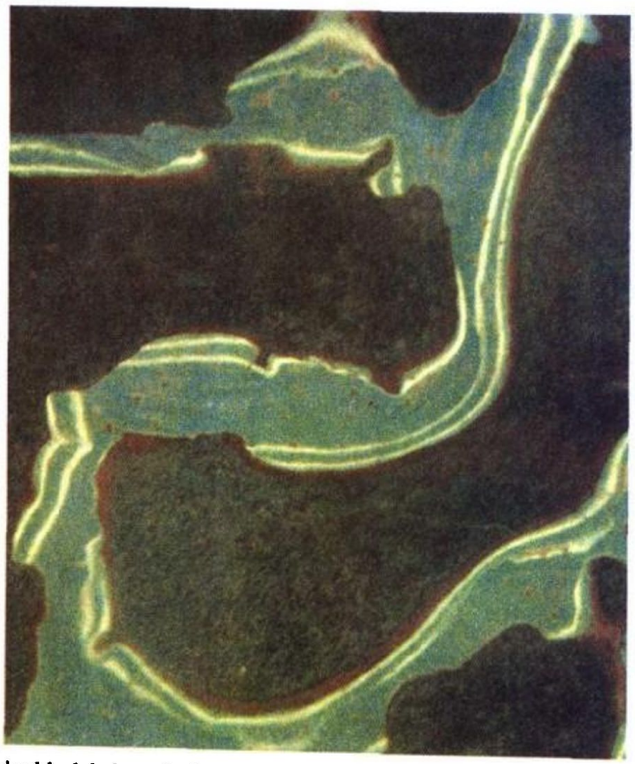


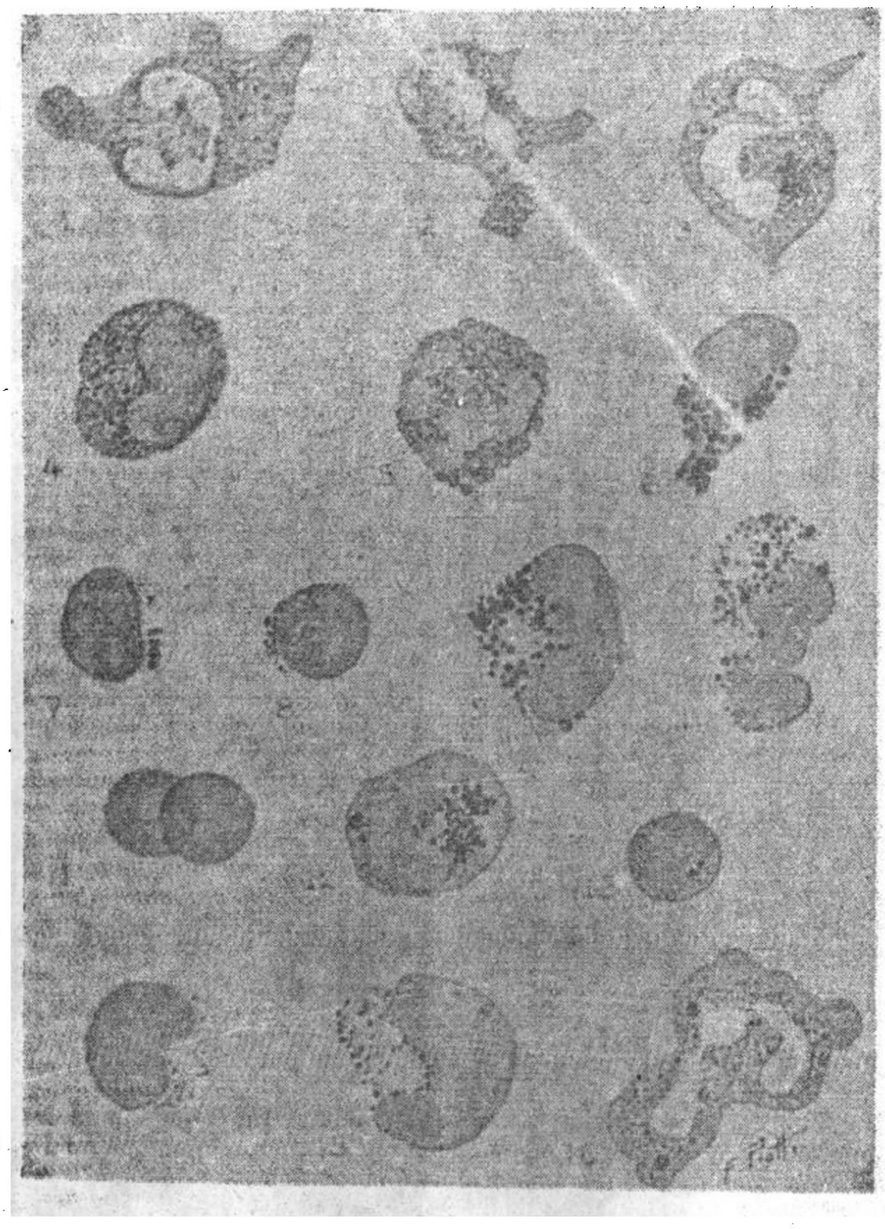


Color fig. 3 Fluorescent micrographs of double-tetracycline-labeled rib section from 1–34 human parathyroid hormone (PTH; **right**) treated and untreated **(left)** young adult beagle dogs. Only one double tetracycline labeled osteone and one resorption cavity can be seen in the untreated specimen **(left)** while almost half of the cortex is inhabited by forming osteones, double-labeled in the PTH-treated bone **(right)**. Note also the drifting of the forming osteones toward the periosteal surface, the faint double-labeling beneath part of the periosteal surface and the increased porosity in the PTH-treated rib **(right)**. 15 μm undecalcified ground, Villanueva-stained section. Yellow and light blue, tetracycline labels; red, osteoid; bone, cortical bone.
(Courtesy of K. Inoue and H. Takahashi.)





