ADULT CARDIAC SURGERY

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Blackwell-Wissenschaft, Meinekestrasse 4, D-1000 Berlin 15, Germany
Blackwell MZV, Feldgasse 13, A-1238 Wien, Austria

Distributors

USA
Blackwell Scientific Publications
238 Main Street
Cambridge, Massachusetts 02142
(Orders: Telephone: 800-759-6102)

AUSTRALIA
Blackwell Scientific Publications (Australia) Pty Ltd
54 University Street
Carlton, Victoria 3053
(Orders: Telephone: 03-347-0300)

CANADA
Times Mirror Professional Publishing Ltd.
5240 Finch Avenue East
Scarborough, Ontario M1S 5A2
(Orders: Telephone: 416-298-1588)

OUTSIDE NORTH AMERICA AND AUSTRALIA
Blackwell Scientific Publications, Ltd.
c/o Marston Book Services, Ltd.
P.O. Box 87
Oxford OX2 0DT
England
(Orders: Telephone: 011-44-865-791155)

Typeset by Pine Tree Composition
Printed and bound by the Maple-Vail Book Manufacturing Group

Printed in the United States of America
92 93 94 95 5 4 3 2 1

Library of Congress Cataloging in Publication Data

Bojar, Robert M., 1951–
Adult cardiac surgery / Robert M. Bojar.
p. cm.
Companion v. to the author's: Manual of perioperative care in cardiac and thoracic surgery.
Includes bibliographical references and index.
ISBN 0-86542-140-4
1. Heart—Surgery. I. Bojar, Robert M., 1951– Manual of perioperative care in cardiac and thoracic surgery. II. Title.
[DNLM: 1. Heart Surgery. WG 169 B686a]
RD598.B64 1992
617.4'12—dc20
DNLM/DLC
for Library of Congress 091-25948
CIP

ADULT CARDIAC SURGERY

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Blackwell Scientific Publications

Oxford London Edinburgh Melbourne
Paris Berlin Vienna

This book is dedicated to my parents,
Leah and Samuel,
whose lifelong devotion to helping others
continues to inspire my efforts.

Foreword

During the past decade, the art and science of cardiac surgery have continued to evolve. Nearly 300,000 procedures were performed worldwide in 1990, predominantly for the treatment of coronary and valvular heart disease. Sophisticated procedures such as valve reconstruction, homograft valve placement, ablation of arrhythmias, and heart, heart-lung, and lung transplantation have become routine in many centers. With advances in the nonsurgical treatment of cardiac disease, we are now encountering a patient population with far-advanced medical and surgical disease that stretches the limits of our knowledge and technology. It is becoming more difficult to decide when not to offer surgery, because surgery at times appears to be the last alternative when no other treatment is available.

The subspecialty fields of medicine and surgery have reached such a degree of sophistication that it is practically impossible for the "specialist" to be intimately aware of all developments in closely related fields. Moreover, because of the complexities of surgically treatable cardiac disease and the severity of illness of surgical candidates in the 1990s, an interdisciplinary approach to patient care is imperative. The referring physician, cardiologist, cardiac anesthesiologist, and cardiac surgeon must all share an understanding of cardiac disease and its treatment options. By nature of their training and daily experience, however, this knowledge has been difficult to obtain.

In *Adult Cardiac Surgery*, Dr. Bojar has approached these issues by providing a comprehensive presentation of nearly all cardiac surgical problems, incorporating basic concepts of pathophysiology, indications for surgery, surgical techniques, and anticipated results. He has selected excellent illustrations that make even the most complex procedures conceptually obvious, even to the nonsurgeon. Topics such as cardiopulmonary bypass and circulatory assist, which have traditionally been "black boxes" to most trainees, are discussed practically and in comprehensible terms. The discussions are state-of-the-art and provide an excellent review not only for the board-eligible cardiologist or cardiac surgeon, but also for the practicing physician who may wish to refresh his/her knowledge on the management of common as well as uncommon procedures, such as repair of aortic dissections, thoracic aneurysms, cardiac tumors, and arrhythmia surgery.

