



1981
YEAR BOOK OF
SURGERY.

SCHWARTZ
NAJARIAN / PEACOCK
SHIRES / SILEN / SPENCER

The YEAR BOOK of

Surgery

1981

Editor

SEYMOUR I. SCHWARTZ, M.D.

*Professor of Surgery, University of Rochester
School of Medicine and Dentistry*

Associate Editors

JOHN S. NAJARIAN, M.D.

*Professor and Chairman, Department of Surgery,
University of Minnesota Medical School*

ERLE E. PEACOCK, Jr., M.D.

*Professor of Surgery,
Tulane University School of Medicine*

TOM SHIRES, M.D.

*Professor and Chairman, Department of Surgery,
New York Hospital—Cornell Medical Center*

WILLIAM SILEN, M.D.

*Johnson and Johnson Professor of Surgery, Harvard Medical School;
Surgeon-in-Chief, Beth Israel Hospital, Boston*

FRANK C. SPENCER, M.D.

*George David Stewart Professor of Surgery; Chairman,
Department of Surgery, New York University; Director,
Department of Surgery, New York University Medical Center
and Bellevue Hospital*

YEAR BOOK MEDICAL PUBLISHERS, INC.

CHICAGO • LONDON

Copyright © August 1981 by YEAR BOOK MEDICAL PUBLISHERS, INC.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without prior written permission from the publisher.

Printed in U.S.A.

Library of Congress Catalog Card Number: CD38-26

International Standard Book Number: 0-8151-7620-1

Table of Contents

The material covered in this volume represents literature reviewed up to December 1980.

ANNUAL OVERVIEW	7
1. GENERAL CONSIDERATIONS	37
2. FLUID, ELECTROLYTES, AND NUTRITION	45
3. SHOCK	67
4. TRAUMA	81
5. WOUND HEALING	109
6. INFECTIONS	125
7. BURNS	133
8. TRANSPLANTATION AND ARTIFICIAL ORGANS	143
9. ONCOLOGY AND TUMOR IMMUNOLOGY	183
10. SKIN, SUBCUTANEOUS TISSUE, AND HAND	199
11. THE BREAST	209
12. HEAD AND NECK	221
13. THORAX	231
14. CONGENITAL HEART DISEASE	249
15. VALVULAR HEART DISEASE	259
16. CORONARY HEART DISEASE	271
17. MISCELLANEOUS CARDIAC DISEASE	279
18. ARTERIES	299
19. VEINS AND LYMPHATICS	331
20. ESOPHAGUS	341
21. STOMACH AND DUODENUM	357
22. SMALL INTESTINE	373
23. COLON AND RECTUM	383
24. LIVER AND SPLEEN	415
25. BILIARY TRACT	435
26. PANCREAS	459
27. ENDOCRINE GLANDS	469



THE 1981 YEAR BOOKS

The YEAR BOOK series provides in condensed form the essence of the best of the recent international medical literature. The material is selected by distinguished editors who critically review more than 500,000 journal articles each year.

Anesthesia: *Drs. Eckenhoff, Bart, Brunner, Cane and Linde.*

Cancer: *Drs. Clark, Cumley and Hickey.*

Cardiology: *Drs. Harvey, Kirkendall, Kirklin, Nadas, Resnekov and Sonnenblick.*

Clinical Pharmacy: *Dr. Woolley.*

Dentistry: *Drs. Hale, Hazen, Moyers, Redig, Robinson and Silverman.*

Dermatology: *Drs. Dobson and Thiers.*

Diagnostic Radiology: *Drs. Whitehouse, Adams, Bookstein, Gabrielsen, Holt, Martel, Silver and Thornbury.*

Drug Therapy: *Drs. Hollister and Lasagna.*

Emergency Medicine: *Dr. Wagner.*

Endocrinology: *Drs. Schwartz and Ryan.*

Family Practice: *Dr. Rakel.*

Medicine: *Drs. Rogers, Des Prez, Cline, Braunwald, Greenberger, Bondy and Epstein.*

