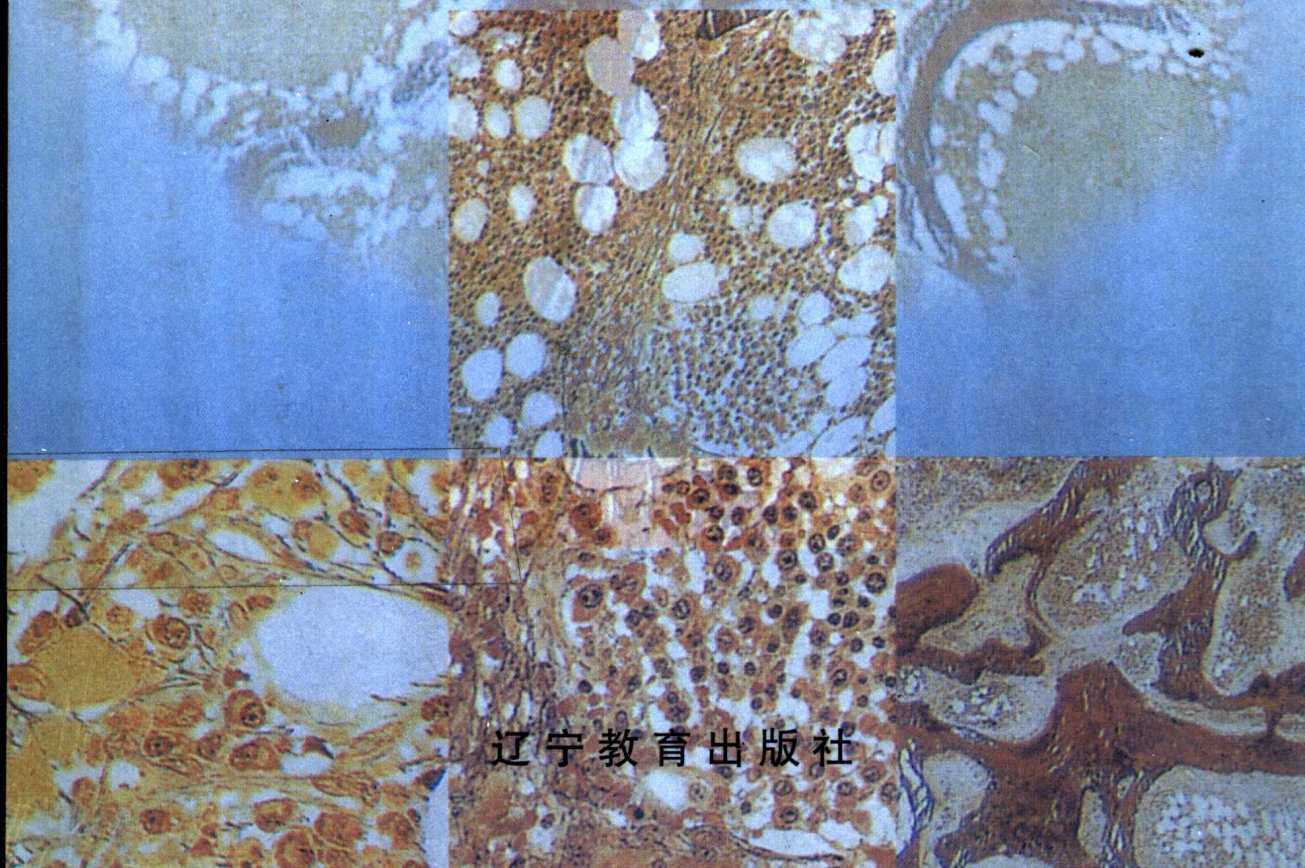
原著 [美] *Peter H. Wiernik*
[美] *George P. Canellos*
[美] *Janice P. Dutcher*
[美] *Robert A. Kyle*

血液肿瘤学

第三版

主译 王良绪



辽宁教育出版社

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译　者（以姓氏笔画为序）

王文生　王　平　王良绪　王茫桔　王继军
李健为　李峰敏　杨玉花　邱志祥　刘　彦
陈渝萍　尚　奕　克晓燕　单　健　赵　昕
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辽宁教育出版社

版权合同登记号图字 06－1999－109 号

图书在版编目(CIP)数据

血液肿瘤学/(美)威尔尼克(Wiernik, P. H.)等著;王良绪主译. －沈阳:辽宁教育出版社,2000.2

书名原文:Neoplastic Diseases of the Blood

ISBN 7－5382－5555－9

Ⅰ. 血… Ⅱ. ①威… ②王… Ⅲ. 白血病－诊疗 Ⅳ. R733.7

中国版本图书馆 CIP 数据核字(1999)第 24542 号

本书包含有药物使用的准确指征、副作用以及剂量,这些可能改变。奉劝读者在服用药物时阅读厂家的有关资料。

出版 辽宁教育出版社
(中国辽宁省沈阳市和平区北一马路 108 号)
网址 http://www.edupress.com.cn
发行 辽宁万有图书发行有限公司
印刷 沈阳新华印刷厂
版次 2000 年 2 月第 1 版
印次 2000 年 2 月第 1 次印刷
开本 787×1092 毫米 1/16
印张 75.75
定价 280.00 元

总发行人 俞晓群
责任编辑 马 芳
韩 梅
责任校对 王文斌
封面设计 曹太文
版式设计 韩 梅

撰稿者
(Contributors)

ALAN C. AISENBERG, M.D.

Professor, Department of Medicine, Harvard Medical School; Physician, Hematology-Oncology Unit, Department of Medicine, Massachusetts General Hospital, Boston, Massachusetts

JAMES O. ARMITAGE, M.D.

Professor and Chairman, Department of Internal Medicine, University of Nebraska College of Medicine, Omaha, Nebraska

DONALD ARMSTRONG, M.D.

Chief, Infectious Disease Service, and Director, Microbiology Laboratory, Memorial Sloan-Kettering Cancer Center, New York, New York

ROBERT L. BAEHNER, M.D.

Executive Chair, Department of Pediatrics, University of Southern California School of Medicine; Vice President, Medical Affairs, Children's Hospital of Los Angeles, Los Angeles, California

REINER BARTL, M.D.

Professor, Department of Internal Medicine III, Klinikum Grosshadern, Ludwig-Maximilians-Universität, Munich, Germany

JOHN M. BENNETT, M.D.

Professor, Departments of Medicine, Pathology, and Laboratory Medicine, University of Rochester School of Medicine and Dentistry; Attending Physician, Department of Medicine, Medical Oncology Unit, Strong Memorial Hospital, Rochester, New York

DANIEL E. BERGSAGEL, C.M, M.D., D.PHIL.

