SURGERY ANNUAL: NINETEEN EIGHTY

series editor LLOYD M. NYHUS

SURGERY ANNUAL

Volume 12

1980

Series Editor

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80 81 82 83 84/10 9 8 7 6 5 4 3 2 1

Library of Congress Catalog Card Number: 69-18093

Prentice-Hall International, Inc., London
Prentice-Hall of Australia, Pty. Ltd., Sydney
Prentice-Hall of India Private Limited, New Delhi
Prentice-Hall of Japan, Inc., Tokyo
Prentice-Hall of Southeast Asia (Pte.) Ltd., Singapore
Whitehall Books Ltd., Wellington, New Zealand

PRINTED IN THE UNITED STATES OF AMERICA
0-8385-8713-5

SURGERY ANNUAL

1980

APPLETON-CENTURY-CROFTS/New York

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PREFACE

This volume maintains the aim of the *Surgery Annual* series: to provide the surgeon with access to critical discussion of recent advances and modifications in the clinical management of surgical diseases and to present new developments in the basic sciences as they relate to the practice of surgery. In-depth discussion of these topics is not readily available in other publications.

We have continued the practice of presenting the work of several surgeons from outside the United States, making the series one of worldwide interest.

Surgeons treating patients who have undergone trauma will be particularly interested in *Surgery Annual 1980.* Included are "Human Host Defense Mechanisms as They Relate to Surgery and Trauma" by Thomas M. Saba and associates from Albany Medical College and "Management of Complex Vascular Injuries to the Extremities" by Leonardo T. Lim and his colleagues from Cook County Hospital. Also included are chapters on hepatic trauma by E. Truman Mays, splenic trauma by Jay L. Grosfeld and Mark A. Malangoni, fat embolism syndrome by Michael R. Shier and Robert F. Wilson, and acute head injury by E. Stephen Gurdjian and Edwin S. Gurdjian.

The editor expresses his appreciation to the Editorial Advisory Board; to the contributors to *Surgery Annual 1980;* to Mr. David Stires, General Manager, and Ms. Laurie Wilkowski, Production Editor, Appleton-Century-Crofts; and to Ms. Catherine Judge, Publications Editor, Department of Surgery, University of Illinois at the Medical Center.

LLOYD M. NYHUS, M.D.

SURGERY ANNUAL

1980

CONTENTS

HUMAN HOST DEFENSE MECHANISMS AS THEY RELATE TO SURGERY AND TRAUMA

THOMAS M. SABA, WILLIAM A. SCOVILL, AND SAMUEL R. POWERS, JR.

CONCEPT OF MULTIPLE ORGAN FAILURE

The predominant cause of late mortality in trauma patients without head injury, after initial successful resuscitation, appears to be progressive organ failure occurring in association with septic complications. Renal failure commonly encountered 15 to 20 years ago is now rare because of improved techniques of fluid management after severe injury.[62] With increasing duration of patient survival, hepatic failure has been more recently recognized, but extensive study of the liver in the trauma patient has been limited.[83] Abnormalities in pulmonary function, cardiovascular stability, peripheral hemodynamics, and oxygen utilization have been the recent focus of study in trauma patients at Albany Medical College. While the injured patient in whom sepsis develops after extensive multiple systems trauma often demonstrates the clinical pattern of organ failure,[33] the severely burned patient with primarily cutaneous injury may also undergo the full spectrum of organ failure in conjunction with systemic bacteremia. Since the burn patient often lacks direct organ trauma, has predictable multiple episodes of systemic bacteremia, and may demonstrate the full spectrum of progressive organ failure, these patients may provide an ideal model to assess the influence of septic complications on progressive organ failure. Moreover, a comparison of burn, trauma, and surgical patients should provide valuable insights into the pathophysiology of disturbed organ function after injury, especially as influenced by septicemia.

Since edema is a frequent finding, in the periphery as well as the lung, diffuse systemic capillary leaking may be a pivotal event in the etiology of posttraumatic pulmonary and cardiovascular failure. Increased pulmonary vascular

This study was supported by NIH Grants GM-21447, CA-16011, and GM-15426.

permeability may be a cause of lung edema during sepsis after trauma.[39] Furthermore, experimental studies on fluid exchange across the lung revealed increased pulmonary vascular permeability during bacteremia.[21,22] The mechanism responsible for the apparently increased vascular permeability in both the lung and the periphery remains to be determined and warrants investigation. While most authors concur that alterations in hydrostatic and oncotic pressures are central to the development of interstitial edema,[91] it has been shown that pulmonary edema in the posttraumatic setting will occur in the absence of left heart failure. Moreover, changes in oncotic pressures during hemodilution cardiopulmonary bypass do not correlate with any change in alveolar-arterial 0_2 gradient.[50] Other proposed mechanisms for the apparently increased pulmonary, peripheral vascular, and lymphatic permeability during bacteremia after major injury include the local action on the microcirculation of such factors as endotoxin, live bacteria, complement, and/or immune complexes.[19,22,24,68,73,75,88] For example, the circulation of immune complexes during prolonged bacteremia may fix complement and induce an increased capillary permeability, leading to the development of pulmonary interstitial edema and altered gas exchange. Furthermore, gram-negative sepsis has been demonstrated experimentally to activate the coagulation cascade, and intravascular coagulation can lead to pulmonary changes similar to those observed during the respiratory distress syndrome.[12,13,16,17,51] Finally, pulmonary interstitial edema caused by leukostasis in the pulmonary vasculature has been shown during hemodialysis[24] and may represent another mechanism of pulmonary injury in the trauma patient.