Color fig. 4 Fluorescent micrographs of double-tetracycline-labeled trabecular bone from transilial biopsies of 1–34 human parathyroid hormone (PTH; **right**) treated and untreated **(left)** young adult beagle dogs. The PTH-treated trabecular bone **(right)** exhibits a marked increase in double tetracycline labeling, wider double labels and abundant scalloped surfaces compared to untreated biopsy specimen **(left)**. 7 μm undecalcified, Villanueva-stained section. Yellow, tetracycline labels; red, osteoid; blue, trabecular bone.
(Courtesy of K. Inoue and H. Takahashi.)



Color fig. 5 Human blood cells stained supravitally. All cells are from the same individual and are drawn to the same scale. Cells 1–13 are stained with neutral red only (granules and phagocytic vacuoles). Cells 14–16 are stained with neutral red and Janus green (mitochondria). (Preparation Courtesy of E. Tompkins.)

1. Polymorphonuclear neutrophil stained for 20 min at 37°C. The small dots are the specific, refractive granules, which appear brown-red or gray, depending on focus. Unlike phagocytic vacuoles, these do not change with time. The pseudopodia are usually free of the streaming granules. The larger droplets represent the phagocytic vacuoles. There are few of these in a normal neutrophil within this period of time.

2. A neutrophil from the same field after the film had been at room temperature for 1 h. Phagocytosis is slight at the lower temperature, and there is little change in the vacuoles.

3. The same cells as in 2 after the film had been at room temperature for 2 h. The number of lobes of the nucleus has changed somewhat as the result of ameboid movements. The cell has become toxically injured after long exposure and is phagocytizing abnormally.

4. Myelocyte film stained at 37°C for 1 h. There are no ameboid movements and little phagocytosis. The specific granules are more refractive and stain more on the acid side than the granules of polymorphonuclear neutrophils.

5. Polymorphonuclear eosinophil from the same film as 4. The granules are highly refractive, and the intensity of their color consequently varies with focus. They are large, rice-shaped, and fairly uniform in size. They are straw-colored when freshly stained but gradually take on an apricot tint with exposure. Eosinophils rarely contain phagocytic vacuoles.

6. Polymorphonuclear basophil. The granules are large, round, very uniform, and highly refractive; the intensity of staining therefore varies with focus. The granules stain a deeper crimson than phagocytic vacuoles or than the granules of any other cells of the blood. The nucleus rarely shows lobing, and the cells are practically never phagocytic.

7. Intermediate-sized lymphocyte from same film as 4 after the film stood at room temperature for 1 h. The cytoplasm is very clear and contains few phagocytic vacuoles. There are many fewer phagocytic vacuoles than in monocytes and they are arranged indiscriminately. Lymphocytes should rarely be confused with monocytes. Double staining with Janus green also serves to differentiate the two types (see 13 and 14).

8. Small lymphocyte from same film as 7 after the film stood at room temperature for 2 h.

9, 10. Monocytes from the same film after it stained for 5 min at 37°C, and 1 h and 2 h, respectively, at room temperature. Monocytes vary constantly in shape and degree of phagocytosis, depending on ameboid movement. They have the greatest number of phagocytic vacuoles of all blood cells and no granules. The vacuoles vary in size and change position constantly. They increase in both size and number with time of exposure.

11. Two normal erythrocytes. They do not stain. Their color is due entirely to their content of hemoglobin.

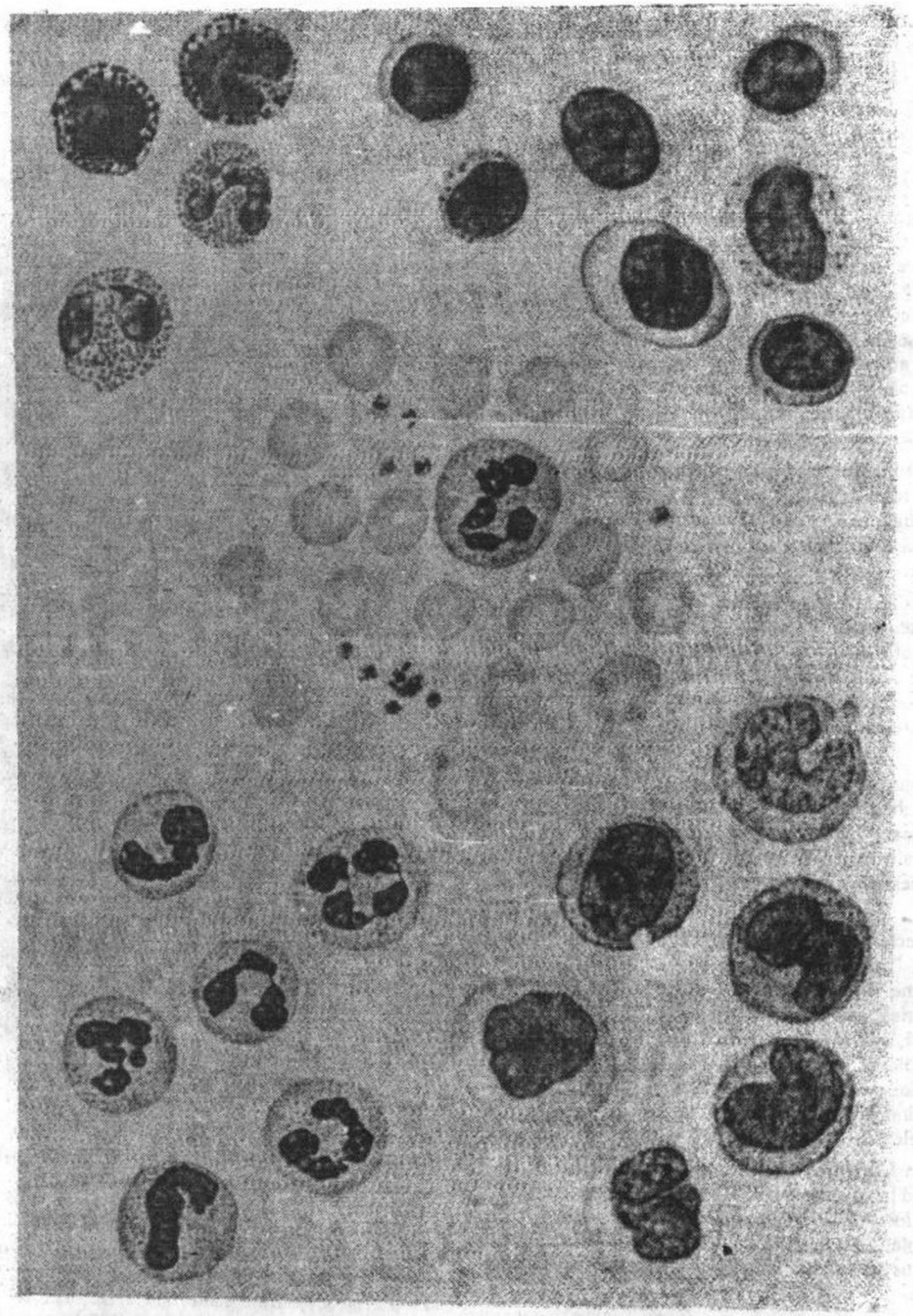
12. Monocyte from same film as 9 and 10. The cell is somewhat younger than those in 9 and 10, less ameboid, and tends to aggregate the phagocytic vacuoles into rosette formation.

