CARDIOVASCULAR ANESTHESIA AND POSTOPERATIVE CARE

SAIT TARHAN

Second Edition

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Cardiovascular Anesthesia and Postoperative Care

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/ Foreword

It is an immense privilege for me, as a surgeon, to make these few introductory comments to this impressive text prepared by my respected colleagues in anesthesiology. There are few partnerships so intensely interdependent, so intimately intertwined, and so personally poignant as that which exists between a cardiovascular anesthesiologist and a cardiac surgeon. Especially is this true if these professionals have been blessed by a long and respectful relationship. As they wrestle with the mutually relevant case-by-case decisions that attend the daily drama of the operating room, instinctive interactions and intercommunications develop almost as though through a single brain. Such is one of the greatest rewards of their distinctive careers, and perhaps one of the greatest benefits they could confer on their patients.

Similar also is the relationship between the disciplines themselves—cardiac anesthesiology and cardiac surgery. This may undoubtedly be true of other branches of surgery, too, but a subtle difference seems to exist. The elegantly manipulable variables of the cardiovascular system, and the sense of immediacy fostered by the moment-by-moment dependence of the patient's life and health on the proper control of the circulation, all serve to excite and illuminate the interdependence of these disciplines.

Immense progress has been made during the past few decades in the management of the cardiac surgical patient. Many texts have dealt with the surgical arm of this subject, but a place remains for a thoroughly comprehensive compilation of the theory and the techniques of cardiovascular anesthesiology. What is particularly advantageous in such an endeavor is the ideal blending of that which is theoretical and that which through experience has proved to be practical.

Aristotle struggled 24 centuries ago with the proper blending of experience and theory with respect to teaching (Book I of *Metaphysics*). He said, "With a view to action, experience seems in no respect inferior to [theory], and men of experience succeed even better than those who have theory without experience . . . [yet men with theory] can teach, and men of mere experience cannot."

This book on cardiovascular anesthesiology exposes the incompleteness of Aristotle's apparent assumption that theory and experience are isolated qualities, interdependent but not cohabitable in the same person. Each of the contributors to this text is involved in the busy day-by-day application to clinical practice of the theory and art that his or her writings encompass. The various chapters comprehensively organize the many facets of knowledge and experience pertaining to the field of cardiac anesthesiology. The authors of these chapters, each having a special area of expertise, are orchestrated into a single team of specialists from one institution whose concepts and methods are constantly being stressed by the demands of clinical practice. This comprehensiveness and cohesiveness give mastery to the book.

The first 45 or more years of cardiac surgery and the first 30 years of "open-heart" operations are mileposts that have already fallen behind. We have thus witnessed a rapid stage of evolutionary progress in cardiovascular anesthesiology, which suggests that this is the perfect time to step back and to observe what has happened, what is known, and what is practiced. The authority whereby this book speaks so effectively involves the contributions

made by its authors to what has happened and what is known, and, most importantly, to the application of this knowledge to a large ongoing single clinical practice. This very comprehensiveness and unification should also serve to expose any possible gaps in knowledge and any potential defects in techniques, thus revealing new challenges that must be addressed—new triumphs to be gained.

Dwight C. McGoon, M.D.

/ Preface to the First Edition

This book represents a cooperative effort to provide clinicians with a detailed perspective on anesthesia for cardiovascular surgery. The chapters are deliberately designed to be read separately, and since they were prepared by several authors, some reiteration was inevitable. The writing style of each author was not altered, which should make reading more interesting.

One of the major problems was what to include and what to omit in a book of this type. It is our belief that a clear understanding of the pathophysiologic derangements produced by the various types of heart disease and a familiarity with the physiologic changes accompanying anesthesia and surgery undoubtedly are the most important factors for optimal anesthetic management. Therefore, an attempt was made to include pathologic and pathophysiologic alterations, as well as surgical procedures used. My colleagues and I concentrated our efforts on providing comprehensive knowledge on subjects that are not readily available in a composite published form. The chapters on radiology, anesthesia for closed cardiac operations, diagnostic procedures, abdominal and thoracic aortic aneurysms, carotid artery surgery, myocardial preservation, renal function, and blood in cardiovascular anesthesia are examples of such topics.