Professor, Department of Medicine, University of Toronto Faculty of Medicine; Consultant Physician, Department of Medicine, Ontario Cancer Institute/Princess Margaret Hospital, Toronto, Ontario, Canada

PHILIP J. BIERMAN, M.D.

Associate Professor, Section of Oncology and Hematology, Department of Internal Medicine, University of Nebraska College of Medicine, Omaha, Nebraska

CLARA D. BLOOMFIELD, M.D.

Professor and Chief, Division of Oncology, Department of Medicine, State University of New York at Buffalo School of Medicine and Biological Sciences; Chair, Department of Medicine, Roswell Park Cancer Institute, Buffalo, New York

GIANNI BONNADONNA, M.D.

Professor, Department of Hematology, University of Milan School of Medicine; Director, Division of Oncology, Department of Cancer Medicine, National Tumor Institute, Milan, Italy

ABY BUCHBINDER, M.D.

Assistant Professor, Department of Medicine, Cornell University Medical College; Attending Physician, Department of Medicine, North Shore University Hospital, Manhasset, New York

BEVERLY BUCK, R.T., B.S.

Instructor, Department of Radiation Oncology, Harvard Medical School; Radiation Technologist, Joint Center for Radiation Therapy, Boston, Massachusetts

GEORGE P. CANELLOS, M.D.

William Rosenberg Professor of Medicine, Department of Medicine, Harvard Medical School; Chief, Division of Clinical Oncology, Dana-Farber Cancer Institute; Physician, Brigham and Women's Hospital, Boston, Massachusetts

ZHONG CHEN, M.D.

Associate Director, Cytogenetics, The Southwest Biomedical Research Institute and Genetrix, Inc., Scottsdale, Arizona

H. JOACHIM DEEG, M.D.

Professor, Department of Medicine, University of Washington School of Medicine; Member, Clinical Research Division, Fred Hutchinson Cancer Research Center, Seattle, Washington

THOMAS G. DeLOUGHERY, M.D.

Assistant Professor, Departments of Medicine and Pathology, Oregon Health Sciences University School of Medicine, Portland, Oregon

SUSAN S. DEVESA, Ph.D.

Chief, Descriptive Studies Section, Biostatistics Branch, Division of Cancer Etiology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

GORDON W. DEWALD, Ph.D.

Professor, Departments of Laboratory Medicine and Medical Genetics, Mayo Medical School; Chief, Division of Laboratory Genetics, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

BARBARA K. DUNN, Ph.D., M.D.

Senior Scientist, Clinical Services Program, Program Resources, Inc./DynCorp; National Cancer Institute-Frederick Cancer Research and Development Center, Frederick, Maryland

JANICE P. DUTCHER, M.D.

Professor, Department of Medicine, Albert Einstein College of Medicine of Yeshiva University; Attending Physician, Department of Oncology, Montefiore Medical Center, Bronx, New York

ARNOLD S. FREEDMAN, M.D.

Associate Professor, Department of Medicine, Harvard Medical School; Division of Hematologic Malignancies, Dana-Farber Cancer Institute, Boston, Massachusetts

EMIL J. FREIREICH, M.D., D.SC. (HON.)

Professor and Director, Adult Leukemia Research Program, Department of Hematology, University of Texas M. D. Anderson Cancer Center, Houston, Texas

BERTHA FRISCH, M.D.

Professor, Departments of Pathology and Hematology, Sourasky Medical Center, Ichilov Hospital and Sackler School of Medicine, Tel-Aviv University, Tel-Aviv, Israel

JANICE L. GABRILOVE, M.D.

Associate Professor, Department of Medicine, Cornell University Medical College; Associate Attending Physician, Division of Hematologic Oncology, Department of Medicine, Memorial Sloan-Kettering Cancer Center; Associate Member, Sloan-Kettering Institute, New York, New York

ROBERT E. GALLAGHER, M.D.

Professor, Departments of Medicine and Oncology, Albert Einstein College of Medicine of Yeshiva University; Attending Physician, Department of Oncology, Montefiore Medical Center, Bronx, New York

ROBERT C. GALLO, M.D.

Chief, Laboratory of Tumor Cell Biology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

MORIE A. GERTZ, M.D.

Professor, Department of Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

JOHN GIBSON, M.B., B.S., F.R.A.C.P., F.R.C.P.A.

Associate Professor, Department of Medicine, University of Sydney; Senior Haematologist, Institute of Haematology, Royal Prince Alfred Hospital, Sydney, Australia

JOHN M. GOLDMAN, D.M., F.R.C.P.

Professor, Department of Haematology, Royal Postgraduate Medical School, Hammersmith Hospital, London, England

SCOTT H. GOODNIGHT, JR., M.D.

Professor, Departments of Medicine and Pathology, Oregon Health Sciences University School of Medicine, Portland, Oregon

MYRTLE Y. GORDON, D.SC., F.R.C.P.

Reader in Experimental Haematology, Department of Haematology, Royal Postgraduate Medical School, Hammersmith Hospital, London, England

PHILIP R. GREIPP, M.D.

Professor, Departments of Medicine and Laboratory Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine and Division of Hematopathology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

JAMES D. GRIFFIN, M.D.

Professor, Department of Medicine, Harvard Medical School; Division of Hematologic Malignancies, Dana-Farber Cancer Institute, Boston, Massachusetts

SEYMOUR GRUFFERMAN, M.D.

Professor and Chairman, Department of Family Medicine and Clinical Epidemiology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania

SUBHASH GULATI, M.D.

Professor, Department of Medicine, Cornell University Medical College; Director, Clinical Research and Stem Cell Transplantation, Division of Hematology-Oncology, New York Hospital-Cornell Medical Center, New York, New York

CLARK W. HEATH, JR., M.D.

Clinical Professor, School of Public Health, Emory University School of Medicine; Vice President, Department of Epidemiology and Surveillance Research, American Cancer Society, Atlanta, Georgia

DIETER F. HOELZER, M.D.

Professor, Department of Internal Medicine; Chief, Department of Hematology, Johann-Wolfgang-Goethe University, Frankfurt, Germany

JAMES F. HOLLAND, M.D.

Distinguished Professor, Department of Neoplastic Diseases, Mount Sinai School of Medicine of the City University of New York; Director Emeritus, The Gerald H. Ruttenberg Cancer Center, Mount Sinai Hospital, New York, New York

JIMMIE C. HOLLAND, M.D.

Professor and Vice-Chairman, Department of Psychiatry, Cornell University Medical College; Chief, Psychiatry Service, Memorial Sloan-Kettering Cancer Center, New York, New York

WALTER T. HUGHES, M.D.

Professor, Department of Pediatrics, University of Tennessee, Memphis, College of Medicine; Chairman, Department of Infectious Diseases, St. Jude Children's Research Hospital, Memphis, Tennessee

RALF HUSS, M.D.