13. Erythrocyte from same film as 12. The cell is younger than those in 11 and contains reticulum, which was stained with neutral red.

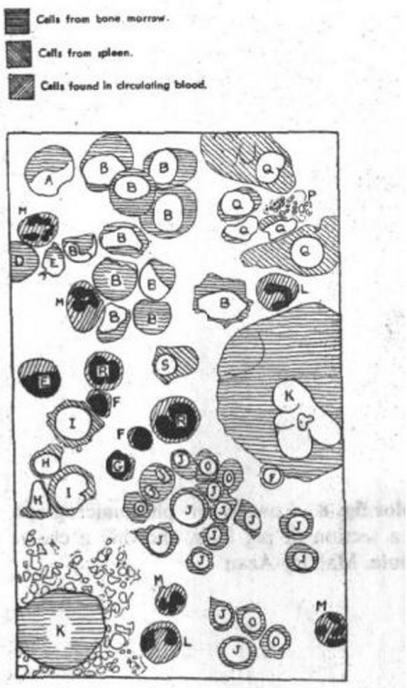
14. Lymphocyte stained with neutral red and Janus green (compare with 7). Janus green inhibits phagocytosis somewhat. The mitochondria stain blue-green, are definitely rod-shaped, and tend to cluster toward the nucleus. They are larger than the mitochondria of monocytes.

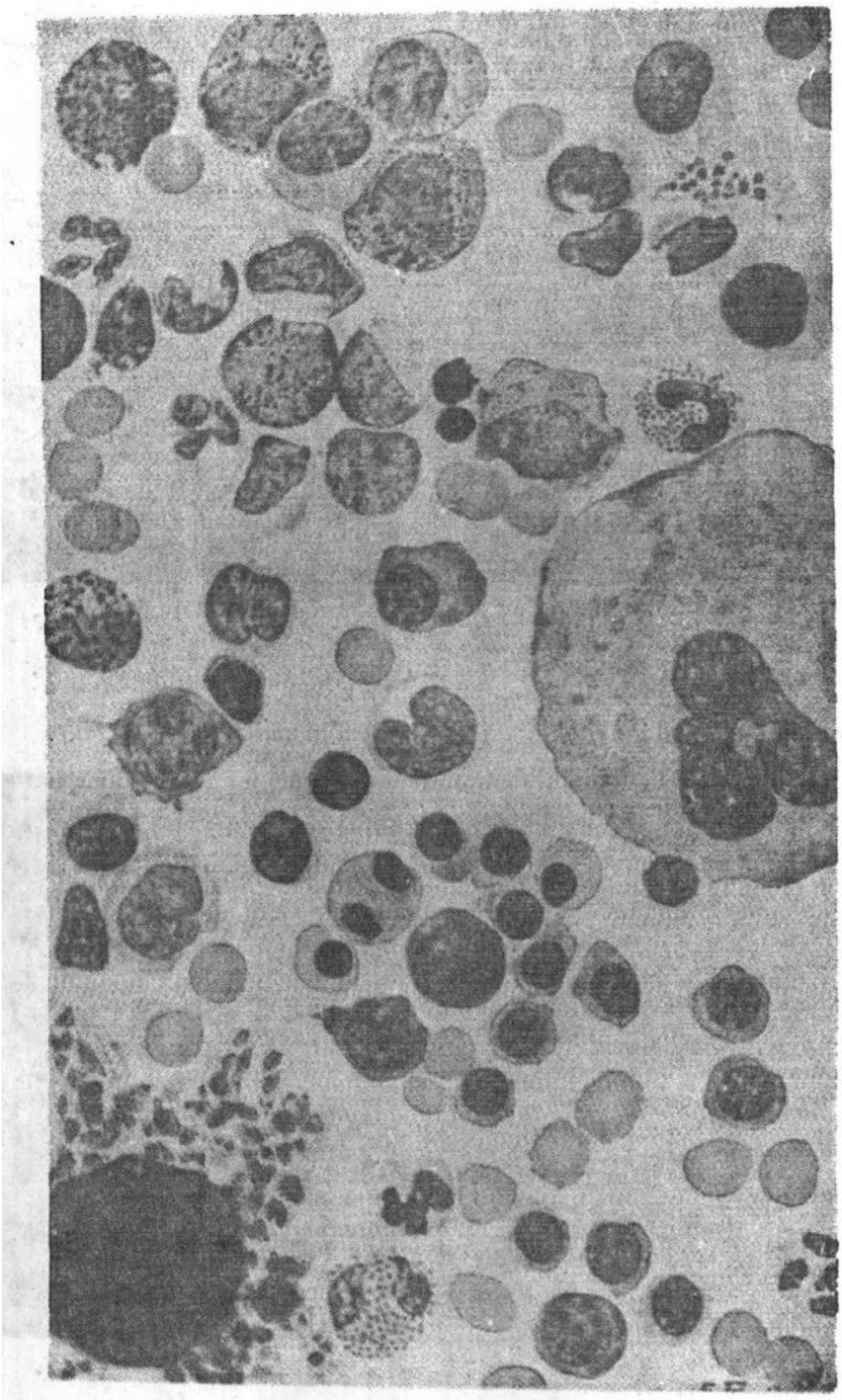
15. Monocyte stained with neutral red and Janus green. Phagocytosis has been inhibited somewhat. The mitochondria are smaller than those in lymphocytes and more scattered (compare with 14).