Thus, it is possible that circulating particles, which might include bacteria, immune complexes, endotoxin, fibrin microaggregates, collagenous debris, injured platelets, and altered leukocytes, may be involved in the pathogenesis of multiple organ dysfunction. From the perspective of this article, it is apparent that these factors are cleared by the reticuloendothelial system (RES), especially the Kupffer cells of the liver and the RE cells of the spleen.[11,15,45,46,73,75,80] These observations, coupled with recent documentation of humoral and/or cellular dysfunction[42,49,69,71,74,75,89,90] in traumatic, burn, ischemic, and hemorrhagic shock and following major surgery, suggest a novel concept of the pathogenesis of multiple organ failure during bacteremia associated with reticuloendothelial failure. Systemic RES host defense depression following combined injuries and shock may be etiologic in the genesis of multiple organ dysfunction.[76,77,88]

Numerous findings have suggested the involvement of the reticuloendothelial system in host defense following traumatic injury.[75] Patients with apparently comparable injuries manifest distinct variations in cardiac, metabolic, pulmonary, and microcirculatory disturbances that cannot be explained by classic cardiopulmonary and hematologic criteria. Disturbances of Kupffer cells of the RES mediated by a nonimmunoglobulin opsonic deficiency may be a potential factor in the genesis of multiple organ failure following various forms of injury.[88-90] In this regard, a delicate balance may exist between RES function and the circulating level of blood-borne particles. The syndrome of multiple organ failure with septicemia following trauma may reflect this imbalance. This could contribute to microvascular embolization, endothelial injury, altered permeability, interstitial edema, and altered organ function.

RETICULOENDOTHELIAL FUNCTION AND SYSTEMIC DEFENSE

Reticuloendothelial (RE) participation in resistance to severe shock and trauma is not a new concept.[5,6,9,32,69,71,75,80,84,99,100] However, only recently has experimental evidence been available which may explain the etiology of this RE failure. Moreover, an effective therapeutic means to circumvent RE failure in the clinical setting remains to be developed. Additionally, the colloid clearance technique[11,15,78,82,86] to monitor the RES in vivo has major limitations in terms of routine clinical applicability because of its invasive nature and ability to compromise RES function.[20,73,78] Thus, the technical capability both to increase Kupffer cell activity and to evaluate Kupffer cell phagocytic capacity in the clinical setting would have major clinical significance. The active role by the RES of the liver and spleen in antibacterial[66] and antitumor immunity,[8,25,27] coupled with the importance of the systemic defense in the removal of blood-borne microthrombi, injured cells, and microaggregates,[27,34-36,46,73] emphasizes the need to understand the physiology and physiopathology of this RES systemic host defense mechanism, especially following injury. This review will summarize the effect of injury on the RES in animals and man, with special emphasis on the Kupffer cell phagocytic activity. While a variety of factors could either independently or collectively alter this clearance process, emphasis will be placed on a humoral opsonic deficiency in the etiology of postinjury RE failure, as documented extensively from this laboratory.

The blood level of a specific, large molecular weight protein, opsonic α_2 surface binding (SB) glycoprotein,[18-20,76,77] has been shown to modulate RES clearance of blood-borne foreign or denatured nonbacterial particulate matter, such as test colloids, fibrin aggregates, and collagenous debris. This protein has been purified from animals[4,18,20] and humans[19,76,77] and can now be measured by bioassay as well as electroimmunoassay.[18-20,76-78] Decline in the plasma level of this protein after trauma will result in RES depression, especially Kupffer cell phagocytic impairment, and restoration of opsonin levels in the later postinjury period is correlated with RES recovery.[20,44,69,70,72,74] RE blockade induced by intravenous injection of particulate matter is caused by an acute depletion of opsonic α_2 SB glycoprotein,[20,77,78] and the intravenous injection of purified opsonin will reverse opsonic deficiency after colloid-induced RES blockade.[4,75] This observation is of central importance to the concept of RE function and resistance to trauma, since RE blockade induced by colloid injection[11,78] will increase susceptibility to traumatic, hemorrhagic, tourniquet, septic, and endotoxic shock.[9,73,99,100] In addition, RE blockade will negate experimentally induced adaptive tolerance to trauma. These experimental observations are coupled with specific documentation of opsonic α_2 SB glycoprotein deficiency in patients who have had elective operations,[26,90] burns, or blunt trauma,[76,88,89] especially during bacteremia and associated with signs of organ failure, such as pulmonary insufficiency.[77,88]

Such findings have led us to hypothesize[76,77] that severe postoperative and posttraumatic hepatic Kupffer cell failure undermines the nonspecific systemic defense and that a deficiency of opsonic α_2 SB glycoprotein is a major contributing factor in the genesis of such RES dysfunction. Several studies have highlighted the increment in extrahepatic and extrasplenic localization of blood-borne