Fellow, Pathologisches Institut, Universität München, Munich, Germany

ELAINE S. JAFFE, M.D.

Chief, Hematopathology Section, Laboratory of Pathology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

ROBERT B. JENKINS, M.D., PH.D.

Associate Professor, Department of Laboratory Medicine, Mayo Medical School; Consultant, Division of Laboratory Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

DOUGLAS E. JOSHUA, M.B., B.S., D.PHIL. (OXON), F.R.A.C.P., F.R.C.P.A.

Clinical Professor, Department of Medicine, University of Sydney; Director, Institute of Haematology, Royal Prince Alfred Hospital, Sydney, Australia

MICHAEL J. KEATING, M.B., B.S.

Doctor Kenneth B. McCredie Chair in Clinical Leukemia Research and Professor, Department of Hematology, University of Texas M. D. Anderson Cancer Center, Houston, Texas

PATRICIA KEEGAN, M.D.

Clinical Assistant Professor, Division of Medical Oncology, University of North Carolina at Chapel Hill School of Medicine, Chapel Hill, North Carolina

KAREN E. KING, M.D.

Fellow, Division of Transfusional Medicine, Department of Pathology, The Johns Hopkins University School of Medicine and The Johns Hopkins Hospital, Baltimore, Maryland

H. PHILLIP KOEFFLER, M.D.

Professor, Division of Hematology-Oncology, Department of Medicine, University of California, Los Angeles, UCLA School of Medicine; Chief, Division of Hematology-Oncology, Cedars-Sinai Medical Center, Los Angeles, California

RAZELLE KURZROCK, M.D.

Professor, Department of Bioimmunotherapy, University of Texas M. D. Anderson Cancer Center, Houston, Texas

ROBERT A. KYLE, M.D.

Professor, Departments of Medicine and Laboratory Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

FREDERICK P. LI, M.D.

Professor, Department of Epidemiology, Harvard School of Public Health; Professor, Department of Medicine, Harvard Medical School; Division of Cancer Epidemiology and Control, Dana-Farber Cancer Institute, Boston, Massachusetts

ALBERT Y. LIN, M.D., M.P.H.

Senior Staff Fellow, Genetic Epidemiology Branch, Division of Cancer Etiology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

MARTHA S. LINET, M.D.

Epidemiologist, Biostatistics Branch, Division of Cancer Etiology, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

DAN L. LONGO, M.D.

Director, Biological Response Modifiers Program, Division of Cancer Treatment, National Cancer Institute-Frederick Cancer Research and Development Center, Frederick, Maryland

JOHN A. LUST, M.D., PH.D.

Professor, Department of Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

IAN T. MAGRATH, M.B., F.R.C.P., F.R.C. PATH.

Professor, Department of Pediatrics, Uniformed Services University of the Health Sciences F. Edward Hébert School of Medicine; Chief, Lymphoma Biology Section, Pediatric Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

KAREN C. MARCUS, M.D.

Assistant Professor, Department of Radiation Oncology, Harvard Medical School; Joint Center for Radiation Therapy, Boston, Massachusetts

PETER M. MAUCH, M.D.

Associate Professor, Department of Radiation Oncology, Harvard Medical School; Joint Center for Radiation Therapy, Boston, Massachusetts

SUSAN M. McKENNA, M.D.

Assistant Professor, Division of Pediatrics, Department of Medicine, University of Southern California School of Medicine; Head, Division of Pediatric Oncology, Department of Health Services, Los Angeles County-University of Southern California Medical Center, Los Angeles, California

MALCOLM R. McKENZIE, M.D.

Professor, Division of Hematology/Oncology, Department of Internal Medicine, University of California, Davis, School of Medicine; Director, Center for Blood Research, Sacramento Medical Foundation Blood Center, Sacramento, California

L. JEFFERY MEDEIROS, M.D.

Director of Hematopathology, Division of Pathology, City of Hope National Medical Center, Duarte, California

KRZYSZTOF MRÓZEK, M.B., M.R.C.P.I.

Cytogenetics Research Laboratory and Department of Medicine, Roswell Park Cancer Institute, Buffalo, New York

MAURICE E. MURPHY, M.B., M.R.C.P.I.

Fellow, Infectious Disease Service, Memorial Sloan-Kettering Cancer Center, New York, New York

LEE M. NADLER, M.D.

Professor, Department of Medicine, Harvard Medical School; Division of Hematologic Malignancies, Dana-Farber Cancer Institute, Boston, Massachusetts

PAUL M. NESS, M.D.

Associate Professor, Division of Transfusion Medicine, Department of Pathology, The Johns Hopkins University School of Medicine; Director, Blood Bank, The Johns Hopkins Hospital, Baltimore, Maryland

SUSAN O'BRIEN, M.D.

Associate Professor, Department of Hematology, The University of Texas M. D. Anderson Cancer Center, Houston, Texas

HOWARD OZER, M.D.

Professor and Chief, Division of Medical Oncology, University of North Carolina at Chapel Hill School of Medicine; Associate Director for Clinical Affairs, Lineberger Comprehensive Cancer Center, Chapel Hill, North Carolina

ELISABETH PAIETTA, PH.D.

Associate Professor, Department of Medicine, Albert Einstein College of Medicine of Yeshiva University; Clinical Laboratory Director, Albert Einstein Cancer Center, Bronx, New York

DOROTHY J. PARK, M.D.

Assistant Professor, Division of Hematology-Oncology, Department of Medicine, University of California, Los Angeles, UCLA School of Medicine; Division of Hematology-Oncology, Cedars-Sinai Medical Center, Los Angeles, California

LAWRENCE D. PIRO, M.D.

Director, Ida M. and Cecil H. Green Cancer Center; Head, Division of Hematology and Oncology, Scripps Clinic and Research Foundation, La Jolla, California

BERNARD J. POIESZ, M.D.

Professor, Departments of Medicine and Microbiology, State University of New York Health Science Center at Syracuse College of Medicine; Head, Division of Hematology/Oncology, and Director, Regional Oncology Center, State University of New York Health Science Center, Syracuse, New York

MICHAEL POTTER, M.D.

Chief, Laboratory of Genetics, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

LINDA A. POTTERN, M.P.H., Ph.D.

Senior Epidemiologist, Office of Disease Prevention, National Institutes of Health, Bethesda, Maryland

WALDEMAR PRUZANSKI, M.D.

Professor, Department of Medicine; Director, Immunology Diagnostic Research Centre; Director, Clinical Immunology and Allergy; Director, Inflammation Research Group, University of Toronto Faculty of Medicine; Head, Division of Immunology, The Wellesley Hospital, Toronto, Ontario, Canada

CHARLES ROMERO, M.D.

