KAPLAN’S CARDIAC ANESTHESIA: THE ECHO ERA

Sixth Edition

Editor

Joel A. Kaplan, MD, CPE, FACC
Professor of Anesthesiology
University of California, San Diego
San Diego, California

Dean Emeritus, School of Medicine
Former Chancellor, Health Sciences Center
University of Louisville
Louisville, Kentucky

Associate Editors

David L. Reich, MD
Horace W. Goldsmith, Professor and Chair
Department of Anesthesiology
Mount Sinai School of Medicine
New York, New York

Joseph S. Savino, MD
Professor of Anesthesiology and Critical Care
Vice Chairman, Strategic Planning and Clinical Operations
University of Pennsylvania School of Medicine
Philadelphia, Pennsylvania
CONTRIBUTORS

AHMAD ADI, MD
Department of Cardiothoracic Anesthesiology
Cleveland Clinic
Cleveland, Ohio

SHAMSUDDIN AKHTAR, MBBS
Associate Professor
Department of Anesthesiology
Yale University School of Medicine
New Haven, Connecticut

KORAY ARICA, MD
Clinical Assistant Professor
Department of Anesthesiology
SUNY Downstate Medical Center
Brooklyn, New York

JOHN G. AUGOUSTIDES, MD, FASE, FAHA
Associate Professor
Cardiovascular and Thoracic Section
Anesthesiology and Critical Care
University of Pennsylvania School of Medicine
Philadelphia, Pennsylvania

JAMES M. BAILEY, MD, PHD
Clinical Associate Professor
Department of Anesthesiology
Emory University School of Medicine
Atlanta, Georgia

DANIEL BAINBRIDGE, MD, FRCPC
Associate Professor
Anesthesia and Perioperative Medicine
Schulich School of Medicine
University of Western Ontario
London, Ontario, Canada

DALIA A. BANKS, MD
Associate Clinical Professor of Anesthesiology
Chief, Division of Cardiothoracic Anesthesia
Director of Cardiac Fellowship
Department of Anesthesiology
University of California, San Diego
La Jolla, California

PAUL G. BARASH, MD
Professor
Department of Anesthesiology
Yale University School of Medicine
New Haven, Connecticut

VICTOR C. BAUM, MD
Professor of Anesthesiology and Pediatrics
Executive Vice-Chair
Department of Anesthesiology
Director, Cardiac Anesthesia
University of Virginia
Charlottesville, Virginia

ELLIOTT BENNETT-GUERRERO, MD
Director of Perioperative Clinical Research
Duke Clinical Research Institute
Professor of Anesthesiology
Duke University Medical Center
Durham, North Carolina

DAN E. BERKOWITZ, MD
Professor, Department of Anesthesiology and Critical Care Medicine
Professor, Department of Biomedical Engineering
Johns Hopkins Medicine
Baltimore, Maryland

SIMON C. BODY, MBCHB, MPH
Associate Professor of Anesthesia
Harvard Medical School
Brigham and Women’s Hospital
Boston, Massachusetts

T. ANDREW BOWDLE, MD, PHD
Professor of Anesthesiology and Pharmaceutics
Chief of the Division of Cardiothoracic Anesthesia
Department of Anesthesiology
University of Washington
Seattle, Washington

MICHAEL K. CAHALAN, MD
Professor and Chair of Anesthesiology
University of Utah School of Medicine
Salt Lake City, Utah

ALFONSO CASTA, MD
Associate Professor
Anesthesia
Harvard University Medical School
Senior Associate in Cardiac Anesthesia
Children’s Hospital Boston
Boston, Massachusetts

CHARLES E. CHAMBERS, MD
Professor of Medicine and Radiology
Milton S. Hershey Medical Center
Pennsylvania State University School of Medicine
Hershey, Pennsylvania

MARK A. CHANEY, MD
Professor
Director of Cardiac Anesthesia
Department of Anesthesia and Critical Care
University of Chicago Medical Center
Chicago, Illinois

ALYSSA B. CHAPITAL, MD, PHD
Assistant Professor of Surgery
Department of Critical Care Medicine
Division Head of Acute Care Surgery
Mayo Clinic
Phoenix, Arizona

ALAN CHENG, MD
Assistant Professor of Medicine
Doctor, Arrhythmia Device Service
Johns Hopkins University School of Medicine
Baltimore, Maryland
CONTRIBUTORS

DAVY C.H. CHENG, MD, MSC, FRCPC, FCAHS
Distinguished University Professor and Chair
Department of Anesthesia and Perioperative Medicine
University of Western Ontario
Chief of Anesthesia and Perioperative Medicine
London Health Sciences Center and St. Joseph’s Health Care
London, Ontario, Canada

ALBERT T. CHEUNG, MD
Professor
Anesthesiology and Critical Care
University of Pennsylvania
Philadelphia, Pennsylvania

JOANNA CHIKWE, MD
Assistant Professor
Department of Cardiothoracic Surgery
Mount Sinai Medical Center
New York, New York

DAVID J. COOK, MD
Professor
Department of Anesthesiology
Chair, Cardiovascular Anesthesiology
Mayo Clinic
College of Medicine
Rochester, Minnesota

DUNCAN G. DE SOUZA, MD, FRCPC
Assistant Professor
Anesthesiology
University of Virginia
Charlottesville, Virginia

KAREN B. DOMINO, MD, MPH
Professor
Vice Chair for Clinical Research
Department of Anesthesiology and Pain Medicine
University of Washington
Seattle, Washington

MARCEL E. DURIEUX, MD, PHD
Professor
Departments of Anesthesiology and Neurological Surgery
University of Virginia
Charlottesville, Virginia

HARVEY L. EDMONDS, JR., PHD
Emeritus Research Professor
Anesthesiology and Perioperative Medicine
University of Louisville School of Medicine
Louisville, Kentucky