Neurology and Neurosurgery: *Drs. De Jong and Sugar.*

Nuclear Medicine: *Drs. Hoffer, Gottschalk and Zaret.*

Obstetrics and Gynecology: *Drs. Pitkin and Zlatnik.*

Ophthalmology: *Dr. Hughes.*

Orthopedics: *Dr. Coventry.*

Otolaryngology: *Drs. Paparella and Strong.*

Pathology and Clinical Pathology: *Dr. Brinkhous.*

Pediatrics: *Drs. Oski and Stockman.*

Plastic and Reconstructive Surgery: *Drs. McCoy, Brauer, Haynes, Hoehn, Miller and Whitaker.*

Psychiatry and Applied Mental Health: *Drs. Freedman, Friedhoff, Kolb, Lourie, Nemiah and Romano.*

Sports Medicine: *Col. Anderson, Mr. George, Drs. Krakauer, Shephard and Torg.*

Surgery: *Drs. Schwartz, Najarian, Peacock, Shires, Silen and Spencer.*

Urology: *Drs. Gillenwater and Howards.*

The YEAR BOOK of

Surgery

1981

Editor

SEYMOUR I. SCHWARTZ, M.D.

*Professor of Surgery, University of Rochester
School of Medicine and Dentistry*

Associate Editors

JOHN S. NAJARIAN, M.D.

*Professor and Chairman, Department of Surgery,
University of Minnesota Medical School*

ERLE E. PEACOCK, Jr., M.D.

*Professor of Surgery,
Tulane University School of Medicine*

TOM SHIRES, M.D.

*Professor and Chairman, Department of Surgery,
New York Hospital—Cornell Medical Center*

WILLIAM SILEN, M.D.

*Johnson and Johnson Professor of Surgery, Harvard Medical School;
Surgeon-in-Chief, Beth Israel Hospital, Boston*

FRANK C. SPENCER, M.D.

*George David Stewart Professor of Surgery; Chairman,
Department of Surgery, New York University; Director,
Department of Surgery, New York University Medical Center
and Bellevue Hospital*

YEAR BOOK MEDICAL PUBLISHERS, INC.

CHICAGO • LONDON

Copyright © August 1981 by YEAR BOOK MEDICAL PUBLISHERS, INC.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without prior written permission from the publisher.

Printed in U.S.A.

Library of Congress Catalog Card Number: CD38-26

International Standard Book Number: 0-8151-7620-1

Table of Contents

The material covered in this volume represents literature reviewed up to December 1980.

ANNUAL OVERVIEW	7
1. GENERAL CONSIDERATIONS	37
2. FLUID, ELECTROLYTES, AND NUTRITION	45
3. SHOCK	67
4. TRAUMA	81
5. WOUND HEALING	109
6. INFECTIONS	125
7. BURNS	133
8. TRANSPLANTATION AND ARTIFICIAL ORGANS	143
9. ONCOLOGY AND TUMOR IMMUNOLOGY	183
10. SKIN, SUBCUTANEOUS TISSUE, AND HAND	199
11. THE BREAST	209
12. HEAD AND NECK	221
13. THORAX	231
14. CONGENITAL HEART DISEASE	249
15. VALVULAR HEART DISEASE	259
16. CORONARY HEART DISEASE	271
17. MISCELLANEOUS CARDIAC DISEASE	279
18. ARTERIES	299
19. VEINS AND LYMPHATICS	331
20. ESOPHAGUS	341
21. STOMACH AND DUODENUM	357
22. SMALL INTESTINE	373
23. COLON AND RECTUM	383
24. LIVER AND SPLEEN	415
25. BILIARY TRACT	435
26. PANCREAS	459
27. ENDOCRINE GLANDS	469

Annual Overview

General Considerations—At a time when the surgeon deals increasingly with the elderly patient, it is important to have relevant data available. Recent studies have emphasized that surgical risks in the elderly are acceptable and that chronological age should not be a deterrent to a necessary operation. When all abdominal operations are considered, there seems to be no statistically significant difference in the complication rate in patients under the age of 49 compared to those between ages 60 and 69 and those between ages 70 and 79.