Research Associate, Cornell University Medical College; Research Associate, New York Hospital-Cornell Medical Center, New York, New York

ANDREW J. ROTH, M.D.

Assistant Professor, Department of Psychiatry, Cornell University Medical College; Assistant Attending Psychiatrist, Psychiatry Service, Memorial Sloan-Kettering Cancer Center, New York, New York

AVERY A. SANDBERG, M.D., D.SC.

Senior Medical Director, The Southwest Biomedical Research Institute and Genetrix, Inc., Scottsdale, Arizona

ARMANDO SANTORO, M.D.

Vice Director, Division of Medical Oncology, National Institute for the Study and Care of Cancer, Milan, Italy

ALAN SAVEN, M.D.

Associate Director for Clinical Research, Ida M. and Cecil H. Green Cancer Center; Member, Division of Hematology and Oncology, Scripps Clinic and Research Foundation, La Jolla, California

CHARLES A. SCHIFFER, M.D.

Professor, Departments of Medicine and Oncology, University of Maryland School of Medicine; Chief, Division of Hematologic Malignancies, University of Maryland Cancer Center, Baltimore, Maryland

KENT A. SEPKOWITZ, M.D.

Assistant Professor, Department of Medicine, Cornell University Medical College; Assistant Attending Physician, Division of Infectious Diseases, New York Hospital-Cornell Medical Center, New York, New York

AZIZA T. SHAD, M.D.

Associate Professor, Pediatric Oncology Branch, National Cancer Institute, National Institutes of Health, Bethesda, Maryland

MURIEL F. SIADAK, P.A-C.

Physician Assistant, Long-Term Follow-Up, Fred Hutchinson Cancer Reserach Center, Seattle, Washington

MURRAY N. SILVERSTEIN, M.D.

Professor, Department of Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

ARTHUR T. SKARIN, M.D.

Associate Professor, Department of Medicine, Harvard Medical School; Director, Hematology Laboratory, Division of Medical Oncology, Department of Medicine, Dana-Farber Cancer Institute, Boston, Massachusetts

RICHARD M. STONE, M.D.

Assistant Professor, Department of Medicine, Harvard Medical School; Associate Physician, Division of Medical Oncology, Department of Medicine, Dana-Farber Cancer Institute, Boston, Massachusetts

KEITH M. SULLIVAN, M.D.

Professor, Department of Medicine, University of Washington School of Medicine; Medical Director, Long-Term Follow-Up, Division of Clinical Research, Fred Hutchinson Cancer Research Center, Seattle, Washington

MARTIN S. TALLMAN, M.D.

Assistant Professor, Division of Hematology-Oncology, Department of Medicine, Northwestern University School of Medicine, Chicago, Illinois

MOSHE TALPAZ, M.D.

Professor and Chairman (Ad Interim), Department of Bioimmunotherapy, The University of Texas M. D. Anderson Cancer Center, Houston, Texas

AYALEW TEFFERI, M.D.

Assistant Professor, Department of Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

NUKHET N. TUZUNER

Professor, Department of Pathology, Instanbul University Carrahpasa Medical Faculty, Instabul, Turkey

BRIAN VAN NESS, Ph.D.

Professor, Institute of Human Genetics and Department of Biocheistry, University of Minnesota Medical School—Minneapolis, Minneapolis, Minnesota

JULIE M. VOSE, M.D.

Associate Professor, Section of Oncology and Hematology, Department of Internal Medicine, University of Nebraska College of Medicine, Omaha, Nebraska

DIETLIND L. WAHNER-ROEDLER, M.D.

Assistant Professor, Department of Medicine, Mayo Medical School; Consultant, Division of Area General Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

HOWARD J. WEINSTEIN, M.D.

Associate Professor, Division of Hematology-Oncology, Department of Pediatrics, Harvard Medical School; Dana-Farber Cancer Institute, Children's Hospital, Boston, Massachusetts

PETER H. WIERNIK, M.D.

Professor, Department of Medicine, Albert Einstein College of Medicine of Yeshiva University; Associate Director for Clinical Research, Albert Einstein Cancer Center; Attending Physician, Department of Oncology, Montefiore Medical Center, Bronx, New York

WOLFGANG WILMANNS, M.D.

Professor, Department of Internal Medicine III, Klinikum Grosshadem, Ludwig-Maximilians-Universität, Munich, Germany

JOHN R. WINGARD, M.D.

Professor, Department of Medicine, Emory University School of Medicine; Director, Bone Marrow Transplant Program, The Emory South Clinic, Atlanta, Georgia

THOMAS E. WITZIG, M.D.

Associate Professor, Department of Medicine, Mayo Medical School; Consultant, Division of Hematology and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota

译者序言

血液肿瘤（neoplastic diseases of the blood）或称血液恶性肿瘤（hematologic malignancies）主要包括急性及慢性白血病，多发性骨髓瘤及其相关疾病，淋巴瘤及其相关疾病。自从本世纪初首先用放射线治疗霍奇金病后，直至40年代才开始对急性白血病、淋巴瘤及多发性骨髓瘤应用化学药物治疗并取得一定疗效。以后各种化学药物不断出现，并按照细胞动力学原理，组成多种药物的联合化疗或化疗与放疗联合方案，使完全缓解率显著提高，长期无病生存者大大增多。以上的初步成就促使目前对血液肿瘤的研究，从基础到临床，已在世界各地广泛蓬勃开展，成为热点。《血液肿瘤学》就是介绍这一领域的研究成果和进展状况。第一与第二版分别于1985年及1991年出版，本版为第三版于1996年出版。本版内容共分5篇：前4篇论述以上4种疾病，第5篇为支持治疗。各篇均详细参考近几年的大量文献资料中的新进展，并对每种疾病从历史回顾、流行病学、病理学、形态学、分子遗传学、分类、诊断、治疗和预后进行详细论述。在支持治疗中，又介绍了骨髓或其他来源的造血干细胞的采集、加工处理及移植技术。尤其是输血小板方面。描写细致具体，对临床工作很有实用价值。最后还增补了病人心理治疗章，有助于消除病人的抑郁、悲观、恐惧情绪，改善其生活质量。

本版4位主编中，有3位是肿瘤专业的，这是因为在国外这些疾病也包括在肿瘤学范围内。参加编写的作者共99名，主要来自美国、德国、意大利、英国、加拿大、澳大利亚和以色列等，他们在该领域中均有很深的造诣，他们所提供的研究成就和经验可作为我国从事血液学及肿瘤学，基础或临床工作者的借鉴。尤其是国内大多数医务工作者没有条件或机会能及时阅读到国外新版的书籍或文献资料，有鉴于此，本书的译本希望能为他们提供一本有价值的参考文献。