Surgery for congenital heart disease has experienced great progress during the last 10 years, and it stands as a subspecialty among general cardiac surgery. Providing anesthesia for these patients also presents a challenge to the anesthesiologist. Therefore, a special emphasis was placed on this subspecialty.

Last but not least, postoperative care of the cardiovascular surgical patient remains one of the most important aspects of the entire surgical undertaking. Proper care can make the difference in the outcome of the patient. Without a section on that subject, a book of this type would be incomplete.

The methods discussed reflect the current practice in our institution. They have worked well for us in a large-volume practice, and no other claim is attached to them. They are by no means beyond challenge. Up-to-date options from other institutions also are discussed whenever appropriate.

The authors of this edition are greatly indebted to many colleagues from different disciplines at our institution who read and had constructive comments on many of the manuscripts. We also express our thanks to Dr. Marc Shampo, Virginia Dunt, Betty Calkins, and Mary Schwager of the Section of Publications, to Floyd Hosmer of the Section of Medical Graphics, and to members of the Section of Photography. Also, our appreciation goes to our secretaries Ann Tvedt, Donna Huntley, and Jeanne Halbach for the countless hours that they spent in preparing the manuscripts.

A promise was made to complete this project in a few months, yet it has taken almost 2 years. We appreciate the forebearance and continuous support of our publishers.

The greatest reward for the contributors and editor alike will be if a reader finds this book understandable and informative.

Sait Tarhan, M.D.

/ Preface to the Second Edition

Since the publication of the first edition of this book, a remarkable expansion of knowledge has occurred in all areas of cardiac anesthesiology, involving new anesthetic agents, techniques, pharmacology, surgical procedures, monitoring, and postoperative care. The anesthesia practice has become more sophisticated, and the subspecialty of cardiac anesthesia has gained prominence.

Therefore, a new book, encompassing all of these new advances, was needed. The first edition of this book had an exceptional acceptance by the readers, indicated by the reviewers' comments and vast distribution of the book throughout the United States and to many institutions abroad. This was prime motivation for updating the work.

Because of new knowledge accumulated during the last few years, a number of the chapters have been completely rewritten. New chapters have been added on anesthetic agents, the pericardium and anesthesia, anesthesia for electrophysiologic testing, surgical treatment of arrhythmias, resection of ventricular aneurysms, blood-related communicable diseases, and postoperative management of adult and pediatric cardiac patients. A few outdated chapters have been deleted from the new edition.

Once again, contributors have been allowed different styles of presentation, which seems desirable, and have been permitted to express their views in the way they think best. Every effort has been made to include the latest literature in this new edition. I think this goal has been accomplished.

I am greatly indebted to the contributors, all of whom are fully occupied with their professional work but who found the time to contribute to this second edition.

I am again greatly indebted to my many colleagues for their support and review of the manuscripts, specifically to Dr. Marc Shampo, Mary Schwager, Betty Calkins, and Sharon Wadleigh of the Section of Publications and to the secretaries in the various departments. Their help was invaluable.

I hope that this second edition will have the same acceptance and credibility as the first edition.

Sait Tarhan, M.D.

/ Contents

1 / Preoperative Evaluation

DEBORAH ANN W. WILKOWSKI
JEFFREY J. LUNN

Preanesthetic evaluation of the patient provides a guide for anesthetic management, supportive techniques, and postoperative care. Regardless of age and physical condition, a patient who is a candidate for a cardiac operation (or major vascular surgery) should be evaluated carefully before surgery.