MARK EDWARDS, MBCHB, FANZCA
Anaesthetist
Department of Cardiothoracic and ORL Anaesthesia
Auckland City Hospital
Auckland, New Zealand

LIZA J. ENRIQUEZ, MD
Departments of Anesthesiology
Montefiore Medical Center
Bronx, New York

GREGORY W. FISCHER, MD
Associate Professor of Anesthesiology
Director of Adult Cardiothoracic Anesthesia
Mount Sinai School of Medicine
New York, New York

LEE A. FLEISHER, MD, FACC, FAHA
Roberts D. Dripps Professor and Chair of Anesthesiology
Professor of Medicine
University of Pennsylvania School of Medicine
Philadelphia, Pennsylvania

VALENTIN FUSTER, MD, PHD, MACC
Director, Mount Sinai Heart
Mount Sinai Hospital
Professor of Medicine
Mount Sinai School of Medicine
New York, New York

MARIO J. GARCIA, MD, FACC, FACP
Chief, Division of Cardiology
Montefiore Medical Center
Professor of Medicine
Albert Einstein College of Medicine
Bronx, New York

JUAN GAZTANAGA, MD
Director, Cardiac MRI/CT Program
Winthrop University Hospital
Mineola, New York

DEAN T. GIACOBBE, MD
Anesthesiologist
University Medical Center at Princeton
Princeton, New Jersey

LEANNE GROBAN, MS, MD
Associate Professor
Department of Anesthesiology
Wake Forest University School of Medicine
Winston Salem, North Carolina

HILARY P. GROCOTT, MD, FRCPC, FASE
Professor of Anesthesia and Surgery
University of Manitoba
St. Boniface Hospital
Winnipeg, Manitoba, Canada

KELLY GROGAN, MD
Associate Professor
Department of Anesthesia and Perioperative Medicine
Medical University of South Carolina
Charleston, South Carolina

ROBERT C. GROOM, MS, CCP
Associate Vice President of Cardiac Services
Director of Cardiovascular Perfusion
Maine Medical Center
Portland, Maine

DAVID W. GROSSHANS, DO
Assistant Professor
Department of Anesthesiology
Wake Forest University School of Medicine
Winston Salem, North Carolina

MASAO HAYASHI, MD
Fellow, Cardiothoracic Anesthesiology
Mount Sinai School of Medicine
New York, New York
<table>
<thead>
<tr>
<th>Name</th>
<th>Position and Affiliations</th>
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| Eugene A. Hessl II, MD, FACS  | Professor  
Department of Anesthesiology  
University of Kentucky College of Medicine  
Lexington, Kentucky                                                                                       |
| Benjamin Hibbert, MD, FRCPC  | Vascular Biology Lab Research Fellow  
Department of Biochemistry and Division of Cardiology  
University of Ottawa Heart Institute  
Ottawa, Ontario, Canada                                                                                      |
| Thomas L. Higgins, MD, MBA, FACP, FCCM | Professor of Medicine, Surgery, and Anesthesiology  
Tufts University School of Medicine  
Boston, Massachusetts  
Interim Chairman, Department of Medicine  
Departments of Medicine and Surgery  
Baystate Medical Center  
Medical Director, Inpatient Informatics  
Baystate Health  
Springfield, Massachusetts                                                                                       |
| Charles W. Hogue, Jr., MD     | Professor of Anesthesiology and Critical Care Medicine  
Chief, Division of Adult Anesthesia  
Johns Hopkins University School of Medicine  
Johns Hopkins Hospital  
Baltimore, Maryland                                                                                              |
| Jiri Horak, MD                | Assistant Professor  
Anesthesia and Critical Care  
University of Pennsylvania  
Philadelphia, Pennsylvania                                                                                      |
| Jay Horrow, MD, MS, FAHA      | Professor of Anesthesiology, Physiology, and Pharmacology  
Drexel University College of Medicine  
Professor of Epidemiology and Biostatistics  
Drexel University School of Public Health  
Philadelphia, Pennsylvania                                                                                      |
| Philippe R. Houmans, MD, PhD  | Professor, Department of Anesthesiology  
Mayo Clinic  
Rochester, Minnesota                                                                                             |
| Stuart W. Jamieson, MB, FRCS  | Endowed Chair and Distinguished Professor of Surgery  
Chief, Division of Cardiovascular and Thoracic Surgery  
Chair, Department of Cardiothoracic Surgery  
University of California, San Diego  
La Jolla, California                                                                                               |
| Mandisa-Maia Jones-Haywood, MD| Assistant Professor  
Anesthesiology  
Wake Forest University School of Medicine  
Winston Salem, North Carolina                                                                                      |
| Ronald A. Kahn, MD            | Professor  
Department of Anesthesiology  
Mount Sinai Medical Center  
New York, New York                                                                                                 |
| Joel A. Kaplan, MD, CPE, FACCC| Professor of Anesthesiology  
University of California, San Diego  
San Diego, California  
Dean Emeritus, School of Medicine  
Former Chancellor, Health Sciences Center  
University of Louisville  
Louisville, Kentucky                                                                                               |
| Jack F. Kerr, AIA             | Senior Healthcare Architect  
Array Healthcare Facilities Solutions  
King of Prussia, Pennsylvania                                                                                      |
| Kim M. Kerr, MD, FCCP         | Clinical Professor of Medicine  
Division of Pulmonary and Critical Care Medicine  
University of California, San Diego  
La Jolla, California                                                                                               |
| Oksana Klimkina, MD           | Department of Anesthesiology  
University of Kentucky Medical Center  
Lexington, Kentucky                                                                                               |
| Colleen Koch, MD, MS, MBA     | Professor of Anesthesiology  
Lerner College of Medicine of Case Western Reserve University  
Vice Chair of Research and Education  
Department of Cardiothoracic Anesthesia  
Cleveland Clinic  
Cleveland, Ohio                                                                                                     |
| Steven N. Konstad, MD, MBA, FACC| Chairman  
Department of Anesthesiology  
Maimonides Medical Center  
Brooklyn, New York  
Professor  
Anesthesiology  
Mount Sinai Medical Center  
New York, New York                                                                                                  |
| Mark Kozak, MD                | Associate Professor of Medicine  
Milton S. Hershey Medical Center  
Pennsylvania State University School of Medicine  
Hershey, Pennsylvania                                                                                               |
| Adam B. Lerner, MD            | Assistant Professor of Anesthesia  
Harvard Medical School  
Director, Cardiac Anesthesia  
Beth Israel Deaconess Medical Center  
Boston, Massachusetts                                                                                               |
| Jerrold H. Levy, MD, FAHA     | Professor and Deputy Chair for Research  
Emory University School of Medicine  
Director of Cardiothoracic Anesthesiology  
Cardiothoracic Anesthesiology and Critical Care  
Emory Healthcare  
Atlanta, Georgia                                                                                                     |
CONTRIBUTORS