我们荣幸地受辽宁教育出版社约稿，从1998年开始进行翻译，参加翻译人员主要为北京医科大学第三医院及第一医院血液科的教授、副教授、主治医师及研究生。由于人员较多，水平不同，国内有关医药的翻译名词常欠统一，因此翻译有错误或不当之处，务请同道批评指正。

北京医科大学第三医院

王良绪

1999年7月8日

前　言

《血液肿瘤学》自从1991年第二版出版之后，人们对这一领域的研究成果和进展有了较深了解，更有理由认为第三版的出版对了解血液恶性肿瘤的本质及其治疗方面已有惊人的进展。第三版引用了最新的，近2年来的大量文献资料，并按照过去几版同样的设计，使其成为一本易懂的、内容渊博的书籍，既面对血液及肿瘤专家，也适用于内科领域中从事这方面专业的人员。本版还增添了新的5章，其余各章也进行了广泛改写。新增5章涉及急性早幼粒细胞白血病的诊断和治疗；多发性骨髓瘤及其相关疾病的分子遗传学；骨髓采集、加工处理、贮存及移植；其他来源的造血干细胞及其应用；血液恶性肿瘤病人的心理治疗。大约有100位作者参加编写，其中新作者占30名以上，为本次出版贡献了他们的经验、智慧和提供了新鲜血液。本版和前几版一样分为5篇，每篇由一位编辑主要负责：慢性白血病及相关疾病（George P. Canellos），急性白血病（Peter H. wiernik），骨髓瘤及相关疾病（Robert A. Kyle），淋巴瘤（Peter H. Wiernik），及支持治疗（Janice P. Dutcher）。Dr. Dutcher 是该版新的编辑，对其它几位老编辑在此仍表示由衷地欢迎。

我们衷心希望血液恶性肿瘤病人将是这本新版的直接受益者。

我们感谢出版公司Churchill Livingstone 鼓励我们承担创作这本新版的工作，我们以此为荣。对出版公司的老编辑Kerry Willis的指导、才智和协助，Marc Strauss耐心地和我们相处，Paul Bernstein这位资深发行编辑的帮助，使之按计划交付完成最后产品，均表示深切的谢意。

特别要感谢的是Elisabeth Paietta, Ph.D.，在本版创作的全过程中，对编辑工作协助起到巨大作用。

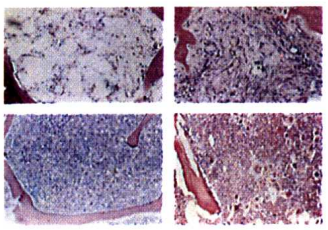
Peter H. Wiernik, M.D.

George P. Canellos, M.D.

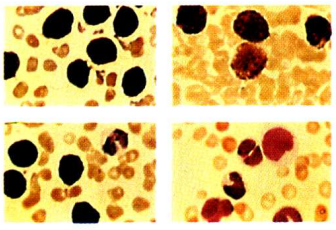
Janice P. Dutcher, M.D.

Robert A. Kyle, M.D.

王良绪　译



图版 3-1A



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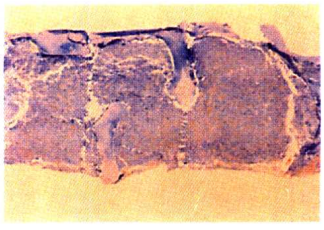

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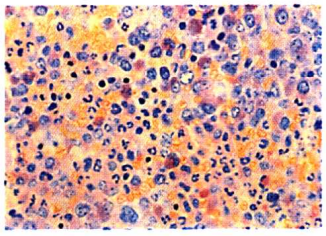

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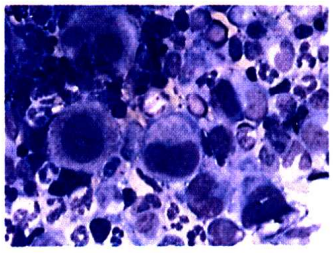

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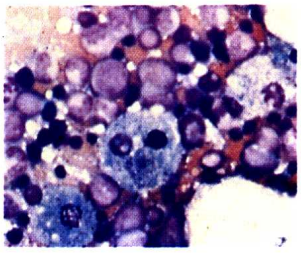

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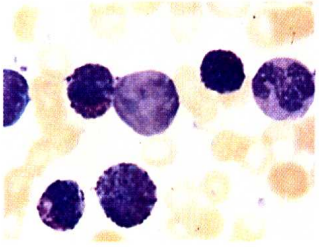

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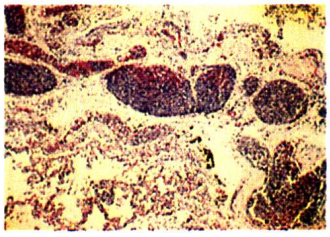


图版 3-1H

图版 3-1

（A）三种可能发生的急性白血病（左下图）的骨髓活检异常。右上图示骨髓广泛地被纤维组织代替（诊断：骨髓纤维化）。右下图示三系细胞增生，其原因是真红细胞增多。为对比左上图示再障骨髓像，被脂肪组织代替（HE染色 × 40）。

（B）在骨髓增生性疾病（MPD）中采用碱性磷酸酶染色，左上图示真红患者中性粒细胞的活性明显增加；右上图示伴髓外化生的骨髓纤维化患者的活性中度增加和白细胞的反应（左下图）；慢粒患者的活性则缺乏（右下图）（Kaplow法，× 1000）。

（C）低倍镜下观察慢粒患者的骨髓活检，有特征性的高度细胞增生，仅 1% ~ 2% 为脂肪组织。在正常骨髓中 50% ~ 60% 为脂肪组织（HE 染色，× 25）。

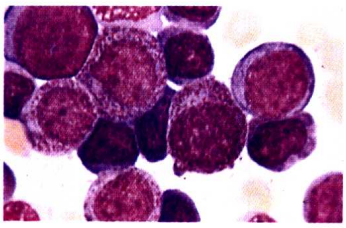
（D）高倍镜下的图C示不同成熟度的粒系细胞明显增生，包括多量的嗜酸细胞、嗜碱细胞不易见到。（HE染色，× 1000）。

（E）慢粒患者骨髓穿刺示巨核细胞增加，包括不典型的和不成熟的细胞（瑞氏 - 姬姆萨染色，× 400）。

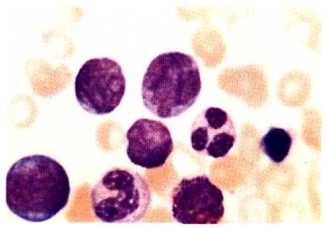
（F）慢粒患者的骨髓穿刺示贮存池细胞增加，包括海蓝色的组织细胞（瑞氏 - 姬姆萨染色，× 1000）。

