Infection and the Perioperative Period

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GRUNE & STRATTON
A Subsidiary of Harcourt Brace Jovanovich, Publishers
New York London
Paris San Diego San Francisco São Paulo
Sydney Tokyo Toronto

Library of Congress Cataloging in Publication Data
Main entry under title:

Infection and the perioperative period.

Bibliography
Includes index.
1. Surgical wound infections. 2. Nosocomial infections. I. Mathieu, Alix. II. Burke, John Francis, 1922– . [DNLM: 1. Cross infection. 2. Preoperative care. 3. Intraoperative care. 4. Postoperative complications. WO 184 I43]
RD98.3.I5 617'.01 81-20148
ISBN 0-8089-1325-5 AACR2

Grune & Stratton, Inc.
111 Fifth Avenue
New York, New York 10003

Distributed in the United Kingdom by
Academic Press Inc. (London) Ltd.
24/28 Oval Road, London NW 1

Library of Congress Catalog Number 81-20148
International Standard Book Number 0-8089-1325-5

Printed in the United States of America

Acknowledgment

Alix Mathieu wishes to express his deepest gratitude and special thanks to Drs. Richard J. Kitz and Philipp O. Bridenbaugh for their constant leadership and encouragement.

Both editors are grateful to all their collaborators, as well to their secretarial and technical personnel, who have so actively and effectively participated in this venture.

Foreword

Nosocomial infections can be traced to the perioperative period in more than one of every 20 patients undergoing surgery. Not all of the infections, however, are related to the surgical wound, and many occur as a result of urinary tract catheterization, invasive intravascular cannulation, or tracheal intubation.

It is timely for anesthesiologists and surgeons to combine their efforts to examine perioperative infections since both types of specialists not only contribute to the problem but, through the application of presently available knowledge, may markedly diminish perioperative sepsis. Drs. Mathieu and Burke have done an admirable job in selecting topics and authors for this contributed text, and their concise editorial abilities have been combined to prevent significant overlap of the topics. This book provides a broad base of examination of the pathophysiologic mechanisms involved in anesthesia and surgery on the development of infection, and it also supplies practical guidelines for both prevention and management of infections. Division of the text into preoperative, intraoperative, and postoperative periods focuses on practical considerations which will be useful for all students of surgery and anesthesiology, including practicing physicians and those in training. The symbiosis generated by the combined efforts of anesthesiologists and surgeons provides a clearer focus on the problems of perioperative infections that has not been readily available in previous texts.

Infection and the Perioperative Period should be a welcomed addition to the personal libraries of anyone concerned with the problem of surgical infections.

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Preface

The rapid expansion of knowledge in the field of infection has made it difficult for anesthesiologists to keep abreast of all information related to the development and spread of infection and its clinical implications relevant to the management of the infectious patient in the perioperative period. This book is an attempt to present the many aspects of infection that would be of clinical importance to anesthesiologists and surgeons. Our goal is not to produce a detailed text on the mechanisms, pathophysiology, and treatment of infection; it is, instead, to present the clinical considerations raised by the infected patient in need of a surgical intervention, to review the basic facts upon which clinical understanding can be based, and to consider the alternative methods of treatment applicable for optimal patient care.

It is clear to us that we are just beginning to explore new and fascinating facets of an old problem, infection. Not all questions have been answered since this area is one in which rapid progress continues, and this makes the understanding of the processes that govern the choice and administration of anesthetics in infected patients all the more challenging. This book is designed primarily for both students and practicing anesthesiologists, but is also intended for any specialist involved in the preparation or care of patients in the perioperative period.

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PART I

Preoperative Considerations

J. Kelly Smith

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Mechanisms of Host Defense against Infection: Aging and Immunity

Our understanding of the mechanisms of host resistance has expanded enormously in the past several decades. It now appears that host defenses not only play a role in combating infection[1] but also contribute in some manner to the control or development of a diverse group of illnesses, including allergic and hypersensitivity diseases,[2] cancer,[3] and collagen-vascular diseases.[1] Immune mechanisms may also be involved in the basic processes of aging.[4,5]

This chapter will review what is currently known about the contributions of the integument, the inflammatory response, lymphocyte-mediated immunity, and cyclic nucleotides to host defenses. It will also review the manner in which disease and therapy may affect these defenses.

THE INTEGUMENT

The integument provides the initial and, in many respects, the most important barrier to microbial invasion. In addition to physically protecting the internal environment, this largest of all organs performs a surprising variety of metabolic functions,[6] some of which contribute to host defense. These functions include the production of: sweat and sebum, which provide a thin mantle of protection for the skin; mucus, the protective blanket of mucosal surfaces; and secretory component, the transporter and stabilizer of immunoglobulin A (IgA). Even gastric secretions perform a protective role. Furthermore, the microstructure of epithelial cell membranes appears to discourage intimate association with many potentially harmful microorganisms.

Assisting epithelial cells is a network of glandular tissues and lymphocytes

which bathe the integument with protective digestive juices and secretory immunoglobulins.

The following discussion focuses on the role of integumental surface receptors and surface secretions in host defense.

Surface Receptors

Early in this century Paul Erhlich proclaimed that *corpora non agunt nici fixata,* (substances do not act unless attached). The significance of this statement, to which there have been few exceptions in science, has only recently been appreciated by researchers interested in the pathogenesis of infection. As Erhlich predicted, it now appears that the ability of bacteria to invade and colonize intact integument may depend heavily on their ability to bind to receptor sites located on the epidermal or mucosal cell walls.

In the skin, the keratinized layer of the epidermis, which is comprised of nonviable cells of little nutritional value to most bacteria,[6] has receptor sites for a restricted variety of ordinarily, nonpathogenic organisms, including *Proprionibacterium acnes* and *Staphylococcus epidermidis*. Although other microorganisms are also capable of colonizing the skin, they serve only as transient, short-term flora, partly because of their lack of attachment. Vigorous handwashing will remove unattached organisms, but will have little effect on the resident bacterial flora of the skin.[7]

Similar restrictions apply to intact mucous membranes. *Neisseria gonorrhoeae,* for example, do not invade intact mucous membrane unless they can first attach to epithelial surfaces by means of their pili.[8,9] Enterotoxigenic *Escherichia coli* produce a protein colonizing factor which enhances their attachment to mucosal epithelial cells in vitro.[10] Bacteria lacking this factor are relatively nonvirulent for volunteers and do not attach readily to mucosal cells.[11] In addition, intestinal epithelial cells have gangliosides in their cellular membrane which act as receptors for cholera and *E. Coli* enterotoxins.[12,13] These toxins are ineffective unless attached.

Surface attachment may be discouraged even in the presence of receptor sites. One explanation for the protective effect of secretory IgA, for example, is that the immunoglobulin coating may block bacterial receptors, thereby preventing microbial adherence to mucous surfaces. Thus, Fubara and Freter[14] have shown that *Vibrio cholerae* when coated with specific anticholera antibody do not adhere to the mucous membrane of the ileum, and Williams and Gibbons[15] have demonstrated a similar inability of antibody-coated *Streptococcus sanguis* to adhere to buccal mucosal cells. Also in this regard, Plaut et al[16,17] have shown that *Neisseria meningitides, Neisseria gonorrhoeae* and *Streptococcus sanguis* produce an extracellular protease that cleaves one form of IgA (IgA_1) into Fab and Fc fragments, which results in loss of functional activity of the antibodies studied so far.