16. Polymorphonuclear neutrophil stained with neutral red and Janus green. The mitochondria are the size of those in monocytes but are less abundant. Janus green is soon toxic to cells, and this cell shows the toxic action in the form of unusual phagocytic vacuoles.

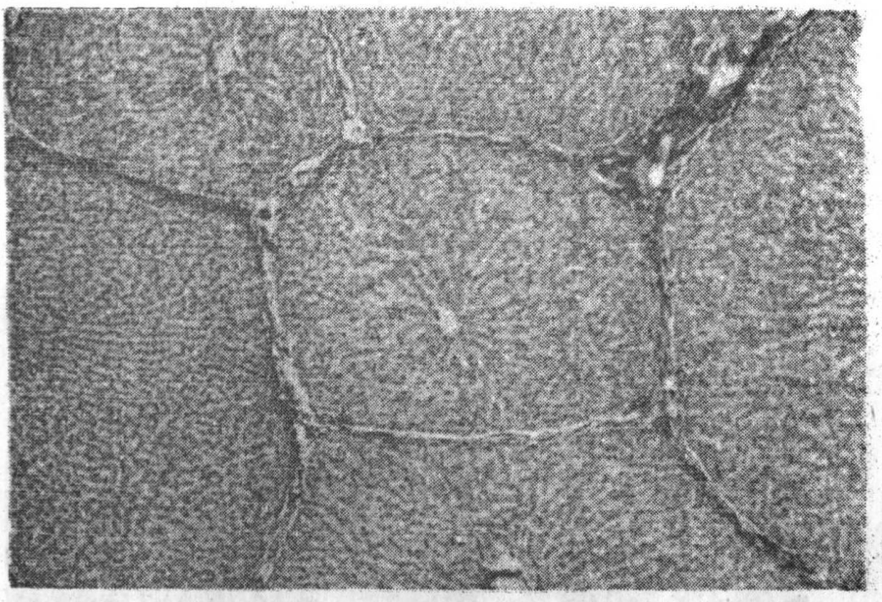


Color fig. 6 Cells from a smear preparation of normal human blood. Wright's stain. In the center, adult red corpuscles, blood platelets, and a polymorphonuclear neutrophil. At left above, two polymorphonuclear basophils and two polymorphonuclear eosinophils. At right above, three large and four small lymphocytes. At left below, polymorphonuclear neutrophils. At right below, six monocytes.