（G）慢粒患者的血涂片，示嗜酸细胞增加（各类计数约 15%）（瑞氏 - 姬姆萨染色，× 1000）。

（H）慢粒加速期患者的肺切片（其WBC数目为400000/mm^3），可见肺内有明显的白细胞淤滞，它可导致患者死亡。（HE 染色，× 40）。



图版 3-1I



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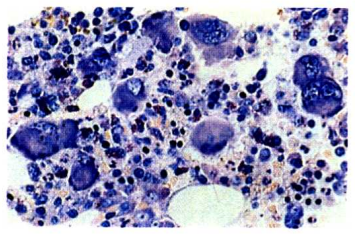
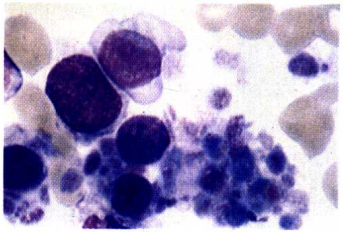
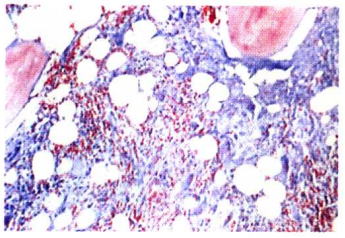


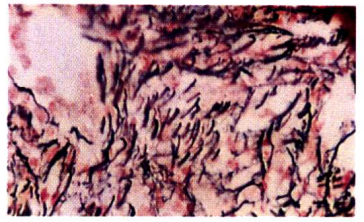
图版 3-1K



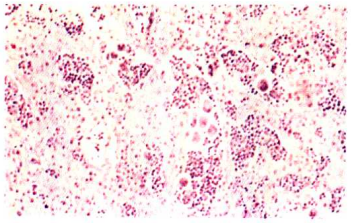
图版 3-1L



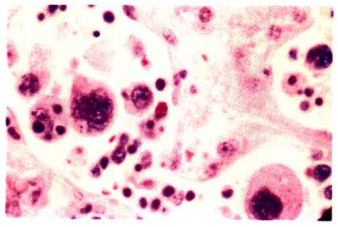
图版 3-1M



图版 3-1N



图版 3-1O



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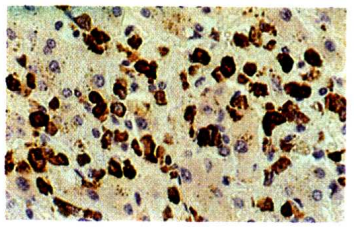
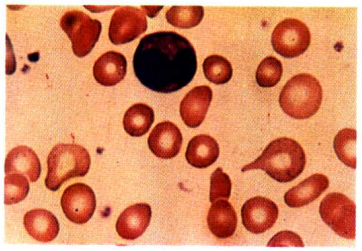
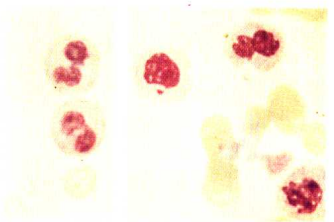


图版 3-1Q



图版 3-1R



图版 3-1S

图版 3-1（续）

（I）图 O 患者的外周血涂片示粒系核左移（瑞氏 - 姬姆萨染色，× 1000）。

（J）慢粒患者早期原始细胞危象的外周血涂片。注意图中的四个原粒细胞和杆状、分叶、嗜碱及有核红细胞（瑞氏 - 姬姆萨染色，× 1000）。

（K）ET 患者骨髓片示其巨核细胞增加（瑞氏 - 姬姆萨染色，× 400）。

（L）ET 患者的外周血涂片示循环中的巨大的不典型血小板周围的巨核细胞碎片（瑞氏 - 姬姆萨染色，× 1000）。

（M）骨髓纤维化患者的骨髓穿刺片，可见明显的纤维组织增生（胶原蓝染）伴残留的骨髓成分（红染）（三色（Trichrome）染色，× 100）。

（N）图 M 患者的骨髓活检示原胶原纤维明显增加（网状组织染色，× 1000）。

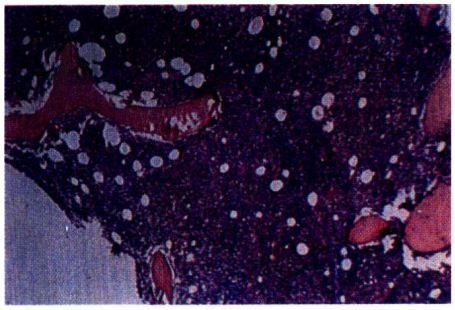
（O）骨髓纤维化患者的肝脏活检示髓外造血，肝窦内可见明显的多个巨核、红系、粒系细胞（HE 染色，× 100）。

（P）图 O 高倍镜下显示肝窦内的几个巨核细胞和其他的骨髓成分（HE 染色，× 1000）。

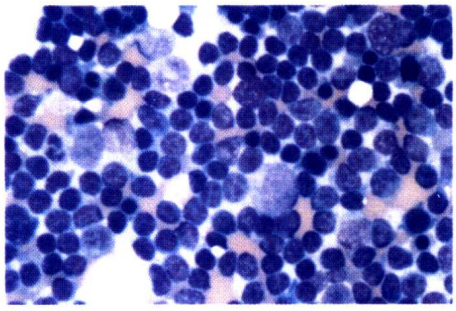
（Q）图 O 患者的肝活检，经用免疫过氧化酶对溶酶体染色显示粒细胞的存在（× 400）。

（R）骨髓纤维化患者的外周血涂片，可见泪滴状红细胞（瑞氏 - 姬姆萨染色，× 1000）。

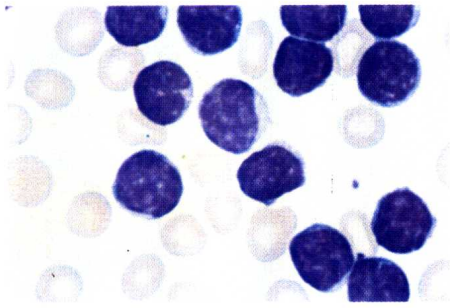
（S）MPD 患者的外周血涂片示获得性 Pelger-Huët 缺陷，其特点是多形核白细胞的核内有小碎片。