The finding of heparin-associated thrombocytopenia recently has gained increased recognition. Beef lung heparin is indicted to a much greater extent than hog mucosa preparation; since thrombocytopenia during the first seven days of therapy is rare, it is realistic to monitor platelet counts only when heparin treatment continues beyond that period.

There is an increasing literature about transcatheter Gelfoam embolization in abdominal, retroperitoneal, and pelvic hemorrhage; reports reveal a high rate of control of bleeding.

Alcoholism, which represents a growing problem in our culture, requires surgical consideration during withdrawal symptoms. It is suggested that alcohol be given to all alcoholics in doses of 10 to 15 ml hourly when illness, injury, or an operation precipitates sudden withdrawal. The syndrome of gastroparesis that occurs with diabetes mellitus or after a gastric operation has been studied under controlled conditions; beneficial effects of metoclopramide and bethanechol have been demonstrated. The effects of antacid versus cimetidine to prevent acute gastrointestinal bleeding have been assessed. In a randomized trial conducted on 75 critically ill patients, cimetidine was shown to be ineffective in consistently elevating the gastric pH above 3.5, and was less effective than the antacid in providing adequate prophylaxis against acute gastrointestinal bleeding.

Fluid, Electrolytes, and Nutrition—A monumental amount of work is progressing in laboratories around the world to study the cause of the negative nitrogen balance and breakdown of tissues that follow serious injury and infection. Various studies reported during the past year confirm that the negative nitrogen balance in severe trauma is due to increased breakdown primarily in muscle protein. Furthermore, significant alterations in muscle protein turnover have been produced in a variety of studies with the use of different forms of amino acids. It would appear that the changes in protein synthetic rate or catabolic rate may depend on very complex reactions between the various organ systems depending on the type of trauma, the se-

verity of the injury, the presence of sepsis, the nutritional intake, and the composition of the nutritional intake. Several groups have demonstrated during the past year that branch-chain amino acids seem to be preferable substrate for correction of the amino acid losses in the posttraumatic catabolic state. In several studies the branch chain amino acids seem to produce a lower nitrogen loss than that which occurred in animals that had received specific amino acids. This apparently results from a combination of branch-chain amino acids serving as an energy source for muscle and as a substrate for alanine production and gluconeogenesis. It would also appear that the branch-chain amino acids tend to block amino acid efflux from muscle after injury.

There is little question that the use of intravenously administered fat is a practical technique for protein sparing in the animal or patient after trauma. Whether this nitrogen-sparing effect of fat in addition to replacement of amino acids is a desirable one has been examined by several studies. Most studies indicate that the combination of amino acids and glucose had a better nitrogen-conserving effect than amino acids and fat as the intravenous therapy in animals. The exact proportions and time for using fat as a nitrogen-sparing substrate will probably be determined by the severity of injury, the presence of sepsis, and the catabolic state of the entire animal or man.

Some very elegant studies available this year state that both extracellular and intracellular patterns of changes in metabolism, particularly in relation to amino acids, should receive increased attention. The specific forms of therapy involving glucose, amino acids, and fat are yet to be completely delineated in response to the many variables of injury and infection. These sophisticated studies are being pursued by several different groups; certainly, in the near future, repletion of the depleted body composition, including carbohydrate and fat, will be based on a far more rational basis than ever before.

Several papers concern classic hyperalimentation and its effects on a variety of responses, such as acute renal failure, the generation of specific enzymes, including 2,3-diphosphoglycerate, and effects on food utilization and gas exchange in patients who are hypermetabolic as a result of injury or infection. Once again, the preliminary and sophisticated data indicate a very useful role for total parenteral nutrition in patients in a catabolic state, such as is incurred by acute renal failure as well as by severe injury and infection. Additional articles recount some of the hazards of parenteral hyperalimentation. In the past, these have included many complications, such as sepsis, hyperosmolar coma, development of trace metal deficiencies, etc. An additional precaution that has appeared in the past 2 years has been unrecognized deficit of water in patients on hyperalimentation who develop extremely high serum sodium levels. The previously delineated caveats for careful monitoring of patients receiving hyperalimentation continue to be repeated.