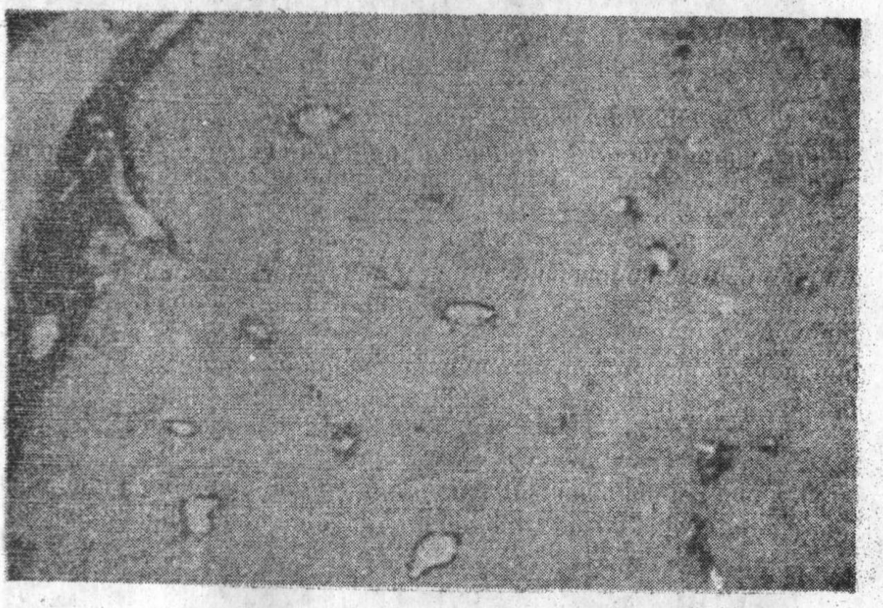




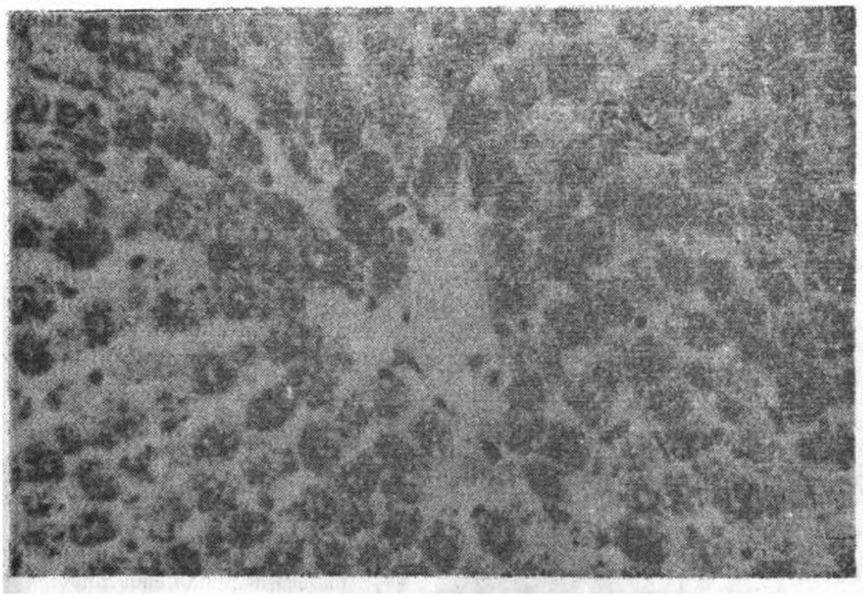
Color fig. 7 Composite plate of blood cells. **A,** eosinophilic myelocyte; **B,** myelocyte; **D,** blast form; **E,** basophilic leukocyte; **F,** small lymphocyte; **G,** medium-sized lymphocyte; **H,** large lymphocyte; **I,** blast form; **J,** basophilic erythroblast; **K,** megakaryocyte; **L,** eosinophilic leukocyte; **M,** neutrophilic leukocyte; **O,** polychromatophilic erythroblast; **P,** platelets; **Q,** reticulum cell; **R,** monocyte; **S,** plasma cell.



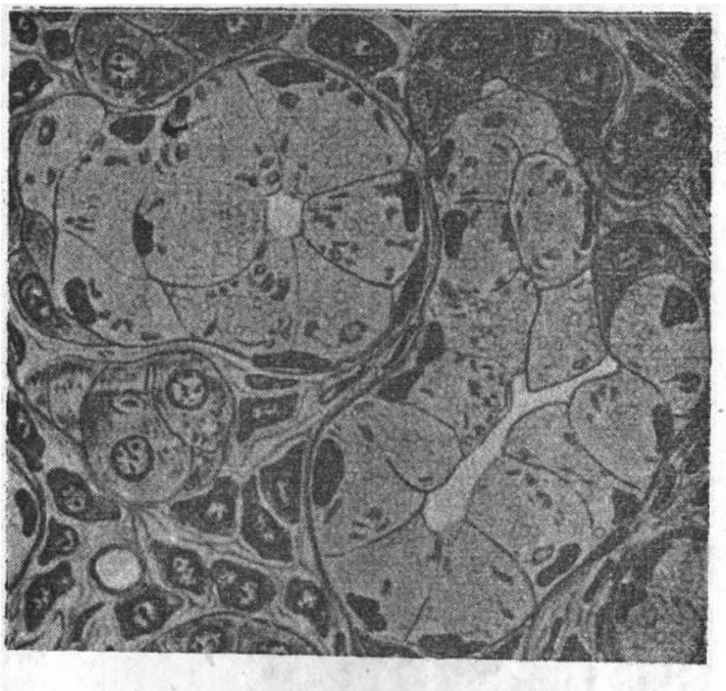
Color fig. 8 Low-power photomicrograph of a section of pig liver, showing a classic lobule. Mallory-Azan.



Color fig. 9 Low-power photomicrograph of a section of human liver, illustrating boundaries of a classic lobule. Mallory-Azan.