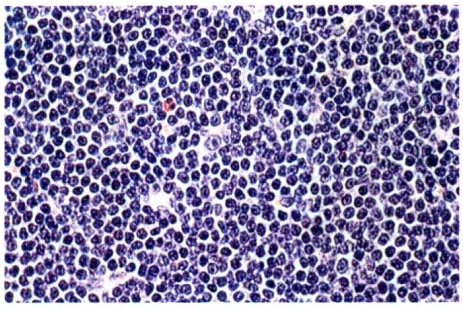
图版 3-2A



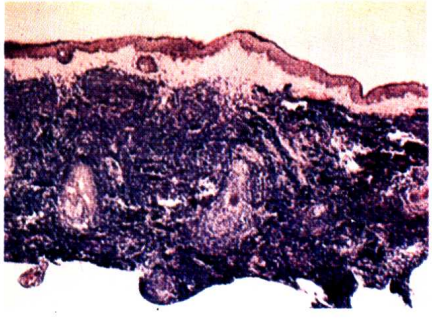
图版 3-2B



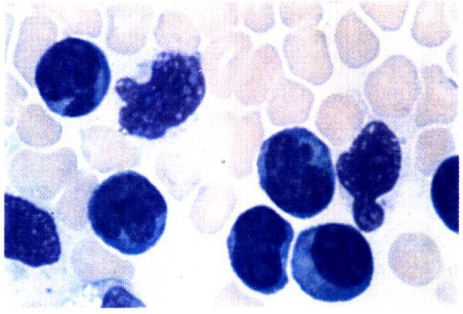
图版 3-2C



图版 3-2D



图版 3-2E



图版 3-2F

图版 3-2

(A) 慢淋患者的骨髓穿刺标本示广泛地被分化良好的小淋巴细胞取代（HE 染色，× 40）。

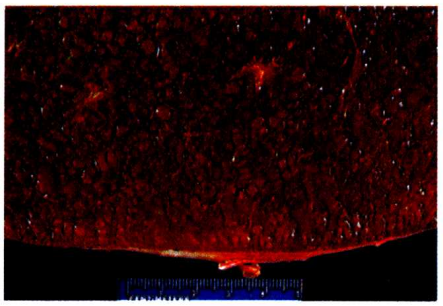
(B) 慢淋患者的骨髓穿刺涂片示以成熟的小淋巴细胞为主，注意深染的块状核染色质（瑞氏－姬姆萨染色，× 450）。

(C) 图B的外周血涂片示分化良好的单一的淋巴细胞群。细胞边缘有淡蓝色的胞浆（瑞氏－姬姆萨染色，× 1000）。

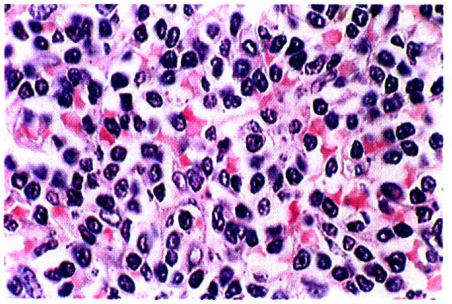
(D) 慢淋患者淋巴经活检示其广泛地被分化良好的小淋巴细胞取代（HE 染色，× 400）。

(E) T-CLL 患者的皮肤活检示小淋巴细胞广泛浸润（HE 染色，× 40）。

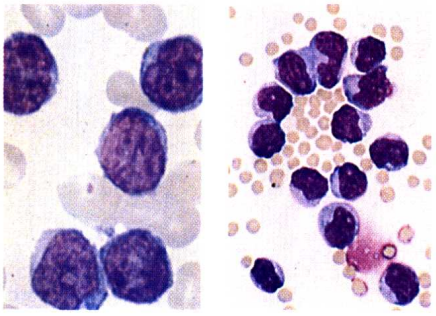
(F) 图E外周血涂片示成熟的小淋巴细胞，核不规则，许多核有切迹或卷曲（瑞氏－姬姆萨染色，× 1000）。



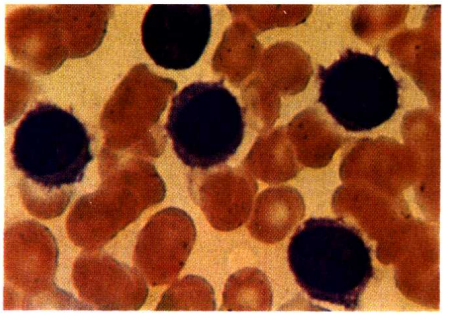
图版 3-2G



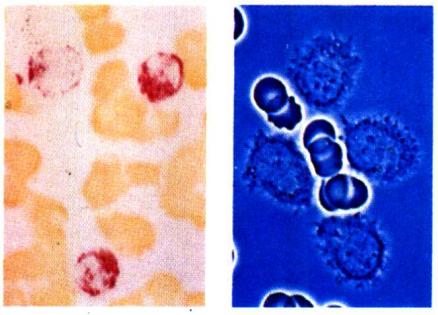
图版 3-2H



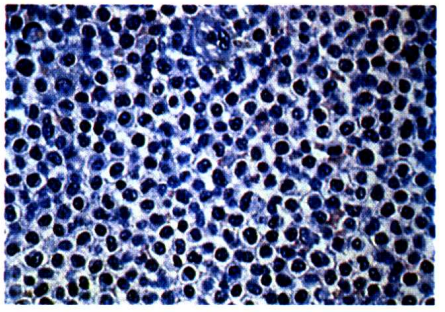
图版 3-2I



图版 3-2J



图版 3-2K



图版 3-2L

图版 3-2（续）

(G) 低倍镜下观察幼淋细胞白血病患者的脾切片，示脾小结被广泛浸润。

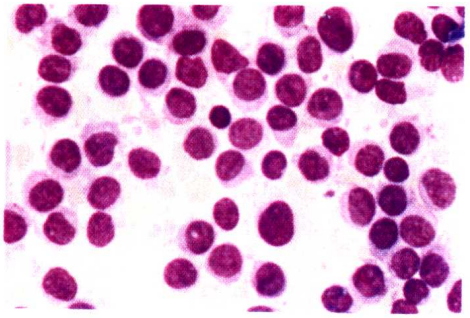
(H) 图版G的脾切片示有广泛的中等大小的不成熟的细胞浸润，部分有明显的核仁(HE染色，×400)。

(I) 图版 G 患者的脾涂片（右方格）显示中等大小的淋巴细胞，具丰富的胞浆，核染色质浓缩，核仁明显（×400）。左方格示外周血内有同样的细胞（瑞氏－姬姆萨染色，×1000）。

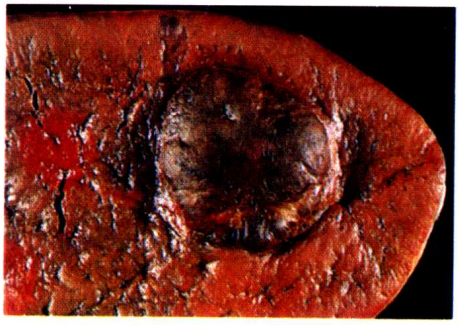
(J) 毛细胞白血病患者的外周血涂片示有特征性的丝样或毛状细胞质突起，其细胞内仅有少量的胞质（瑞氏－姬姆萨染色，×1000）。