Adult Cardiac Surgery provides the important information that the reader should know about each subject. It is one of the most clearly written books on cardiac surgery that I have come across in a long time. I recommend it highly to anyone—nonsurgeon and surgeon alike—involved in the care of the adult cardiac surgical patient.

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Surgeon-in-Chief
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Preface

As cardiac surgery becomes more sophisticated, it has become difficult for individuals involved in various aspects of patient care to obtain a comprehensive understanding of cardiac disease and its surgical management. It has become especially difficult for the noncardiac surgeon or even for the cardiac surgeon who infrequently encounters certain problems to be aware of the intricacies of patient selection, the essence of a surgical procedure, or the anticipated results of surgery that the experienced cardiac surgeon has learned through daily experience.

I have frequently observed the difficulty and frustration of many individuals in obtaining this knowledge. For example, because of the time commitment to patient care and the limitation of preoperative evaluation in the hospital setting, most cardiac surgeons-in-training, surgical residents, medical students, physician assistants, and cardiac nurses have little time to learn about the pathophysiology of cardiac surgical disease, its diagnosis, and the nature of surgical treatment. Cardiologists infrequently visit the operating room to actually see how a surgical procedure is performed and may not understand the technical difficulties or hemodynamic problems that may influence the surgical result. Anesthesiologists are often unaware of the details of unusual surgical techniques that influence their approach to the patient. The cardiac surgeon-in-training is rarely involved in the details of preoperative evaluation, spends most of the time learning technical maneuvers, and, in some centers, may not be actively involved in postoperative care.

I have often been asked by these individuals if there were any reference source where they could learn about the assessment and treatment of patients with cardiac surgical disease without the expense of a major comprehensive subspecialty textbook that usually covers thoracic and pediatric cardiac topics as well. I have become convinced that all members of the health care team are truly interested in understanding more about how cardiac surgeons decide what to do, why to do it, and for whom it should be done. This book is, hopefully, basic enough for the noncardiac surgeon, but also detailed enough to provide a state-of-the-art discussion on nearly all adult cardiac surgical topics for the cardiac surgeon.

I have tried to provide a comprehensive approach to the common and uncommon problems of acquired heart disease that the cardiac surgeon may encounter in the adult patient population. I have elected not to discuss congenital heart disease that may require follow-up or surgical intervention in adults. Each chapter is extensively illustrated to demonstrate important points or operative techniques. I have attempted to avoid a literature review within the text to maintain its readability, but have referred to and provided an extensive reference list at the end of each chapter for the interested reader. Selected references that provide ex-

cellent summaries of various topics are listed at the beginning of the reference section for each chapter.

It is hoped that this book, along with its companion, *Manual of Perioperative Care in Cardiac and Thoracic Surgery*, will enable more individuals involved in the care of the adult cardiac surgical patient to gain a better understanding of the field of cardiac surgery and thus improve their delivery of care and enhance communication with their patients.

Robert M. Bojar, M.D.

ACKNOWLEDGMENTS

This book originated from a desire to share accumulated knowledge and experience with others who are involved in the care of the adult cardiac surgical patient. I am grateful to several of my colleagues in cardiac surgery and cardiology who set aside valuable time to review and critique the manuscript. I am particularly grateful to Drs. Douglas D. Payne and Deeb N. Salem for their review of several chapters and to Drs. Christopher A. Clyne, James T. Diehl, N.A. Mark Estes III, James E. Udelson, Paul J. Wang, Andrew R. Weintraub, and Mr. Richard E. Murphy, P.A., for their assistance and comments. Drs. Weintraub and Steven Schwartz were kind enough to provide me with excellent pictures of echocardiograms and the unique intravascular ultrasound images of aortic dissection. I would also like to thank the staff of the Educational Media Center of Tufts-New England Medical Center for their assistance with illustrations and photographs used throughout the book.

NOTICE

The dosages of all drugs and the indications for their use in this book have been recommended in the medical literature and conform to the practices of the general medical community. The medications described do not necessarily have specific approval of the Food and Drug Administration for use in the diseases and dosages for which they are recommended. The package insert for each drug should be consulted for use and dosage as approved by the FDA. Because standards for usage change, it is advisable to keep abreast of revised recommendations, particularly those concerning new drugs.