HISTORY

Risk Factors

Many studies have been done in an attempt to distinguish the patient who is at high risk for developing cardiovascular disease. The Framingham study[1] demonstrated that the patient at high risk had a high cholesterol level, hypertension (systolic >160 mm Hg), a smoking history, electrocardiographic evidence of left ventricular hypertrophy, and glucose intolerance. Overall, the study showed that the chances of a patient developing cardiovascular disease by age 65 years are 37% for men and 18% for women. Data from the Coronary Artery Surgery Study[2, 3] (CASS) showed that age, male sex, cigarette smoking, and serum cholesterol level best differentiated the group with coronary artery disease from those without it. A positive family history of coronary artery disease and the presence of hypertension or diabetes were of additional, but less, discriminating value. Variables most predictive of surgical mortality included age, female sex, increased heart size, congestive heart failure, left ventricular wall motion abnormalities, and left main coronary artery disease. The urgency of operation was also associated with operative mortality.[2] Obesity has been shown to be a significant independent predictor of cardiovascular disease, especially in females.[4] Goldman[5] developed a scoring system to predict patients who are at high risk for noncardiac surgery. Evidence of left ventricular dysfunction and recent myocardial infarction (within 6 months of operation) have the highest predictive value (Table 1–1).

Wenger,[6] using epidemiologic data and data reported by CASS, noted that myocardial infarction in women is characterized by increased morbidity and mortality, an excess of congestive heart failure, and a less favorable response to coronary bypass surgery. Women have a mortality rate of more than 75% during the first month after myocardial infarction. The rates of recurrence of infarction and of 1-year mortality for women are more than twice those for men (45% vs. 20%). The advanced age of women at the time of infarction may contribute to this increased mortality. The operative mortality in women is at least twice that in men.[7] CASS demonstrated that the female sex is a more reliable predictor of perioperative mortality from coronary bypass surgery than is the severity of angina or the degree of left ventricular dysfunction.[8]

Cardiac Status

Preoperative assessment of the heart can be divided into two categories: the "pumping" function of the heart and the "fuel supply" of the pump. Patients with impaired left ventricular function may present with classic signs and symptoms of congestive heart failure—that is, dyspnea on exertion, orthopnea, and edema. The most frequent causes include hypertension, ischemic heart disease, valvular disease, and various cardiomyopathies. Congenital cardiac anomalies in children may be discovered when congestive heart failure develops. Poor feeding and slow growth may be early clues. Breathlessness is the most frequent symptom of heart failure, although it is not a reliable indicator of the extent of the failure. The severity of symptoms in both children and adults is related to the rapidity of onset of the heart failure. Acute insults such as myocardial infarction may result in severe symptoms of

TABLE 1–1.

Computation of Multifactorial Index Score to Estimate Cardiac Risk in Noncardiac Surgery*

Finding	Points
S_3 gallop or jugular venous distention on preoperative physical examination	11
Transmural or subendocardial myocardial infarction during previous 6 months	10
Premature ventricular beats (more than 5/min documented at any time)	7
Rhythm other than sinus rhythm or presence of premature atrial contractions on last preoperative electrocardiogram	7
Age more than 70 years	5
Emergency operation	4
Intrathoracic, intraperitoneal, or aortic site of operation	3
Evidence for important valvular aortic stenosis†	3
Poor general medical condition‡	3

*From Goldman L.[5] Used by permission of the American College of Physicians.
†Findings on cardiologist's examination, noninvasive testing, or cardiac catheterization.
‡As evidenced by electrolyte abnormalities (potassium <3.0 mEq/L; HCO_3 <20 mEq/L), renal insufficiency (blood urea nitrogen >50 mg/dl; creatinine >3.0 mg/dl); abnormal blood gases (PO_2 <60 mm Hg; PCO_2, >50 mm Hg), abnormal liver status (elevated aspartate transaminase level or signs of chronic liver disease at physical examination); or any condition that has caused the patient to be chronically bedridden.

congestive heart failure, even though only a small portion of the myocardium is damaged. In conditions that develop slowly, such as aortic stenosis, mechanisms of adaptation develop gradually so that large portions of the heart muscle may be impaired before symptoms become apparent.