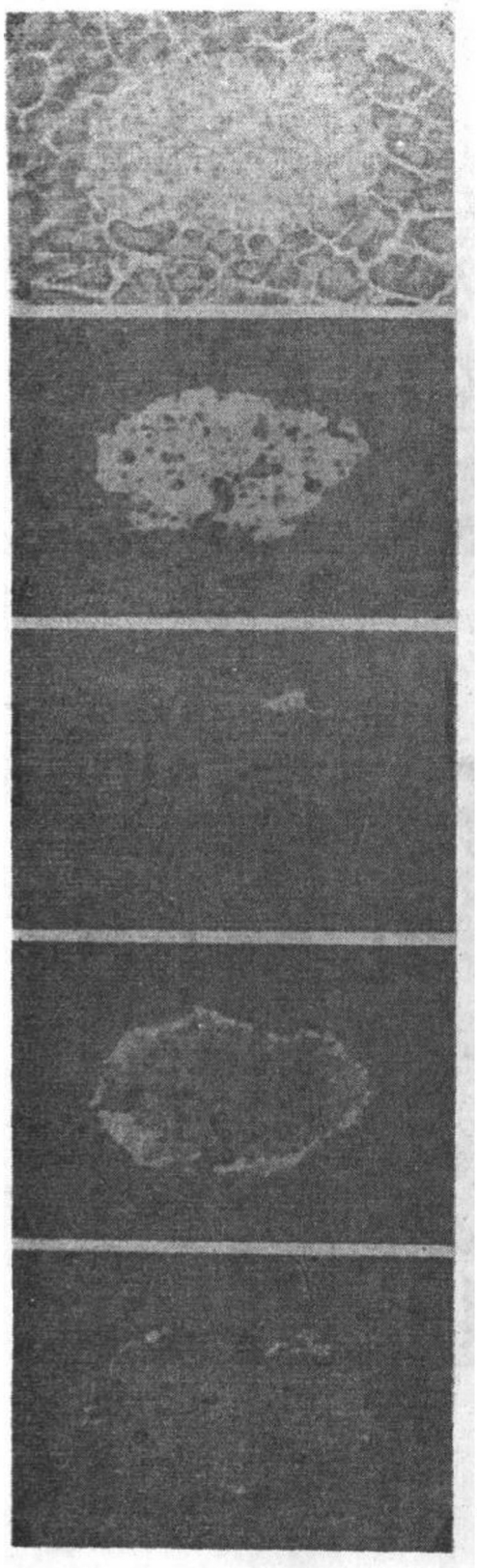


Color fig. 10 Glycogen deposits within the parenchymal cells are stained red by Best's carmine. The quantity of glycogen within the cells varies with the time interval after the last meal and the position of the cell in the lobule. Glycogen is not preserved in routine histological preparations. Rat liver. Carnoy's fixative. Hematoxylin and Best's carmine.



Color fig. 11 Section of the sublingual gland of a 30-year-old man. The mucus-secreting cells are stained blue; the serous cells are gray. Zenker fixation; iron-hematoxylin and Mallory's connective tissue stain.

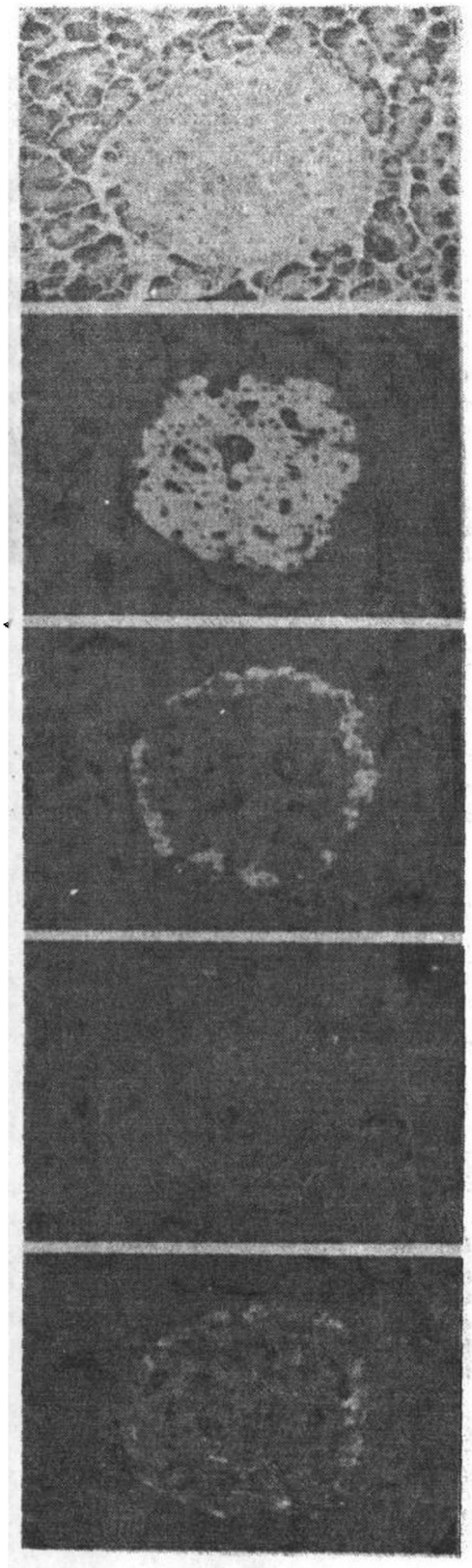
Color Figure 12

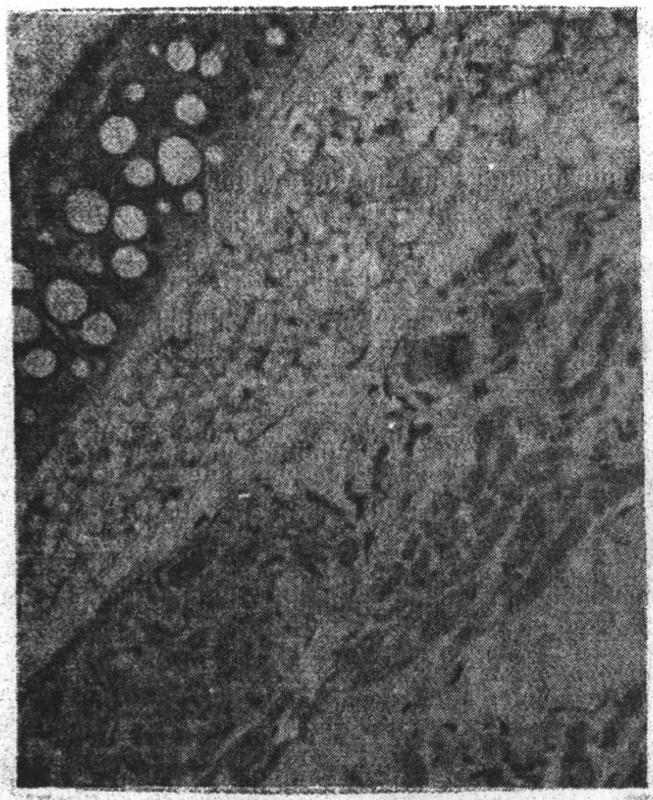


Color fig. 12 Successive serial sections for light microscopy (thickness 3 μm) of two islets of Langerhans of the rat stained respectively with haemalum eosin **(HE)** and with anti-insulin, anti-glucagon, anti-pancreatic polypeptide and anti-somatostatin antisera revealed by the indirect immunofluorescence method.