Contents

Foreword		ix
Preface		xi
Acknowledgments		xiii
1	Cardiopulmonary Bypass *with Judith M. Deiss, B.A., C.C.P.* *Cardiopulmonary Perfusionist* *New England Medical Center*	1
2	Myocardial Protection	37
3	Coronary Artery Bypass Surgery	77
4	Valvular Heart Disease, Including Hypertrophic Cardiomyopathy	153
5	Aortic Dissections	241
6	Thoracic Aneurysms	273
7	Surgery for Arrhythmias *with Hassan Rastegar, M.D.* *Associate Professor of Cardiothoracic Surgery* *Tufts University School of Medicine* *Senior Cardiothoracic Surgeon* *New England Medical Center*	313
8	Cardiac Tumors	355
9	Cardiac and Great Vessel Trauma	373
10	Surgery of the Pericardium	403
11	Circulatory Assist Devices	423
12	Heart Transplantation	461
13	Heart-Lung and Lung Transplantation	507

1

CARDIOPULMONARY BYPASS

JUDITH M. DEISS
ROBERT M. BOJAR

Historical Perspective
Pathophysiologic and Damaging Effects of CPB
Basic Components of the Bypass Circuit
Connection of the Patient to the Heart-Lung Machine
The Conduct of Bypass and Hemodynamics
Profound Hypothermia and Circulatory Arrest
Catastrophic Complications of CPB
Other Interesting Problems of CPB
Additional Uses of Extracorporeal Circulation

CARDIOPULMONARY BYPASS

JUDITH M. DEISS
ROBERT M. BOLAR

HISTORICAL PERSPECTIVE

The use of artificial circulation to maintain organ system function intrigued physiologists during the nineteenth century. Primitive experiments were conducted to provide extracorporeal oxygenation and supply oxygenated blood to isolated parts of the body. The first heart-lung machine that could oxygenate blood without interrupting blood flow was designed by von Frey and Gruber in 1885. Although the idea that this technology might be useful for cardiac surgery has been ascribed to the Russian scientist Brukhonenko, the one individual responsible for bringing this concept to fruition is John Gibbon. In 1931, he was monitoring a critically ill patient with a pulmonary embolism who subsequently died following an emergency pulmonary embolectomy. He felt that a means of providing extracorporeal oxygenation with return of blood to the arterial circuit could have saved the patient's life. Believing that this concept might have application to patients undergoing heart surgery, he worked diligently over the next 20 years to develop a heart-lung machine which he successfully used for repair of an atrial septal defect in 1953.[1] His research took advantage of the discovery of heparin and protamine, an improved understanding of circulatory physiology and the effects of hypothermia, and advances in bioengineering and biomaterials.

Experimental work at the University of Minnesota by Dennis and in Europe by Crafoord, Björk, and Senning in the late 1940s and early 1950s also contributed to the development of cardiopulmonary bypass (CPB). Because of the limited early success of CPB, Lillehei performed congenital heart surgery in the early 1950s using controlled cross-circulation, with the child connected to the parent's circulation, which provided oxygenation and perfusion during the procedure. Subsequently, Kirklin and associates at the Mayo Clinic performed the first series of operations using CPB in 1955. Further understanding of the physiology of hypothermia and hemodilution, the development of more efficient oxygenators, and advances in the biocompatibility of foreign surfaces have led to a refinement of extracorporeal circuits and the widespread use of CPB for cardiac surgical procedures throughout the world.

PATHOPHYSIOLOGIC AND DAMAGING EFFECTS OF CPB

Cardiopulmonary bypass provides oxygenation and systemic perfusion in a nonphysiologic manner. Perfusion is conducted using heparinization, hemodilution, nonpulsatile flow, and some degree of hypothermia. The extracorporeal circuit exposes blood to nonendothelial surfaces and shear stresses and to abnormal substances which can produce embolization.[2] As a result of these phenomena, numerous pathophysiologic abnormalities occur that can be potentially damaging to organ system function. Extensive clinical experience has shown that significant structural and functional abnormalities are relatively unusual following short-term use of extracorporeal circulation because of the body's ability to compensate for these damaging effects of CPB.