In addition to ventricular function, the supply of oxygen to the heart must be considered. This supply is equally important in cardiac and major vascular procedures. The primary cause of morbidity and mortality with peripheral vascular reconstruction is ischemic heart disease leading to myocardial infarction.[9, 10] Coronary artery disease is responsible for nearly half of all perioperative deaths and late deaths that occur after vascular surgery. The presence of angina or previous myocardial infarction either by history or electrocardiogram is indicative of coronary artery disease. Numerous studies have confirmed that patients with previous myocardial infarction are at increased risk of reinfarction with anesthesia and surgery. Steen et al.[11] studied a series of 587 patients who had suffered previous myocardial infarction and who were to undergo anesthesia and surgery. The overall reinfarction rate was 6.1%, and the mortality rate in the group with reinfarction was 69%. In patients who underwent surgery within 3 months of the myocardial infarction, the reinfarction rate increased to 27%. The rate decreased to 11% if 3 to 6 months had passed since the myocardial infarction. The reinfarction rate stabilized at 4% to 5% if the interval was more than 6 months. Rao et al.[12] noted a decrease in the reinfarction rate in patients having noncardiac surgery within 3 months of a myocardial infarction from 36% to 5.7% and from 26% to 2.3% if the myocardial infarction had occurred 4 to 6 months before surgery. These decreases were accomplished by invasive cardiovascular monitoring and aggressive use of inotropic and vasoactive drugs during the perioperative period. Other risk factors associated with reinfarction included preoperative hypertension, associated congestive heart failure, intraoperative hypotensive episodes, intraoperative hypertension and tachycardia, and noncardiac thoracic or upper abdominal operations of more than 3 hours in duration.[11, 12]

The severity of coronary artery disease is not closely related to the severity of symptoms or the electrocardiographic changes. Patients with normal left ventricular function and no history of congestive heart failure may present with syncope or "sudden death." Further evaluation may reveal life-threatening coronary artery disease or critical valvular lesions such as aortic stenosis.

Unstable angina, angina at rest, or angina associated with acute congestive heart failure implies severe coronary artery disease. In patients with a varying exercise threshold for their angina, coronary spasm must be considered.

Frequently, the patient's ability to exercise gives an important clue to left ventricular function and the severity of coronary artery disease. Unfortunately, many patients suffering from peripheral vascular occlusive disease have limited exercise capacity as a result of claudication. In these patients, assessment of their cardiac status may require sophisticated diagnostic tests.

Diabetes

Diabetes and its complications must be considered. The onset of clinically evident cardiac disease frequently is observed years after the onset of diabetes. Even diabetic patients whose diabetes is well controlled, who do not have ketosis, and who clinically are in good condition with no significant obstructive coronary or valve disease have a significant reduction of stroke volume index and significant elevation of left ventricular end-diastolic pressure when compared with control subjects of similar ages.[13] If afterload is increased to a systemic diastolic pressure of between 15 and 18 mm Hg above control, diabetic patients may have a significant increase in left ventricular end-diastolic pressure (compared with a small increase of left ventricular end-diastolic pressure in normal subjects).[14] These data are considered consistent with the responses of a preclinical cardiomyopathy.[14] These factors must be considered in patients with long-standing diabetes who are to undergo surgery.

The incidence of silent myocardial infarction is higher in diabetic than in nondiabetic patients. Friedberg[15] reported this during the late 1950s, and the observation subsequently has been documented and statistically confirmed. Soler et al.[16] noted that 101 of 285 diabetic patients with myocardial infarctions were admitted directly to the ward and were not placed in coronary units. Infarctions were not suspected because heart failure and uncontrollable diabetes were the prominent symptoms. A painless myocardial infarction was considered to be due to myocardial anoxia (possibly hypoxia) from diffuse changes in the small myocardial vasculature.[15] When so little healthy heart muscle is left, coronary artery occlusion loses its impact. Later, it was proved that intrinsic cardiac autonomic neuropathy was responsible for blocking the reception and conduction of pain.[17]