Shock—The literature appearing during the past year relating to

all forms of shock has changed emphasis rather dramatically. The quality of the research and the level of sophistication of the investigations have improved perceptibly. As in the past, whereas all forms of shock are being studied, those receiving primary interest are still those seen most commonly in patients: hemorrhagic shock and septic shock.

A surprising number of papers appeared during the past year investigating different mechanisms of cellular response to shock injury. Many of these papers dealt with the specific function of mitochondrial injury in response to hypovolemic shock. Some new data were obtained indicating that the mitochondrion, an organelle that is dependent on oxygen for energy, would be expected to be one of the first cellular elements to exhibit injury in hypoxia. Some studies were reported to use cells *in vitro* in a tissue culture medium to demonstrate early mitochondrial injury.

Several elegant investigations reported during the past year indicated that shock is far more than regional or local hypoperfusion. The previously accepted definition of shock, too little blood flow per organ system per unit of time, is simply no longer sufficient. Although this definition is still true, it is also apparent that there are secondary effects to hypovolemic shock that occur quite rapidly and either cause or potentiate cellular injury. One study showed quite clearly that the impairment of oxygen consumption that occurs in hypovolemic animals can be transferred by cross perfusion into a normal animal. Obviously, this is not a shock-induced change in the recipient animal, since measurements of blood flow and catecholamine release and other parameters were not significantly changed by the cross perfusion.

Various types of mitochondrial failure have been studied. Some specific functions of hepatic mitochondria demonstrating oxidative phosphorylation uncoupling with subsequently impaired gluconeogenesis and hypoglycemia after hepatic glycogen storage depletion were presented. Multiple organs are being studied, and cellular and sub-cellular functions are now being identified specifically; this will explain long-observed biochemical and clinical phenomena in hypovolemic shock.

Further explanation of some of the changes in organ function and excitability in shock has been offered by a study revealing that after cellular injury with sodium influx into cells and potassium leak from cells, there is an alteration in hypovolemic shock in the interstitium itself. There is clearly trapping of potassium both in the total state as well as in the chemically active state during shock, which is reversibly released after therapy of shock. Elevations of interstitial potassium that are disproportionate to the amount returned to venous plasma during shock may well explain many of the metabolic, and particularly the cardiorespiratory effects observed during sustained hypovolemic shock. The level of cellular injury has also been examined in a number of other organs during the past year; this includes

a definition of the type of injury in metabolically active organs that occurs in both the liver and kidney.

Furthermore, the effects of new therapeutic agents with alteration of cellular function are appearing with increasing frequency. For example, far better definition of the cellular effects of glucagon have been intensively studied during the past year, and the use of prostaglandins in various fractions has been shown to have an unexplained benefit, particularly in long-term survival in animals in hypovolemic shock.

A new series of papers appeared this year on the use of albumin solutions in one form or another, usually human serum albumin, for resuscitation of patients in sustained hemorrhagic shock. It is clear from the patient studies that resuscitative shock and hemorrhagic shock need to be differentiated. Several fine papers describe the ideal resuscitative regimen for isovolemic resuscitation of patients, such as those undergoing a standardized but major elective operative procedure. It is interesting that the authors of three well-documented clinical studies concluded that for resuscitation for isovolemic hypovolemia, or isovolemic replacement as hypovolemia is incurred, the ideal replacement solutions do not include albumin. These rather elegant clinical studies show that optimal resuscitation was obtained with replacement by red blood cells and an extracellular fluid mimic in the form of Hartmann's solution. There was no question that if albumin as 5% solution was added to the resuscitative solution, the colloid osmotic pressure of the serum could be maintained. However, when the resuscitative regimen was aimed at maintaining a constant pulmonary capillary wedge pressure, or a left-sided atrial pressure, the reduction in colloid osmotic pressure of the plasma that occurred in the absence of albumin was physiologically inconsequential. It was also seen in these studies, however, that a rise in pulmonary capillary wedge pressure, easy to accomplish with minimal amounts of albumin-containing solution, quickly resulted in pulmonary insufficiency. The conclusions of several groups indicate that the maintenance of serum colloid osmotic pressure with albumin infusions during isovolemic replacement of blood loss is unnecessary and potentially harmful. These studies were accompanied by assessments of other practices, such as, in general, the probably largely unnecessary use of increasing amounts of normal human serum albumin in many hospitals.