The exposure of blood to foreign surfaces occurs predominantly at the boundary layers in the oxygenator–heat exchanger and within defoaming devices or filters. Platelet clumping and degranulation occur upon contact with nonendothelial surfaces. This

results in a reduction in platelet number, as well as inhibition of platelet adhesive and aggregating properties that can contribute to a bleeding tendency following bypass. Denaturation of oncotic and carrier proteins, including albumin, lipoproteins, and gamma globulins, occurs. This can lead to increased blood viscosity, clumping of red blood cells, and release of fat emboli. A reduction in gamma globulins, as well as the impairment in neutrophil and T-cell function that occurs during CPB, may increase the patient's susceptibility to infection.

The proteins of the "humoral amplification systems" are activated by exposure to nonendothelialized surfaces. The initial step appears to be activation of Hageman factor (factor XII). This stimulates the coagulation and fibrinolytic cascades, leading to microcoagulation and consumption of clotting factors even during satisfactory heparinization. Activation of the complement system and release of vasoactive substances such as kallikrein and bradykinin produce an alteration in vascular tone and a generalized inflammatory response that increases capillary permeability, leading to expansion of interstitial volume.[3] The humoral amplification systems are interactive and can perpetuate the adverse effects of each other.

The shear stresses that result from use of the blood pumps, various suction devices, and from cavitation at the end of cannulas affect the blood elements. Red blood cells may undergo immediate lysis or sustain membrane injury that leads to delayed hemolysis. Hemoglobinuria may occur and impair renal tubular function. Damage to leukocytes produces degranulation with release of vasoactive substances and impairment of phagocytic properties. Complement activation causes pulmonary sequestration of aggregated neutrophils, producing an inflammatory response that may contribute to lung injury.[4]

The presence of other abnormal substances within the heart-lung circuit can result in microemboli. These include bubbles from the oxygenator, fibrin particles, platelet aggregates, and air. Despite the use of a filtered cardiotomy and an arterial line filter, there is good evidence that microembolization occurs during CPB and contributes to subtle end-organ damage.[5]

Endocrine Effects

The abnormal physiology of CPB contributes to a variety of endocrine responses that influence vasomotor tone, organ system perfusion or function, and carbohydrate and lipid metabolism. There is a marked elevation in epinephrine levels, especially during hypothermia, which causes peripheral vasoconstriction and intraorgan redistribution of blood flow. Epinephrine also impairs insulin release, contributing to hyperglycemia, and causes release of free fatty acids. Norepinephrine levels are not uniformly elevated, but are noted to rise early during CPB in patients who develop postoperative hypertension. Renin and aldosterone levels are increased, promoting sodium retention and potassium excretion. This effect is counterbalanced by a dramatic increase in vasopressin, which produces a paradoxical increase in sodium and water diuresis. Elevation in angiotensin levels favors vasoconstriction. Additional endocrine effects that occur during CPB are an increase in levels of plasma-free cortisol and free thyroxine.[6]

Hemodilution

The use of balanced salt solutions as a pump prime provides normovolemic hemodilution during CPB. The major benefit of hemodilution is a reduction in the requirement for homologous blood transfusion

and its attendant risks. In addition, hemodilution improves tissue perfusion by reducing blood viscosity, even though it reduces the amount of hemoglobin-bound oxygen transported to tissues. The improved microcirculatory flow can deliver sufficient oxygen to tissues with hematocrits as low as 20%, and perhaps slightly lower.[7] This improved flow can counteract some of the deleterious effects of hypothermia on tissue perfusion, including vasoconstriction and impaired oxygen release from hemoglobin. Because the reduction in plasma oncotic pressure caused by hemodilution may contribute to interstitial edema, many centers add albumin or a synthetic colloid to the pump prime. However, the use of albumin has not been shown to provide a significant clinical benefit.[8] Hemodilution may also contribute to a postbypass coagulopathy by diluting the concentration of coagulation factors and platelets.