(K) 耐酒石酸在磷酸染色(左方格)中毛细胞为阳性，右方格示相差显微镜下见到的毛细胞(×1000)。

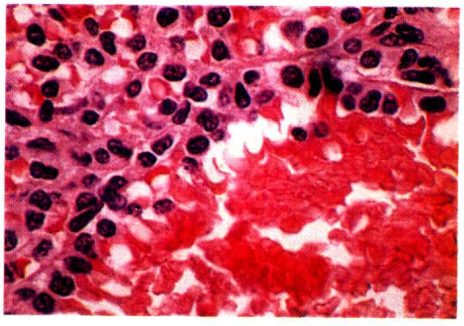
(L) 毛细胞白血病患者的骨髓活检示有广泛的毛细胞浸润。



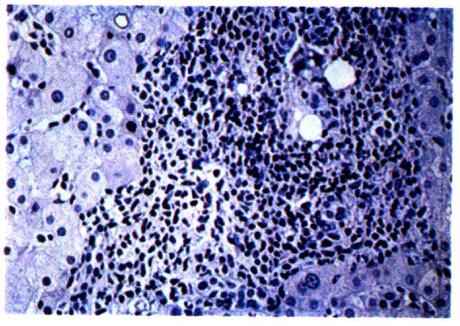
图版 3-2M



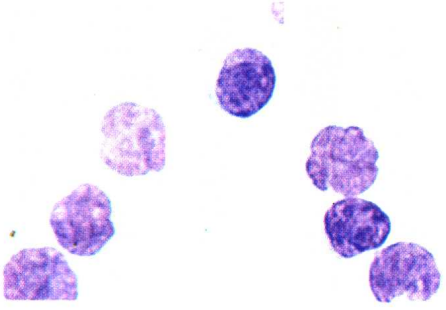
图版 3-2N



图版 3-2O



图版 3-2P



图版 3-2Q

图版 3-2（续）

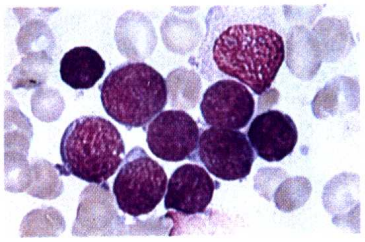
（M）图版 L 患者的骨髓涂片示多个毛细胞，其特点是核圆、卵圆或豆形，部分有核仁，中量的细胞质。核偏心，可能与制片过程人为所致有关。背景可见细胞质的碎片，骨髓穿刺涂片不易见到毛状突起（瑞氏－姬姆萨染色，× 450）。

（N）毛细胞白血病患者的脾切片示间隙充满红细胞形成血湖（blood lake）。

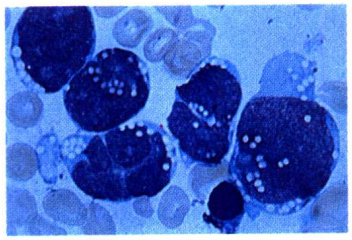
（O）显微镜下图版 N 的血湖边缘，红细胞湖内含有成行的新生毛细胞。正常情况下脾窦内的内皮细胞成行排列。在红髓内亦可见背景内有多量的毛细胞（HE 染色，× 1000）。

（P）毛细胞白血病患者的肝活检显示像脾窦一样，肝门周围亦有明显的肿瘤细胞浸润（HE染色，× 400）。

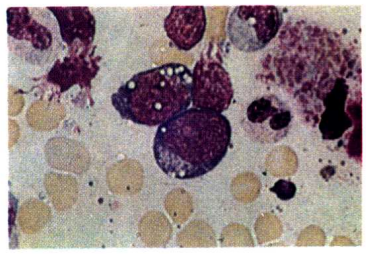
（Q）Sézary 综合征患者外周血涂片示其小淋巴细胞核不规划或核卷曲（瑞氏－姬姆萨染色，× 1000）。



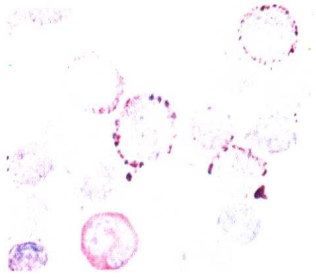
图版 13-1



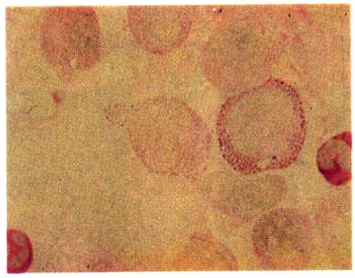
图版 13-2



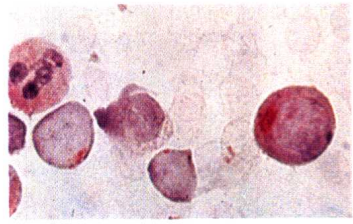
图版 13-3



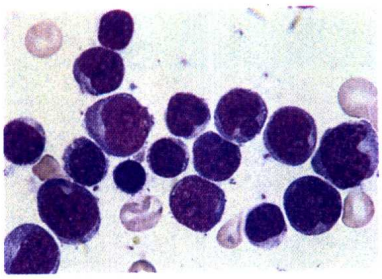
图版 13-4



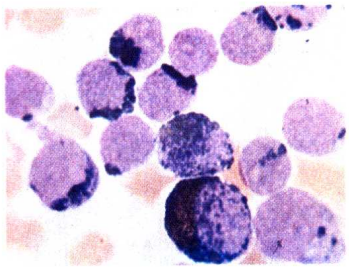
图版 13-5



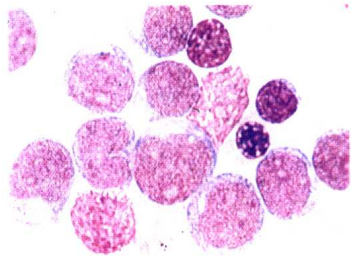
图版 13-6



图版 13-7



图版 13-8



图版 13-9

图版 13-1　ALL-L1：核浆比例高、核膜规则。
图版 13-2　ALL-L2：胞浆比 L1 多、核仁明显。
图版 13-3　ALL-L3：2 个 Burkitt 细胞伴嗜碱性细胞质。
图版 13-4　ALL 中原始淋巴细胞的 PAS 反应：注意其块状颗粒和散在背景中的中粒细胞的颜色。
图版 13-5　AMML 中原粒单细胞的 PAS 反应：注意其粗大的颗粒。
图版 13-6　T 细胞型，酸性磷酸酶反应。
图版 13-7　I 型原始细胞（无颗粒），具淋巴细胞样的形态。
图版 13-8　AML 中的过氧化物酶反应，可显示 Auer 小体的存在。
图版 13-9　AML（FABM1 型）与图版 13-8 为同一患者，注意其原始细胞分化很差且有明显的核仁。