MARTIN J. LONDON, MD
Professor of Clinical Anesthesia
University of California at San Francisco
San Francisco, California

BARRY A. LOVE, MD
Assistant Professor of Pediatrics and Medicine
Director of Congenital Cardiac Catheterization Laboratory
Mount Sinai Medical Center
New York, New York

FEROZE MAHMOOD, MD
Director of Vascular Anesthesia and Perioperative Echocardiography
Department of Anesthesia and Critical Care
Beth Israel Deaconess Medical Center
Boston, Massachusetts

GERARD R. MANECKE, JR., MD
Clinical Professor of Anesthesiology
Chair, Department of Anesthesiology
University of California, San Diego
La Jolla, California

CHRISTINA T. MORA MANGANO, MD, FAHA
Professor, Department of Anesthesia
Stanford University
Chief, Division of Cardiovascular Anesthesia
Stanford University Medical Center
Palo Alto, California

VERONICA MATEI, MD
Fellow
Department of Anesthesia
Yale University School of Medicine
New Haven, Connecticut

WILLIAM J. MAUERMANN, MD
Assistant Professor of Anesthesiology
Mayo Clinic
Rochester, Minnesota

TIMOTHY M. MAUS, MD
Assistant Clinical Professor of Anesthesiology
Director of Perioperative Transesophageal Echocardiography
University of California, San Diego
La Jolla, California

NANHI MITTER, MD
Assistant Professor
Adult Cardiothoracic Anesthesia Fellowship Program, Director
Anesthesiology and Critical Care Medicine
Johns Hopkins Hospital
Baltimore, Maryland

ALEXANDER J.C. MITTNACHT, MD
Director, Pediatric Cardiac Anesthesia
Associate Professor
Department of Anesthesiology
Mount Sinai Medical Center
New York, New York

EMILE R. MOHLER, MD, MS
Associate Professor of Medicine
University of Pennsylvania
Director of Vascular Medicine
University of Philadelphia Health System
Philadelphia, Pennsylvania

JOHN M. MURKIN, MD, FRCPC
Professor of Anesthesiology (Senate)
Director of Cardiac Anesthesia Research
Schulich School of Medicine
University of Western Ontario
London, Ontario, Canada

ANDREW W. MURRAY, MB, CHB
Assistant Professor
Department of Anesthesiology
University of Pittsburgh School of Medicine
Cardiac Anesthesiologist
University of Pittsburgh Medical Center–Presbyterian
Director of Cardio–Thoracic Anesthesiology
Veteran’s Administration Medical Center–Oakland
Pittsburgh, Pennsylvania

MICHAEL J. MURRAY, MD, PHD
Professor of Anesthesiology
Mayo Clinic College of Medicine
Consultant
Department of Anesthesiology
Mayo Hospital
Scottsdale, Arizona

HOWARD J. NATHAN, MD, FRCPC
Professor and Vice Chairman (Research)
Department of Anesthesiology
University of Ottawa
Ottawa, Ontario, Canada

GREGORY A. NUTTALL, MD
Professor of Anesthesiology
Mayo Clinic
Rochester, Minnesota

DANIEL NYHAN, MD
Professor
Division Chief, Cardiothoracic Anesthesia
Anesthesia and Critical Care Medicine
Johns Hopkins University
Baltimore, Maryland

EDWARD R.M. O’BRIEN, MD
Professor of Medicine, Cardiology
Research Chair, Canadian Institutes of Health Research/Medtronic
University of Ottawa Heart Institute
Ottawa, Ontario, Canada

WILLIAM C. OLIVER, JR., MD
Professor
Department of Anesthesiology
College of Medicine
Mayo Clinic
Rochester, Minnesota

PAUL S. PAGEL, MD, PHD
Professor of Anesthesiology
Director of Cardiac Anesthesia
Medical College of Wisconsin
Clement J. Zablocki Veterans Affairs Medical Center
Milwaukee, Wisconsin
ENRIQUE J. PANTIN, MD
Assistant Professor
Department of Anesthesiology
University of Medicine and Dentistry of New Jersey
Robert Wood Johnson Medical School
New Brunswick, New Jersey

JOSEPH J. QUINLAN, MD
Professor
Department of Anesthesiology
University of Pittsburgh
Chief Anesthesiologist
University of Pittsburgh Medical Center–Presbyterian
Pittsburgh, Pennsylvania

JAMES G. RAMSAY, MD
Professor of Anesthesiology
Director, Anesthesiology Critical Care
Emory University School of Medicine
Atlanta, Georgia

KENT H. REHFELDT, MD
Consultant
Assistant Professor of Anesthesiology
Department of Anesthesiology
Mayo Clinic
Rochester, Minnesota

DAVID L. REICH, MD
Horace W. Goldsmith Professor and Chair
Department of Anesthesiology
Mount Sinai School of Medicine
New York, New York

ROGER L. ROYSTER, MD, FACC
Professor and Executive Vice Chairman
Department of Anesthesiology
Wake Forest University School of Medicine
Winston-Salem, North Carolina