Hypothermia

Systemic hypothermia is often used during CPB because it reduces tissue metabolism and oxygen consumption. This is beneficial for several reasons. First, it improves myocardial protection during a period of ischemic or cardioplegic arrest by minimizing the temperature gradient between the heart and the systemic circulation and adjacent structures. Secondly, systemic hypothermia provides a margin of safety for noncardiac organ systems (principally the brain and kidneys) in the event that a brief period of low flow or circulatory arrest is necessary. This may result from a technical mishap (line disruption, air lock in the venous line, poor venous return, air embolism, oxygenator failure), or may be useful when added exposure is required, as, for example, during construction of a difficult anastomosis.

Hypothermia is an abnormal physiologic state in humans. Although it lowers metabolic demands, it also increases systemic vascular resistance and reduces blood supply to most organ systems. The concomitant use of hemodilution offsets the potential adverse effects of hemoconcentration and increased blood viscosity on microcirculatory flow. Hypothermia shifts the oxygen-hemoglobin curve to the left, impairing oxygen release to the tissues. However, the increased solubility of oxygen at lower temperatures and the lower metabolic demand offset this potential adverse effect on tissue oxygenation. Hypothermia contributes to thrombocytopenia and platelet dysfunction, but is associated with clinical coagulopathies only during the rewarming phase from profound hypothermia ($<20°C$).

One of the controversies during hypothermia is the appropriate acid-base milieu for optimal cellular protection. This is of particular concern in preventing cerebral injury because of the reduced cerebral blood flow during hypothermia. The pH-stat method of acid-base balance refers to the maintenance of a $PaCO_2$ of 40 torr and a pH of 7.4 at the patient's core temperature (temperature-corrected). The theoretical advantage of this approach is that the addition of CO_2 to the circuit can provide cerebral vasodilatation and improve oxygen availability that is reduced by hypothermia. The alpha-stat method refers to a non-temperature-corrected pH of 7.4 and $PaCO_2$ of 40 torr, so that the pH is actually much higher at hypothermia. This preserves intracellular electrochemical neutrality and optimizes the function of metabolic intracellular enzymes.[9] Alpha-stat management has been shown to maintain cerebral autoregulation and flow/metabolism coupling. Thus the brain is able to adjust cerebral blood flow at various perfusion pressures

according to metabolic demand. In contrast, pH-stat impairs cerebral autoregulation, and cerebral blood flow becomes pressure dependent below 55 mm Hg. Because flow/metabolism coupling is disturbed, the augmented cerebral blood flow produced by adding CO_2 might potentially lead to intracerebral shunting away from regions of ischemia.[10] However, clinical evidence of intracerebral steal has not been conclusively demonstrated in patients with cerebrovascular disease using the pH-stat method.[11]

Nonpulsatile Perfusion

Perfusion provided by roller pumps or a centrifugal pump is nonpulsatile and nonphysiologic. Pulsatile perfusion may be provided by partial bypass, the intra-aortic balloon pump, pulsatile pumps, or modified roller pumps. Whether pulsatile perfusion is less deleterious to organ system function than nonpulsatile perfusion is controversial. Pulsatile flow may be associated with improved microcirculatory flow and tissue metabolism. There is some evidence that it prevents the vasoconstriction associated with nonpulsatile perfusion, causes less edema formation, less elevation in catecholamine and vasopressin levels, higher prostacyclin and lower thromboxane levels, and improves organ perfusion, especially to the brain and kidney. Because definitive evidence of a clinical benefit has not been demonstrated, nonpulsatile perfusion continues to be used by nearly every cardiac surgical team.[12-14]

Organ System Function during Bypass

Cerebral perfusion is maintained by autoregulation of cerebral blood flow. This is highly effective when perfusion pressure is maintained above 50 mm Hg, and may still function at lower pressures during hypothermia. The use of alpha-stat management appears to preserve flow/metabolism coupling better than pH-stat and is considered the preferable method of acid-base management to preserve cerebral perfusion. Nonetheless, adequate systemic perfusion pressure must be maintained because, below the limits of satisfactory autoregulation, cerebral blood flow is dependent on the cerebral perfusion pressure.