Another set of clinical research papers looked further into the use of extra albumin during resuscitation from already sustained hemorrhagic shock in patients. The investigators indicate that albumin supplementation again maintained total serum protein and serum albumin at normal levels; however, despite this, the addition of albumin to resuscitative fluids during treatment of hemorrhagic shock had a remarkably negative inotropic effect on the heart.

Another good clinical study indicated that when supplemental albumin was added to a standard resuscitative regimen, including blood and saline solutions, the subsequent or postshock need for

whole blood replacement was increased because of the reduction in coagulation factors produced by the use of supplemental albumin in the initial resuscitative phase. This same group of investigators also found greater impairment of salt and water excretion after the use of supplemental albumin for resuscitation from hemorrhagic shock.

Multiple system organ failure is being delineated far more clearly than previously has been possible. Large clinical series of patients have now been identified and the very high incidence of systemic sepsis in the production of one or more organ failures after injury has been established. Many of these studies suggest that a common cellular insult may be the fundamental pathologic event, particularly in those patients with uncontrolled systemic infection. Alteration of white cell function, and therefore host resistance, in response to hypovolemic shock is now being investigated in some detail in many patients after trauma and hemorrhagic shock. While most of these studies have been concerned with changes in response to sepsis, burns, and general trauma, a few investigate the response to hypovolemic shock and indicate a very early and profound alteration in white blood cell function in this disorder.

Trauma—It is gratifying to see a sharp increase in the number of papers published this past year dealing in some detail with prehospital care.

An excellent article deals with the advantages of categorization of hospitals for receiving patients with life-threatening injury or illness. Patients in the six high-risk categories, including trauma patients, and also those with heart attacks, burns, spinal cord injury, neonatal distress, etc., should be taken to the closest and most appropriate facility as the primary guiding principle for pre-hospital care. More data are presented proving that survival is markedly improved when victims of serious injury are taken to trauma centers. Increased understanding is needed that categorization is not an attempt to limit hospital services, restrict progress, or downgrade facilities. It is, as the authors point out, an effort to show what is needed and to improve the quality of care in the high-risk patient. There are, of course, as a spin-off of categorization in a community, certain cost benefits achieved by the increased efficiency which is available with experienced staff and sophisticated equipment.

It is also gratifying to see documentation that active involvement of the lay public in emergency care systems leads to more optimal prehospital care for the sick and injured. Better definition is provided in several articles of the role of the lay person in prehospital care, including the level, probably dependent on community size, to which cardiopulmonary resuscitation (CPR) training should be made available. In certain large communities, CPR training will obviously be limited to certain high-risk areas. On the other hand, it may be made available to virtually all persons within the geographic area of smaller communities, the so-called saturation model of CPR training.

Several articles deal with specific extension of availability of emergency care as a geographically dictated phenomenon. For example,

one paper describes in great detail the epidemiology of trauma in a mountain area that essentially dictated the need and development of helicopter emergency air service. The success of such a trained team approach in this specific geographic setting is very impressive.

This year, as in recent years, the role of peritoneal lavage in the management of patients with abdominal trauma continues to be refined and evaluated. Its role in patients with blunt abdominal trauma seems to be very well established, whereas its place in the evaluation of patients with penetrating trauma whether due to gunshot wound or stab wound continues to be the subject of significant investigations.