MARC A. ROZNER, PHD, MD
Professor of Anesthesiology and Perioperative Medicine
Professor of Cardiology
University of Texas MD Anderson Cancer Center
Adjunct Assistant Professor of Integrative Biology and Pharmacology
University of Texas Houston Health Science Center
Houston, Texas

JOSEPH S. SAVINO, MD
Professor of Anesthesiology and Critical Care
Vice Chairman, Strategic Planning and Clinical Operations
University of Pennsylvania School of Medicine
Philadelphia, Pennsylvania

ASHISH SHAH, MD
Assistant Professor of Surgery
Johns Hopkins University School of Medicine
Surgical Director, Lung Transplantation
Johns Hopkins Cardiac Surgery
Baltimore, Maryland

JACK S. SHANEWISE, MD, FASE
Professor and Director
Division of Cardiothoracic Anesthesiology
Columbia University College of Physicians and Surgeons
New York, New York

SONAL SHARMA, MD
Research Associate
Department of Anesthesiology
University of Virginia
Charlottesville, Virginia

STANTON K. SHERNAN, MD, FAHA, FASE
Associate Professor of Anesthesia
Director of Cardiac Anesthesia
Department of Anesthesiology, Perioperative, and Pain Medicine
Brigham and Women’s Hospital
Harvard Medical School
Boston, Massachusetts

LINDA SHORE-LESSERSON, MD
Professor of Anesthesiology
Chief, Cardiothoracic Anesthesiology
Montefiore Medical Center
Bronx, New York

NIKOLAOS J. SKUHAS, MD, FASE
Associate Professor of Anesthesiology
Director, Cardiac Anesthesia
Weill Cornell Medical College
New York, New York

THOMAS F. SLAUFTHER, MD, MHA, CPH
Professor and Head, Section on Cardiothoracic Anesthesiology
Wake Forest University School of Medicine
Winston-Salem, North Carolina

BRUCE D. SPIESS, MD, FAHA
Professor of Anesthesiology and Emergency Medicine
Director of VCURES
VCU–Medical College of Virginia
Richmond, Virginia

MARK STAFFORD-SMITH, MD, CM, FRCPC
Professor of Anesthesiology
Director of Fellowship Education
Director of Cardiothoracic Anesthesia and Critical Care Medicine Fellowship
Division of Cardiothoracic Anesthesia and Critical Care Medicine
Department of Anesthesiology
Duke University Medical Center
Durham, North Carolina

ALFRED H. STAMMERS, MSA, CCP, PBMT
Director of Perfusion Services
Division of Cardiothoracic Surgery
Geisinger Health Systems
Danville, Pennsylvania
MARC E. STONE, MD
Associate Professor of Anesthesiology
Program Director, Fellowship in Cardiothoracic Anesthesiology
Mount Sinai School of Medicine
New York, New York

KENICHI TANAKA, MD, MSC
Associate Professor
Anesthesiology
Emory University School of Medicine
Atlanta, Georgia

MENACHEM WEINER, MD
Assistant Professor
Anesthesiology
Mount Sinai School of Medicine
New York, New York

STUART J. WEISS, MD, PHD
Associate Professor of Anesthesiology and Critical Care
University of Pennsylvania School of Medicine
Philadelphia, Pennsylvania

JEAN-PIERRE YARED, MD
Director, Critical Care Medicine in the Heart and Vascular Institute
Cleveland Clinic Foundation
Cleveland, Ohio
FOREWORD

Nothing endures but change.
Heraclitus

The Next Frontier in Cardiac Surgery and Interventions

Medicine is in constant flux. Humans constantly are pushing the realm of scientific discovery into meaningful medical applications that ultimately alleviate suffering. The art and science of anesthesia care, as the practice of medicine, continues to progress significantly, especially in cardiac anesthesia. Our responsibilities have expanded beyond creating insensitivity to pain to the practice of sophisticated medical techniques based on fundamental scientific principles. As a specialty, we are much more involved in disease assessment and physiologic manipulation. The distinctions among anesthesiologist, diagnostican, and even interventionalist have blurred. The cardiac anesthesiologists’ pivotal role constantly is growing in the successful outcome of a patient population that is becoming ever more complex.

These advances in our specialty come from our ever-expanding knowledge of cardiopulmonary physiology, biochemistry, pharmacology, and neuroscience. However, much of our deeper understanding has come from advancements in technology. This edition of Kaplan’s Cardiac Anesthesia comes at a time that witnesses the practice of our subspecialty at a major crossroads. Cardiac surgery is undergoing a revolution in the way both simple and complex heart disease will be treated. Simultaneously, anesthesiology and cardiology are undergoing major advancements in imaging. Regional anesthesia now moves beyond the art of landmark assessment to the science of looking and guiding. In cardiology, it is fascinating to see that as new imaging or quantification technologies are brought online, new physiologic variables of the heart are discovered, rediscovered, or simply appreciated better. Moreover, newer imaging methodologies will serve as the eyes for catheter-guided hands in what can only be called a revolution in the development of new cardiac implantables and repair techniques that avoid sternotomy and cardiopulmonary bypass. Enter the “Echo Era.”

We have moved away from an era of palpation of the post-mitral repair thrill to sophisticated techniques to quantify a myriad of cardiac physiologic parameters. We are also moving away from an era of opening the chest to operate on the still heart. Newer image-guided procedures ultimately will lead to less invasive incisions, less infection, and less end-organ insult from cardiopulmonary bypass. Cardiopulmonary bypass will still predominate over the next few years, but this decade will witness an explosion of newer catheter-based techniques that avoid reanimating the nonbeating heart. Imaging will be the cornerstone of these new minimally invasive procedures. Advances in materials science and microelectronics ultimately will put three-dimensional eyes onto the tips of catheters, and these procedures will be performed by physicians who now operate inside the beating heart. Valve surgery is changing in a major way with adult senile calcific stenosis. Progressive change is accelerating transcatheter aortic valve intervention (TAVI). More than 20,000 cases have been performed. These procedures already avoid sternotomy and cardiopulmonary bypass to the point at which some patients are treated without endotracheal intubation and general anesthesia. Time will tell whether this procedure can be done safely. Nonetheless, the course is set and clear; cardiopulmonary bypass has brought us into the 21st century and imaging will advance us in the decades to come. Cardiac anesthesiologists now face a career-changing decision: will they embrace being key members of the new interventional team, or will they be content to be sideline observers of these new procedures?