In certain clinical situations, autoregulation is impaired and cerebral blood flow becomes pressure dependent. For example, patients with chronic hypertension appear to have impaired cerebral autoregulation below 80 mm Hg, and autoregulation is also impaired beyond flow-limiting carotid stenoses. In these situations, flow becomes pressure dependent, and the minimally acceptable cerebral perfusion pressure is considered to be higher than normal.

Because cerebral perfusion is generally presumed to be adequate when the mean arterial pressure is maintained above 50–60 mm Hg, postoperative cerebral dysfunction has been more commonly attributed to macro- or microembolization during surgery. Studies using intraoperative transcranial Doppler and fluorescein retinal angiography have documented a high incidence of retinal and cerebral microemboli of blood cell aggregates during CPB.[5,15] The use of arterial filters and membrane oxygenators have reduced, but not completely eliminated, the problem of microembolization.[16]

The lungs are left deflated during bypass without adverse effect on pulmonary function. The elevation of circulating catecholamines during and after CPB may be related to their lack of inactivation by the lungs. Adequate venous drainage or venting must be assured during CPB to prevent pulmonary hypertension and pulmonary edema. Alveolar capillary damage with an increase

in interstitial lung water and pulmonary sequestration of neutrophils may result from the activation of complement, release of vasoactive substances such as kinins, and the generation of oxygen free radicals. Although it is unclear whether neutrophil aggregation attributed to complement activation actually causes pulmonary damage,[17] there is evidence that leukocyte depletion ameliorates free radical–mediated lung injury.[4] Marked impairment of pulmonary function may follow prolonged periods of bypass.

Renal function is influenced by hemodilution, hypothermia, and various endocrine effects during nonpulsatile perfusion. Changes in the renin-angiotensin-aldosterone system promote an increase in renal vascular resistance and a tendency to sodium and water retention, while increases in vasopressin favor sodium and water diuresis. The vasoconstrictive effects of hypothermia reduce renal cortical blood flow, glomerular filtration rate, tubular function, and free water and osmolar clearance. In contrast, hemodilution protects against renal damage during CPB by increasing outer cortical renal plasma flow, and increasing sodium, potassium, osmolar, and free water clearance. The diminished urinary osmolarity protects renal tubular integrity. Thus, these effects tend to counterbalance each other and produce an adequate urine output during bypass.[18]

In general, urine output will be diminished during hypothermia and improved at normothermia and after resumption of pulsatile perfusion. If urine output does not exceed 1 mL / kg / hour during CPB, it can be promoted with mannitol, furosemide, or low-dose dopamine.[19] Renal dysfunction may also result from hemoglobinuria during long bypass runs. In patients with preexisting renal dysfunction, dialysis or continuous arteriovenous hemofiltration (CAVH) can be used during bypass to eliminate excess plasma water and electrolytes. Although renal dysfunction may follow a prolonged period of bypass (>4 hours), the most common cause of perioperative renal failure is low cardiac output syndrome.

Gut mucosal ischemia is very common during cardiopulmonary bypass but rarely produces clinical evidence of gastrointestinal disease in the absence of postoperative low cardiac output syndrome. Contributory factors include splanchnic vasoconstriction induced by elevated angiotensin II levels during nonpulsatile perfusion, splanchnic shunting during rewarming from systemic hypothermia, microembolization of platelet or leukocyte aggregates with release of vasoactive substances, and preexisting atherosclerotic disease in the splanchnic bed. Gut mucosal ischemia can contribute to low cardiac output syndromes, bacteremia, and multiorgan system failure. Postoperative ischemic pancreatitis occasionally occurs despite an uneventful postoperative course and is probably ascribable to one of these contributory factors during the period of CPB.[20]

BASIC COMPONENTS OF THE BYPASS CIRCUIT

The cardiopulmonary bypass circuit for cardiac surgery consists of a venous drainage line, a venous reservoir, an oxygenator–heat exchanger, a pump, and an arterial return line (Figure 1.1). In addition, there is a suction line that provides drainage from intracardiac chambers (''vent'' line) and other lines that aspirate pooled blood into a cardiotomy reservoir before its reentry into the venous reservoir (''pump suckers''). Additional adjuncts include a cardioplegia delivery system, an arterial line filter, various in-line monitors and blood gas an-