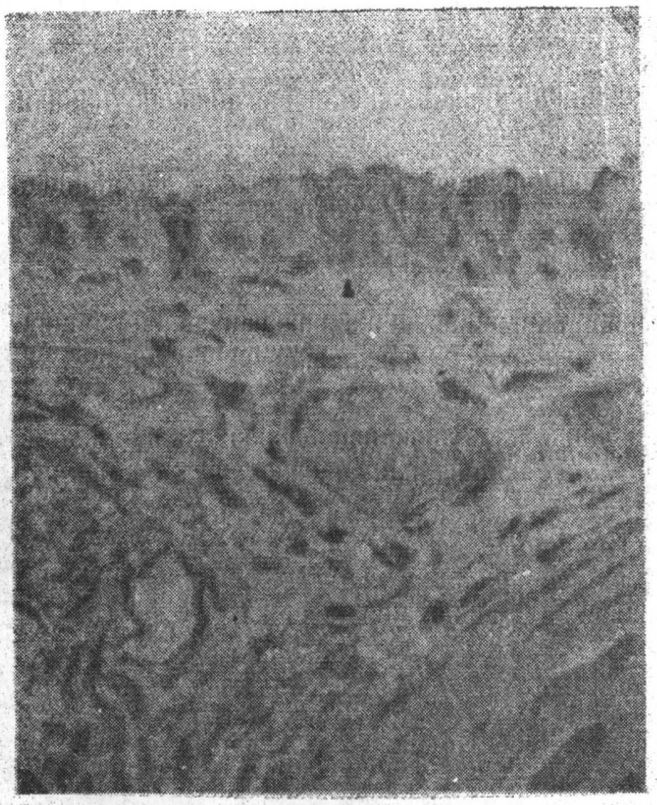
The series to the left shows the distribution of the four endocrine cell types in a "glucagon-rich" or "dorsal" type islet (which originates from the large dorsal primordium during embryogenesis).

The series to the right reveals the distribution of endocrine cell types in a "pancreatic polypeptide-rich" or "ventral" type islet (originating from the small ventral primordium). The reverse ratio between glucagon and pancreatic polypeptide in these two islets is evident. × 175. (Courtesy of L. Orci.)





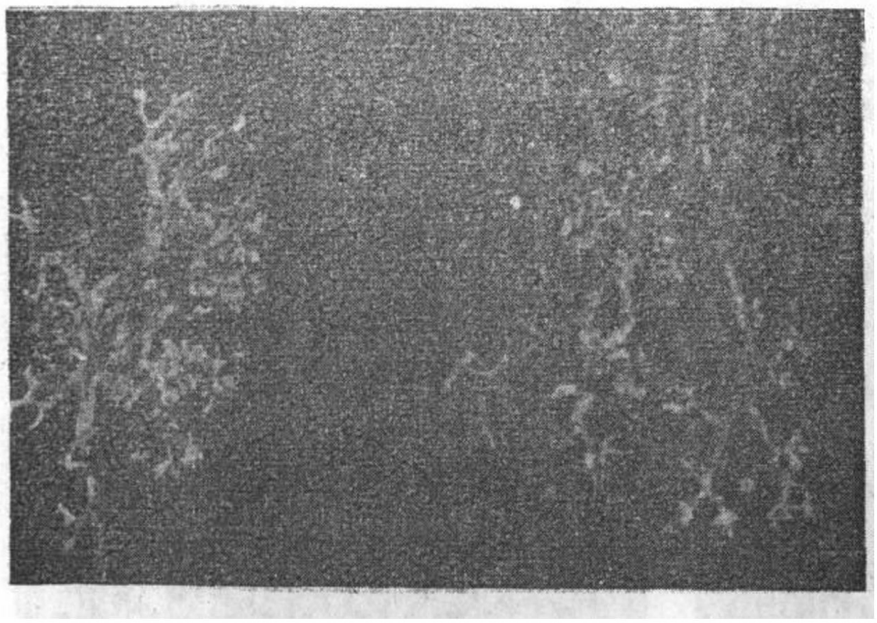
Color fig. 13 Ganglion cells of the pulmonary plexus near the tracheal bifurcation of a hamster. PAS-lead hematoxylin. ×90.