The pivotal role of echocardiography as both monitoring and diagnostic tool evidenced itself in the 1990s with mitral valve repair. The technology revolution is only going to accelerate. New advancements will include technologies that look at structures with more detail in space and time. Ultimately, newer parallel-processing algorithms in beamforming and automated machine analysis of cardiac images will allow assessment of 3D regurgitant volume, myocardial contraction, and full four-chamber and valvular quantification. Because computers have become more powerful, imaging will be embraced only as it progresses in simplicity.

This new echo era will advance both diagnostics and therapeutic guidance. I have been most privileged that my path from medical student to cardiac anesthesiologist has been mentored by Drs. Kaplan, Reich, and Savino. This edition’s framework, penned by a world-renowned group of experts, not only is current and complete but also will equip its readers well for the dynamic ride to come.

Ivan S. Salgo, MD, MS
Chief of Cardiovascular Investigations, Ultrasound
Philips Healthcare
Andover, Massachusetts
The sixth edition of Kaplan’s Cardiac Anesthesia has been written to further improve the anesthetic management of the patient with cardiac disease undergoing both cardiac and noncardiac surgery. Since publication of the first edition in 1979, at the beginning of the modern era of cardiac surgery, continued advances in the field have made cardiac anesthesia the leading subspecialty of anesthesiology. To maintain its place as the standard reference textbook in the field, this edition has been completely revised, expanded, and updated throughout to reflect the ongoing changes in cardiovascular care, especially the rapid growth and use of ultrasound and other imaging technologies. Significant contributions to the text have been made by leading cardiologists and cardiac surgeons to fully cover the broader aspects of the total care of the cardiac patient.

This edition is subtitled The Echo Era to emphasize today’s expanded role of transesophageal echocardiography (TEE) and other ultrasound techniques in the perioperative period. The developments leading to the clinical use of TEE are described, and many of the authors discuss the expanding applications in monitoring and diagnosis by the modern cardiac anesthesiologist. Specific clinical situations are described using the decision-making process highlighted by Weiss and Savino: (1) framing the question asked of the anesthesiologist/echocardiographer; (2) collecting echocardiographic and nonechocardiographic information; (3) making the clinical decision based on integration of knowledge, framing, and information; and (4) implementing the recommendations after a full discussion with the surgeon and other clinicians (e.g., cardiologists).

These case discussions dealing with clinical decision making are augmented by the full-color presentation of the text, multiple color echo and Doppler images, cine clips, and supplementary material on the Expert Consult premium website accompanying the print version of the text. The website also will be used to update the book as new material appears between editions. Some of the new information will be provided by integrating key clinical areas first described in the Journal of Cardiothoracic and Vascular Anesthesia. The reader will be able to move seamlessly from the text to the new electronic information technology available with the book.

The content of the sixth edition ranges from the basic sciences through translational medicine to the clinical care of the sickest and most complex cardiac patients. The final section of this edition is entitled “Education in Cardiac Anesthesia” and emphasizes reducing errors to further improve the quality of our patient care. Training and certification in cardiovascular anesthesia are discussed, as well as the educational process and certification available for TEE. Because of the success of the new teaching aides used in the last edition, the Key Points of each chapter appear at the start of the chapters, and Teaching Boxes appear with many of the important “take-home messages.” The emphasis throughout the book is on using the latest scientific developments to guide proper therapeutic interventions in the perioperative period.

Kaplan’s Cardiac Anesthesia: The Echo Era was written by acknowledged experts in each specific area or related specialties. It is the most authoritative and up-to-date collection of material in the field. Each chapter aims to provide the scientific foundation in the area as well as the clinical basis for practice, and outcome information is included when it is available. All of the chapters have been coordinated in an effort to maximize the clinical utility. Whenever possible, material has been integrated from the fields of anesthesia, cardiology, cardiac surgery, physiology, and pharmacology to present a complete clinical picture. Thus, this edition should continue to serve as the definitive text for cardiac anesthesia residents, fellows, attendings, practitioners, cardiologists, cardiac surgeons, intensivists, and others interested in the management of the patient with cardiac disease for either cardiac or noncardiac surgery.

Cardiac anesthesia is a complex and comprehensive field of medicine, incorporating many aspects of the specialties of anesthesiology, cardiology, and cardiac surgery. Monitoring modalities always have been an integral part of the practice and have provided us with data to improve our therapeutic interventions. Over the past 30 years, these monitors have become progressively more sophisticated. Many of these monitoring techniques have been adapted from cardiologists and then applied to the cardiac surgical setting. This has been true of electrocardiographic monitoring, with the introduction of the V lead for the intraoperative detection of myocardial ischemia modified from its use during exercise tolerance testing. The pulmonary artery catheter (PAC) was developed for use in the coronary care unit by Dr. Swan, but as he told me, the perioperative use of the PAC in high-risk patients with heart failure and cardiogenic shock was a better role for it, and this use would outlast its role for cardiologists; it turned out to be very true!