One interesting paper this year has shown clearly the unreliability of peritoneal lavage in predicting the injury inflicted within the abdomen by gunshot wounds to the lower chest or upper abdomen with or without clinical signs of injury. The authors have done a careful study of over 300 patients and concluded that peritoneal lavage as a predictor of injury in the presence of gunshot wound was unsatisfactory. Further attempts to reduce the negative laparotomy rate in management of asymptomatic patients with stab wounds to the abdomen continue. It appears that in addition to its role in hemodynamically unstable patients, those with clinical signs of abdominal injury, and those in whom local exploration reveals penetrating injury to the abdomen, there may be a place for highly selected use of peritoneal lavage in evaluating asymptomatic patients with small penetrating injuries. One such study supports this procedure. However, other investigations indicate that with penetration in the asymptomatic patient, lavage does not have sufficient accuracy to determine immunity from laparotomy. In patients with blunt abdominal trauma and those in whom evaluation is not possible, such as in drug abuse or spinal cord injury, peritoneal lavage continues to be of inestimable value.

Further evidence was provided this year in support of emergency room thoracotomy in selected patients who appear to have had total circulatory arrest due to blunt or penetrating trauma just before arrival in the emergency department. The neurologic outcome from such a drastic approach has been evaluated carefully, and the great majority of patients so approached and resuscitated are free of significant neurologic sequelae. This technique bears further use in many hospitals in patients who were formerly considered irretrievably unresuscitable.

Several supportive articles were again published this year advocating the therapeutic use of arterial embolization in selected instances after trauma. There is little question that in some areas, such as the kidney, this technique will become a permanent and useful one for surgeons caring for such patients.

This year, several papers further define the limits and indications for attempts at repair of the carotid artery after trauma. This is a particularly perplexing problem, because some patients who have sustained trauma obviously should not be operated on, for fear of converting a grey infarct into a red hemorrhagic one with significant

intracranial bleeding. Articles on management of retroperitoneal hematoma continue to appear each year. Apart from hematomas associated with pelvic fracture, evidence is mounting to show that upper abdominal retroperitoneal hematomas, regardless of size or expansion, should be explored in the operating room.

A plethora of articles has appeared on the management of trauma to the spleen, particularly in children. Most authors are making some major attempt to define the role of splenectomy for trauma. Little doubt exists that if there is massive pulverization or irreparable destruction of the splenic blood supply, splenectomy for trauma is life-saving. However, it is also clear that the role of the spleen in host defense against infection is an important one. Most authors have continued to delineate the dangers of nonoperative management of splenic injury in view of the fact that nearly a third of these patients have other significant intra-abdominal injury. Further definition of surgical repair continues to appear, including partial splenectomy, as well as the subsequent use of pneumococcal vaccine and antibiotic prophylaxis when splenectomy is required. The amount of viable splenic tissue that needs to be kept intact to provide host defense against infections is currently under active study, but has not been determined yet. One other approach tried during the past year has been splenic artery ligation for salvage of the spleen; this may be useful in highly selected instances.

Probably the largest volume of papers to appear during the past year regarding the traumatized patient has been a series of studies designed to measure the alteration in host defenses after trauma, shock, and sepsis. Specific refinements of the definition of anergy and the role of phagocytic cells in the host defense to trauma are among those areas most intensively studied. One topic again under intensive study both in the United States and abroad has been the depressed phagocyte function in the injured patient. Early attempts at prevention of loss of phagocytic function or stimulation of phagocyte function by opsonic glycoprotein therapy continued to be evaluated. There is early enthusiasm for the use of the fibronectin substance as therapy. Additional studies evaluate lymphocyte response to injury; a specific definition of alteration in the quality as well as quantity of leukocytes after injury continues to be developed. A nice study examining the relationship between injury and complement activation in a canine model of soft tissue trauma also has been developed. There is little question that there is some complement activation with platelet aggregation after various insults. Complement activity declines after soft tissue trauma as well as after endotoxin infusion and burn injury. These studies continue to implicate complement consumption in response to injury. The bulk of available evidence indicates that macrophages are in some way rendered inefficient after injury, and their important role in immunologic regulation certainly should be further studied in an attempt to reverse this loss of immunocompetence.

Wound Healing—The previously troublesome problem caused by greater trochanteric decubitus ulcers in paraplegic patients seems