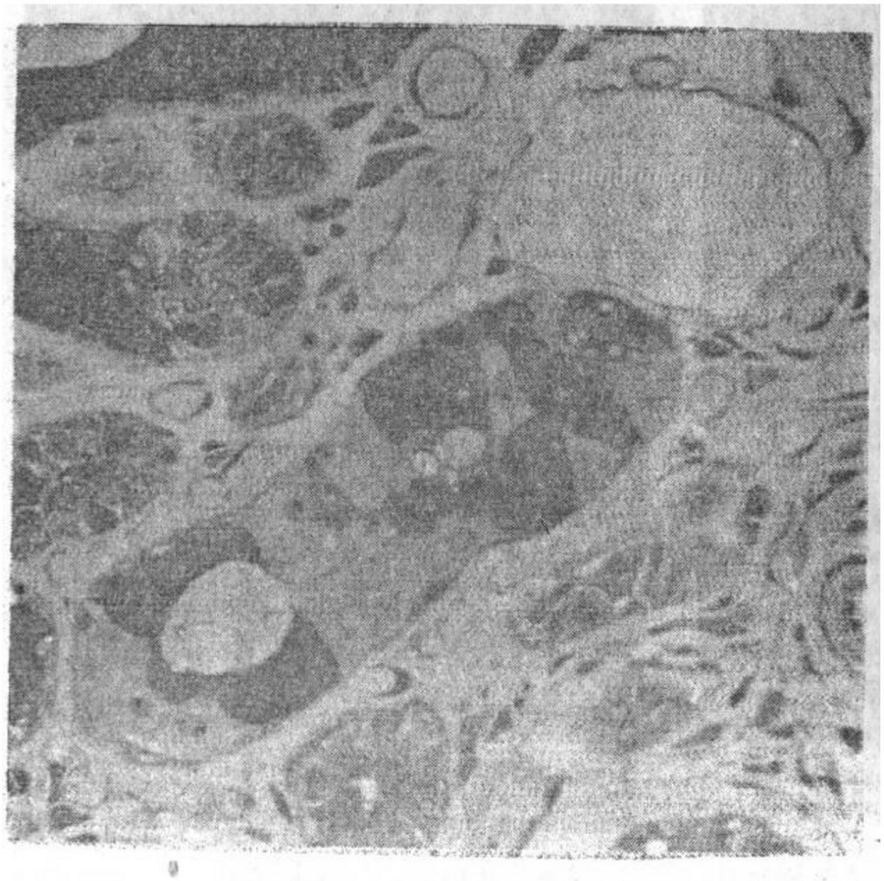
Color fig. 14 Tracheal mucosa of a rat showing a brush cell **(arrowhead)** in the epithelium. PAS-lead hematoxylin. ×800.

Color fig. 15 Epithelium, fiber systems, and glands on the dorsal aspect of the trachea in a mouse. Resorcin-fuchsin, toluidine blue, and alcian green. ×175.

Color fig. 16 Tracheal smooth muscle of mouse inserting on the elastic skeleton just deep to the thick longitudinal fibers. Resorcin-fuchsin, hematoxylin, and alcian green. ×300.



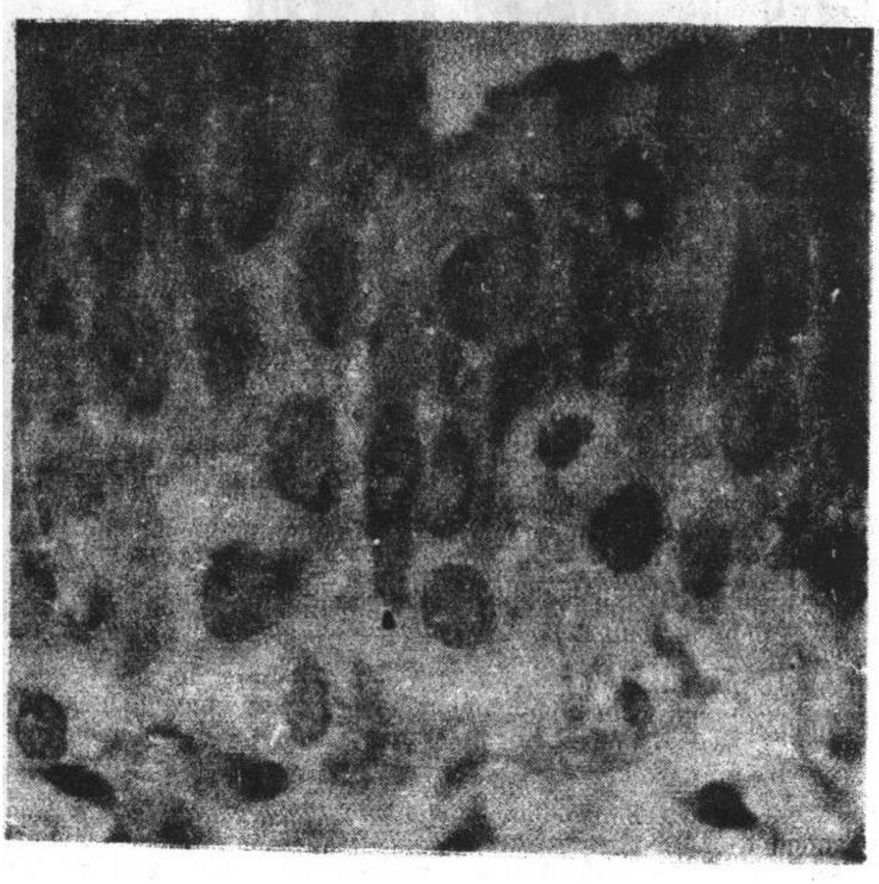
Color fig. 17 The peripheral airway (greenish) with its accompanying pulmonary artery (red) on the left, and together with the pulmonary vein (blue) on the right, as these structures appear in casts.
(From Lauweryns, J. 1962. De Longvaten. Brussels: Ed. Arscia.)



Color fig. 18 Secretory acini and intermediate duct of a human bronchial gland, showing the principal cell types present: serous (stippled purple), mucous (magenta overall), oncocytes (pale grayish), and myoepithelial (lying along basement membrane). PAS-lead hematoxylin. × 420.

Color fig. 19 Hamster bronchiole showing predominance of non-ciliated bronchiolar cells, one with many secretory granules. PAS-lead hematoxylin. ×1,200.

Color fig. 20 Prussian blue-stained iron particles in the bronchus of a mouse, located in macrophages on the surface and in the connective tissue as well as in the epithelium in-between. Basic fuchsin. ×1,100.



Color fig. 21 Human bronchial small-granule cell with basally located secretory matter (**arrowhead**), as well as globule leukocytes (foamy cytoplasm) and other more usual epithelial cells. PAS-lead hematoxylin ×2,000.