Now, we have arrived at the echo era in which TEE—adapted from transthoracic echocardiography use in cardiology—is used widely in cardiac anesthesia for monitoring, diagnosis, and helping to guide the surgery in procedures such as mitral valve repairs. This technique certainly has led to changes in the operative procedures, as well as improvements in our care and choices of pharmacologic treatments, as pointed out in this edition. However, the practice of cardiac anesthesia is and always has been more than the interpretation of any one monitor. Those who believe and emphasize that obtaining certification in TEE makes an anesthesiologist into a cardiac anesthesiologist are sadly mistaken. The practice of cardiac anesthesia includes the use and interpretation of TEE, as it does with other monitors, but it also includes much, much more, and explains the overall size and depth of this book, incorporating all of the areas involved in the complete care of a cardiac surgical patient. It was this overall care in the perioperative period that led J. Willis Hurst, MD, one of the world’s leading cardiologists, to state, in his foreword to the first edition of Kaplan’s Cardiac Anesthesia, that “This cardiologist views the modern cardiac anesthesiologist with awe.”

The editors gratefully acknowledge the contributions made by the authors of each of the chapters. They are the dedicated experts who have made the field of cardiac anesthesia what it is today and are the teachers of our young colleagues practicing anesthesia around the world. This book would not have been possible without their hard work and expertise.

Joel A. Kaplan, MD, CPE, FACC
To the pioneers of cardiac surgery and anesthesia who have led us to this exciting era of techniques and technologies that continue to improve our patient care.

Joel A. Kaplan, MD, CPE, FACC
Electrocardiogram Atlas: A Summary of Important Changes on the Electrocardiogram

**Lead Placement**

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<td>V₆</td>
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LA, left arm; LL, left leg; RA, right arm.

**Normal Electrocardiogram—Cardiac Cycle**

The normal electrocardiogram (ECG) is composed of waves (P, QRS, T, and U) and intervals (PR, QRS, ST, and QT).

![ECG intervals diagram](image)
# Arrhythmias

## Sinus Tachycardia

**Rate:** 100–160 beats/min  
**Rhythm:** Regular  
**PR interval:** Normal; P wave may be difficult to see  
**QT interval:** Normal  
Note: Should be differentiated from paroxysmal atrial tachycardia (PAT). With PAT, carotid massage terminates arrhythmia. Sinus tachycardia may respond to vagal maneuvers but reappears as soon as vagal stimulus is removed.

## Premature Atrial Contraction

**Rate:** < 100 beats/min  
**Rhythm:** Irregular  
**PR interval:** P waves may be lost in preceding T waves; PR interval is variable  
**QT interval:** QRS normal configuration; ST segment and T wave normal  
Note: Nonconducted premature atrial contraction (PAC) appearance similar to that of sinus arrest; T waves with PAC may be distorted by inclusion of P wave in the T wave.

## Premature Ventricular Contraction

**Rate:** Usually < 100 beats/min  
**Rhythm:** Irregular  
**PR interval:** P wave and PR interval absent; retrograde conduction of P wave can be seen  
**QT interval:** Wide QRS (> 0.12 sec); ST segment cannot be evaluated (e.g., ischemia); T wave opposite direction of QRS with compensatory pause; fourth and eighth beats are premature ventricular contractions

## Multifocal Atrial Tachycardia

**Rate:** 100–200 beats/min  
**Rhythm:** Irregular  
**PR interval:** Consecutive P waves are of varying shape  
**QT interval:** Normal  
Note: Seen in patients with severe lung disease. Carotid massage has no effect. At heart rates < 100 beats/min, may appear as wandering atrial pacemaker. May be mistaken for atrial fibrillation.

## Paroxysmal Atrial Tachycardia

**Rate:** 150–250 beats/min  
**Rhythm:** Regular  
**PR interval:** Difficult to distinguish because of tachycardia obscuring P wave; P wave may precede, be included in, or follow QRS complex  
**QT interval:** Normal, but ST segment and T wave may be difficult to distinguish  
Note: Therapy depends on degree of hemodynamic compromise. Carotid sinus massage may terminate rhythm or decrease heart rate. In contrast with management of paroxysmal atrial tachycardia in awake patients, synchronized cardioversion rather than pharmacologic treatment is preferred in hemodynamically unstable anesthetized patients.
Sinus Arrhythmia

Rate: 60–100 beats/min  
Rhythm: Sinus  
PR interval: Normal  
QT interval: R-R interval variable  
Note: Heart rate increases with inhalation and decreases with exhalation ±10–20% (respiratory). Nonrespiratory sinus arrhythmia seen in elderly with heart disease. Also seen with increased intracranial pressure.

Atrial Fibrillation

Rate: Variable (~150–200 beats/min)  
Rhythm: Irregular  
PR interval: No P wave; PR interval not discernible  
QT interval: QRS normal  
Note: Must be differentiated from atrial flutter: (1) absence of flutter waves and presence of fibrillatory line; (2) flutter usually associated with higher ventricular rates (> 150 beats/min). Loss of atrial contraction reduces cardiac output (10–20%). Mural atrial thrombi may develop. Considered controlled if ventricular rate < 100 beats/min.

Atrial Flutter

Rate: Rapid, atrial usually regular (250–350 beats/min); ventricular usually regular (<100 beats/min)  
Rhythm: Atrial and ventricular regular  
PR interval: Flutter (F) waves are saw-toothed; PR interval cannot be measured  
QT interval: QRS usually normal; ST segment and T waves are not identifiable  
Note: Carotid massage will slow ventricular response, simplifying recognition of the F waves.