Patients with diabetes of juvenile or adult onset should undergo surgery only if their diabetic status is well controlled. The methods for this control are outside the realm of this chapter and can be found in any general medical text. One fact, however, still remains. The reduced incidence of ketoacidosis and infections since the advent of insulin therapy has not been associated with a notable reduction in cardiovascular morbidity and mortality in diabetic patients.[15] The use of insulin in a more physiologic mode early during the course of the disease and the regulation of other hormones that affect carbohydrate metabolism have been proposed as means of preventing or delaying cardiovascular complications.[15] The primary effects of diabetes on the heart and its vasculature may be significantly influenced by associated hyperlipidemia, obesity, or hypertension. In one series,[18] 7% of patients undergoing coronary artery bypass had diabetes. The incidences of previous myocardial infarction, hypertension, and peripheral vascular disease in the diabetic patients compared with the nondiabetic patients were 62.5% versus 38%, 22.5% versus 12%, and 25% versus 10.5%, respectively. In spite of these apparent differences, at a mean follow-up of 3.9 years after surgery, no significant difference was noted in relief of symptoms or in survival between the diabetic and nondiabetic groups.

Hypertension

High blood pressure is a common health problem. Between 13% and 27% of the total population and 50% of patients more than 65 years of age may be hypertensive. Hypertension results from an increased systemic vascular resistance. There is decreased luminal diameter of arterioles secondary to thickening of the arteriolar walls. Initially, cardiac output is normal because hypertrophy of the heart muscle compensates for the increased workload. However, severe left ventricular hypertrophy limits diastolic compliance and ventricular filling. Eventually, stroke volume and cardiac output decrease.[19] The response of arterioles to vasoconstrictor and vasodilator stimuli is greater in the hypertensive patient than in a normotensive patient. This exaggerated response, combined with a decrease in total blood volume, results in increased blood pressure lability in hypertensive patients. Prys-Roberts[19] reported that patients with severe untreated hypertension (diastolic >120 mm Hg)

demonstrated an exaggerated hypotensive response to the induction and maintenance of anesthesia and showed an exaggerated hypertensive response to stimuli such as laryngoscopy and endotracheal intubation. These events were associated with myocardial ischemia. Goldman and Caldera[20] showed that patients with mild-to-moderate hypertension (diastolic >90 but <110 mm Hg) had no increase in postoperative morbidity when compared to normotensive patients. A major complication associated with hypertension is coronary artery disease, resulting in angina and myocardial infarction. Between 30% and 60% of hypertensive patients die of myocardial infarction.[21]

The autoregulatory limits of cerebral and renal blood flow are shifted upward in hypertensive patients.[21] Renal or cerebral perfusion may be significantly decreased if the blood pressure of severely hypertensive patients is lowered rapidly.[22] Patients with severe untreated hypertension (diastolic >120 mm Hg) who are scheduled for elective surgery should be rescheduled after a thorough medical evaluation and institution of appropriate antihypertensive therapy.[19]

For several years, it was advocated that antihypertensive therapy be discontinued before surgery. The observation that cerebrovascular accidents occurred more frequently when the antihypertensive therapy was discontinued led to the current practice of maintaining antihypertensive therapy preoperatively.[19, 21] Asiddao et al.[23] reviewed 166 cases of unilateral carotid endarterectomy. Patients with poor preoperative blood pressure control (blood pressure >170/90 mm Hg) had an incidence of postoperative hypertension of 52% compared to 35% in patients with adequate control and 17% in normotensive patients. Transient neurologic deficits occurred in 23.8% of patients with poorly controlled hypertension. The incidence of transient deficits was 2.5% in patients with controlled hypertension and 1.5% in normotensive patients ($P < 0.01$).[23]

Renal Function

Renal insufficiency occurs commonly in patients with coronary artery disease and atherosclerotic vascular disease. The most frequent cause of renal insufficiency in patients with hypertension is nephrosclerosis. Direct involvement of the renal arteries with atherosclerotic plaque may also cause hypoperfusion of the kidneys, resulting in impaired renal function. Diabetes predisposes to decreased kidney function. Conditions that produce low cardiac output, such as congestive heart failure, may also significantly impair renal function and may require medical intervention. Vasodilator therapy with hydralazine, clonidine, minoxidil and other agents has been effective in improving cardiac output.[24, 25] Because renal blood flow may not be improved by the addition of these agents, especially if the patient's blood pressure is decreased significantly, the serum creatinine level must be monitored closely.

In patients in complete renal failure, the volume and electrolyte status should be optimized by dialysis before operation. Ultrafiltration can be used during cardiopulmonary bypass to help maintain volume homeostasis.