Wolff-Parkinson-White Syndrome

Rate: < 100 beats/min  
Rhythm: Regular  
PR interval: P wave normal; PR interval short (< 0.12 second)  
QT interval: Duration (> 0.10 second) with slurred QRS complex; type A has delta wave, RBBB, with upright QRS complex V1; type B has delta wave and downward QRS-V1; ST segment and T wave usually normal  
Note: Digoxin should be avoided in the presence of Wolff-Parkinson-White syndrome because it increases conduction through the accessory bypass tract (bundle of Kent) and decreases atrioventricular node conduction; consequently, ventricular fibrillation can occur.
**Sinus Bradycardia**

Rate: < 60 beats/min  
Rhythm: Sinus  
PR interval: Normal  
QT interval: Normal  

*Note: Seen in trained athletes as normal variant.*

**Sinus Arrest**

Rate: < 60 beats/min  
Rhythm: Varies  
PR interval: Variable  
QT interval: Variable  

*Note: Rhythm depends on the cardiac pacemaker firing in the absence of sinoatrial stimulus (atrial pacemaker 60–75 beats/min; junctional 40–60 beats/min; ventricular 30–45 beats/min). Junctional rhythm most common. Occasional P waves may be seen (retrograde P wave).*

**Atrioventricular Block**

(First-Degree)  
Rate: 60–100 beats/min  
Rhythm: Regular  
PR interval: P-wave normal; PR interval progressively lengthens with each cycle until QRS complex is dropped (dropped beat); PR interval following dropped beat is shorter than normal  
QT interval: Normal but may have widened QRS complex if block is at level of bundle branch. ST segment and T wave may be abnormal, depending on location of block  

*Note: Atrioventricular block represents complete failure of conduction from atria to ventricles (no P wave is conducted to the ventricle). The atrial rate is faster than ventricular rate. P waves have no relation to QRS complexes, for example, they are electrically disconnected. In contrast, with atrioventricular dissociation, the P wave is conducted through the atrioventricular node, and the atrial and ventricular rates are similar. Immediate treatment with atropine or isoproterenol is required if cardiac output is reduced. Consideration should be given to insertion of a pacemaker. See also a complication of mitral valve replacement.*

(Second-Degree) Mobitz Type I/ Wenckebach Block  
Rate: 60–100 beats/min  
Rhythm: Atrial regular; ventricular irregular  
PR interval: P-wave normal; PR interval progressively lengthens with each cycle until QRS complex is dropped (dropped beat); PR interval following dropped beat is shorter than normal  
QT interval: QRS complex normal but dropped periodically  

*Note: Commonly seen (1) in trained athletes and (2) with drug toxicity.*

(Second-Degree) Mobitz Type II  
Rate: < 100 beats/min  
Rhythm: Atrial regular; ventricular regular or irregular  
PR interval: P waves normal, but some are not followed by QRS complex  
QT interval: Normal but may have widened QRS complex if block is at level of bundle branch. ST segment and T wave may be abnormal, depending on location of block  

*Note: In contrast with Mobitz type I block, the PR and RR intervals are constant and the dropped QRS occurs without warning. The wider the QRS complex (block lower in the conduction system), the greater the amount of myocardial damage.*

(Third-Degree) Complete Heart Block  
Rate: <45 beats/min  
Rhythm: Atrial regular; ventricular regular; no relation between P wave and QRS complex  
PR interval: Variable because atria and ventricles beat independently  
QT interval: QRS morphology variable, depending on the origin of the ventricular beat in the intrinsic pacemaker system (atrioventricular junctional vs. ventricular pacemaker); ST segment and T wave normal  

*Note: Ativoventricular block represents complete failure of conduction from atria to ventricles (no P wave is conducted to the ventricle). The atrial rate is faster than ventricular rate. P waves have no relation to QRS complexes, for example, they are electrically disconnected. In contrast, with atrioventricular dissociation, the P wave is conducted through the atrioventricular node, and the atrial and ventricular rates are similar. Immediate treatment with atropine or isoproterenol is required if cardiac output is reduced. Consideration should be given to insertion of a pacemaker. See also a complication of mitral valve replacement.*

**Atrioventricular Dissociation**

Rate: Variable  
Rhythm: Atrial regular; ventricular regular; ventricular rate faster than atrial rate; no relation between P wave and QRS complex  
PR interval: Variable because atria and ventricles beat independently  
QT interval: QRS morphology depends on location of ventricular pacemaker. ST segment and T wave abnormal  

*Note: In contrast with atrioventricular dissociation, the atria and ventricles beat independently. The P wave is conducted through the atrioventricular node, and the atrial and ventricular rate are similar. In contrast, atrioventricular block represents complete failure of conduction from atria to ventricles (no P wave is conducted to the ventricle). The atrial rate is faster than the ventricular rate. P waves have no relation to QRS complexes; for example, they are electrically disconnected. Digitalis toxicity can present as atrioventricular dissociation.*
**Left Bundle Branch Block**

- **Rate:** <100 beats/min
- **Rhythm:** Regular
- **PR interval:** Normal
- **QT interval:** Complete LBBB (QRS > 0.12 second); incomplete LBBB (QRS = 0.10–0.12 second); lead V\(_1\) negative RS complex; I, aVL, V\(_6\) wide R wave without Q or S component; ST-segment and T-wave deflection opposite direction of the R wave

Note: Left bundle branch block (LBBB) does not occur in healthy patients and usually indicates serious heart disease with a poorer prognosis. In patients with LBBB, insertion of a pulmonary artery catheter may lead to complete heart block.

**Right Bundle Branch Block**

- **Rate:** <100 beats/min
- **Rhythm:** Regular
- **PR interval:** Normal
- **QT interval:** Complete right bundle branch block (RBBB; QRS > 0.12 second); incomplete RBBB (QRS = 0.10–0.12 second); varying patterns of QRS complex; rSR (V\(_1\)); RS, wide R with M pattern; ST-segment and T-wave opposite direction of the R wave

Note: In the presence of RBBB, Q waves may be seen with a myocardial infarction.
Torsades de Pointes

Torsades de Pointes: Sustained

Rate: 150–250 beats/min
Rhythm: No atrial component seen; ventricular rhythm regular or irregular
PR interval: P wave buried in QRS complex
QT interval: QRS complexes usually wide and with phasic variation twisting around a central axis (a few complexes point upward, then a few point downward); ST segments and T waves difficult to discern
Note: Type of ventricular tachycardia associated with prolonged QT interval. Seen with electrolyte disturbances (e.g., hypokalemia, hypocalcemia, and hypomagnesemia) and bradycardia. Administering standard antiarrhythmics (lidocaine, procainamide, etc.) may worsen torsades de pointes. Treatment includes increasing heart rate pharmacologically or by pacing.