Cerebrovascular Disease

A history of transient ischemic attacks or cerebrovascular accidents is important in the overall assessment of any patient who is a candidate for cardiac surgery. During cardiopulmonary bypass, the maintenance of adequate mean arterial pressure is important for all patients; however, patients with carotid artery occlusive disease may require higher mean arterial pressures to ensure adequate perfusion across the stenoses. In a retrospective survey of perioperative stroke after coronary artery bypass, Jones et al.[26] reported an overall incidence of 0.9%, which increased to 3.3% in patients with asymptomatic bruit and to 8.6% in patients with a history of transient ischemic attack or focal deficit. Patients with severe carotid artery occlusive disease may benefit from carotid endarterectomy before coronary artery bypass grafting.[26, 27]

Thyroid Dysfunction

Patients who are hypothyroid must be managed cautiously. Severe hypotension, cardiac arrest, prolonged unconsciousness, congestive heart failure, and myxedema coma have all been reported to occur in patients who are severely hypothyroid after anesthesia and surgery.[28] It has been postulated that hypothyroid patients have increased sensitivity to coronary spasm.[29] Thyroid replacement in myxedematous patients with coronary atherosclerosis may exacerbate angina or precipitate a myocardial infarction.[28] The direct inotropic and chronotropic effects of thyroid hormone are not inhibited by β-adrenergic blockers. In addition, pronounced bradycardia may result

when β-adrenergic blockers are given to hypothyroid patients. Thyroid replacement therapy in patients who need coronary artery surgery probably should be delayed until after surgery.[30] Patients with mild-to-moderate hypothyroidism can undergo cardiovascular surgery safely.[28, 31]

Medications

Antihypertensive medications generally can be categorized as vasodilators, diuretics, ganglionic blockers, angiotensin converting enzyme inhibitors, and agents that exert their pharmacologic action on the central nervous system (Table 1–2). The normal daily dose of antihypertensives should be administered on the morning of surgery (Table 1–3).

α_2-Adrenergic receptor agonists include α-methyldopa and clonidine. α-Methyldopa is converted to α-methyl norepinephrine, which stimulates central and peripheral α_2-adrenergic receptors. Central α_2-adrenergic receptor stimulation results in decreased sympathetic activity, increased vagal activity, and lower plasma norepinephrine and epinephrine levels. Renin secretion is also decreased. α-Methyldopa maintains or increases renal blood flow and is a good choice in patients with renal insufficiency. About 20% of patients treated with α-methyldopa have a positive result on the Coombs test. This may pose

TABLE 1–2.
Classification of Drugs Used in the Management of Hypertension*

Diuretics	Benzothiadiazines	Hydrochlorothiazide, bendroflumethiazide, cyclopenthiazide
	Loop diuretics	Furosemide, bumetanide, ethacrynic acid
	Distal tubule diuretics	Spironolactone, amiloride, triamterene
Vasodilators	Diazoxide, sodium nitroprusside (acute i.v. therapy)	Hydralazine, prazosin, minoxidil
Adrenergic inhibitors		
Drugs that inhibit central sympathetic activity		Clonidine, methyldopa
Drugs that inhibit norepinephrine synthesis, release or reuptake in adrenergic neurons	Rauwolfia alkaloids Guanidine derivatives	Reserpine, guanethidine, bethanidine, debrisoquin
Drugs that block adrenergic receptors	α-adrenergic receptor antagonists	Phenoxybenzamine, phentolamine
	β-adrenergic receptor antagonists	Acebutolol, atenolol, metoprolol†
		Propranolol, sotalol, oxprenolol, nadolol, timolol, pindolol‡
		Labetalol§
Inhibitors of the renin-angiotensin-aldosterone system		Captopril, enalapril
Calcium entry channel blockers		Nifedipine, lidoflazine, nicardipine

*From Prys-Roberts C.[19] Used by permission of the *British Journal of Anaesthesia*.
†All cardioselective drugs.
‡All nonselective drugs.
§All combinations of α- and β-adrenergic blockers.