Coarse Ventricular Fibrillation

Fine Ventricular Fibrillation

Ventricular Fibrillation

Rate: Absent
Rhythm: None
PR interval: Absent
QT interval: Absent
Note: "Pseudoventricular fibrillation" may be the result of a monitor malfunction (e.g., ECG lead disconnect). Always check for carotid pulse before instituting therapy.

Ventricular Tachycardia

Rate: 100–250 beats/min
Rhythm: No atrial component seen; ventricular rhythm irregular or regular
PR interval: Absent; retrograde P wave may be seen in QRS complex
QT interval: Wide, bizarre QRS complex; ST segment and T wave difficult to determine
Note: In the presence of hemodynamic compromise, immediate direct current (DC) synchronized cardioversion is required. If the patient is stable, with short bursts of ventricular tachycardia, pharmacologic management is preferred. Should be differentiated from supraventricular tachycardia with aberrancy (SVT-A). Compensatory pause and atrioventricular dissociation suggest a PVC. P waves and R' (V1) and slowing to vagal stimulus also suggest SVT-A.

Atrial pacing

Pacemaker Tracings

Atrial pacing as demonstrated in this figure is used when the atrial impulse can proceed through the atrioventricular node. Examples are sinus bradycardia and junctional rhythms associated with clinically significant decreases in blood pressure. (Arrows are pacemaker spike.)

Ventricular Pacing

In this tracing, ventricular pacing is evident by absence of atrial wave (P wave) and pacemaker spike preceding QRS complex. Ventricular pacing is used in the presence of bradycardia secondary to atrioventricular block or atrial fibrillation. (Arrows are pacemaker spike.)

DDD Pacing

The DDD pacemaker (generator), one of the most commonly used, paces and senses both atrium and ventricle. Each atrial and ventricular complex are preceded by a pacemaker spike.
**Coronary Artery Disease**

**Transmural Myocardial Infarction**

Q waves seen on ECG, useful in confirming diagnosis, are associated with poorer prognosis and more significant hemodynamic impairment. Arrhythmias frequently complicate course. Small Q waves may be normal variant. For myocardial infarction (MI), Q waves > 0.04 second and depth exceeds one third of R wave (inferior wall MI). For inferior wall MI, differentiate from right ventricular hypertrophy by axis deviation.

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Subendocardial myocardial infarction

Persistent ST-segment depression and/or T-wave inversion in the absence of Q wave. Usually requires additional laboratory data (e.g., isoenzymes) to confirm diagnosis. Anatomic site of coronary lesion is similar to that of transmural myocardial infarction electrocardiographically.

Myocardial Ischemia

Rate: Variable
Rhythm: Usually regular, but may show atrial and/or ventricular arrhythmias
PR interval: Normal
QT interval: ST segment depressed; J-point depression; T-wave inversion; conduction disturbances; coronary vasospasm (Prinzmetal)

ST segment elevation; (A) TP and PR intervals are baseline for ST-segment deviation, (B) ST-segment elevation, (C) ST-segment depression

Note: Intraoperative ischemia usually is seen in the presence of "normal" vital signs (e.g., ±20% of preinduction values).
Other Important ECG Changes

Digitalis Effect

Rate: < 100 beats/min
Rhythm: Regular
PR interval: Normal or prolonged
QT interval: ST-segment sloping (“digitalis effect”)

Note: Digitalis toxicity can be the cause of many common arrhythmias (e.g., premature ventricular contractions, second-degree heart block). Verapamil, quinidine, and amiodarone cause an increase in serum digitalis concentration.

Electrolyte Disturbances

<table>
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<th>Electrolyte Disturbances</th>
<th>↓ Ca²⁺</th>
<th>↑ Ca²⁺</th>
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<tr>
<td>QT interval</td>
<td>Increased</td>
<td>Decreased</td>
<td>T wave flat U wave</td>
<td>T wave peaked QT increased</td>
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Note: ECG changes usually do not correlate with serum calcium. Hypocalcemia rarely causes arrhythmias in the absence of hypokalemia. In contrast, abnormalities in serum potassium concentration can be diagnosed by ECG. Similarly, in the clinical range, magnesium concentrations rarely are associated with unique ECG patterns. The presence of a “u” wave (> 1.5 mm in height) also is seen in left main coronary artery disease, certain medications, and long QT syndrome.

Calcium

Hypocalcemia

Normal

Hypercalcemia
Potassium

Hypokalemia ($K^+ = 1.9$ mEq/L)

Hyperkalemia ($K^+ = 7.9$ mEq/L)

Hypothermia

Rate: < 60 beats/min  
Rhythm: Sinus  
PR interval: Prolonged  
QT interval: Prolonged

Note: Seen at temperatures less than 33° C with ST-segment elevation (J point or Osborn wave). Tremor caused by shivering or Parkinson disease may interfere with ECG interpretation and may be confused with atrial flutter. May represent normal variant of early ventricular repolarization. (Arrow indicates J point or Osborn waves.)

Subarachnoid Hemorrhage

Rate: < 60 beats/min  
Rhythm: Sinus  
PR interval: Normal  
QT interval: T-wave inversion is deep and wide. Prominent U waves are seen. Sinus arrhythmias are observed. Q waves may be seen and may mimic acute coronary syndrome.
Pericarditis

- Rate: Variable
- Rhythm: Variable
- PR interval: Normal
- QT interval: Diffuse ST- and T-wave changes with no Q wave and seen in more leads than a myocardial infarction.

Pericardial Tamponade

- Rate: Variable
- Rhythm: Variable
- PR interval: Low-voltage P wave
- QT interval: Seen as electrical alternans with low-voltage complexes and varying amplitude of P, QRS, and T waves with each heartbeat