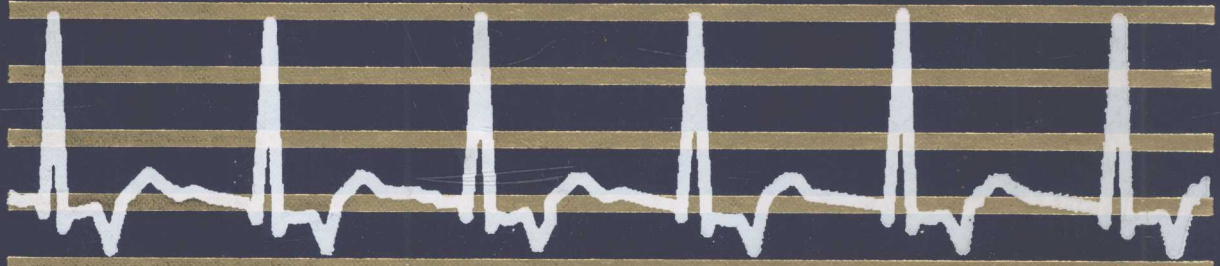
PERIOPERATIVE CARDIAC DYSRHYTHMIAS

MECHANISMS • RECOGNITION • MANAGEMENT



SECOND EDITION

JOHN L. ATLEE, III

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Perioperative Cardiac Dysrhythmias: Mechanisms, Recognition, Management

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To the memory of Peter E. Dresel, Ph.D., sorely missed teacher, counselor, and friend.

And to Barbara, Sarah, and Johnny, for great patience and understanding.

Foreword

As I predicted in my foreword to the first edition of this volume, a second edition has been written within 5 years! As I also predicted, the first edition filled a void in the anesthetic and critical care literature which had been conspicuous for a number of years. However, I am happy to say that, in my opinion, the second edition is a major improvement on the first edition. In addition to the expected increase in the number of references (almost 50%!), there also has been a major reorganization of the volume. In particular, Chapter 4 that covered only the surface electrocardiogram in the first edition has now been revised and enlarged to include other diagnostic techniques for electrocardiology and electrophysiology. In particular for the anesthesiologist, the inclusion of a detailed explanation of esophageal, intravascular and atrial electrocardiography, continuous ambulatory (Holter) monitoring, stress and exercise testing, the very precise electrophysiologic methods for studying arrhythmia genesis, and the expanded techniques of signal averaging and body surface mapping, are valuable. Pacemaker electrocardiography has been included in this chapter on electrocardiology and electrophysiology (instead of in a separate chapter), which I believe improves the flow of the book. In addition to the displacement of Chapter 8, Chapters 6, 7, and 9 have been completely reorganized and condensed into two chapters. Chapter 6, entitled *Methods for Treatment of Cardiac Dysrhythmias,* is basically a scientific and theoretical approach to the diagnosis and management of dysrhythmias, including an excellent section on the pharmacology and pharmacokinetics of the antidysrhythmic drugs, and a separate section on nonpharmacologic dysrhythmia management mostly related to electrical means of antidysrhythmia therapy including pacemaker, cardioversion, defibrillators and the newer implantable automatic defibrillators, and invasive treatment of dysrhythmias including surgical and catheter. The final chapter is now, as Dr. Atlee says, "a pragmatic approach to the management of perioperative dysrhythmias." The emphasis in this chapter is on the description of those situations likely to occur in the operating room, postanesthetic recovery room, and the intensive care unit, and specific methods of diagnosing and managing these dysrhythmias. Overall, I believe that not only has the second edition included advances made in field of diagnosis and treatment of cardiac arrhythmias, but also has been reorganized so that the volume will be more helpful to the practitioner.

As I predicted, Dr. Atlee has continued his investigations into the interaction of anesthetic drugs and cardiac arrhythmias. In fact, in an attempt to better investigate this field, he has shifted his base of operations across the state of Wisconsin from Madison to Milwaukee so that he could take advantage of the particularly fine electrophysiologic laboratory established in Dr. Kampine's department. I look forward to even more innovative research on the subject matter of this volume by Dr. Atlee and his new colleagues.

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Preface to the Second Edition

Perioperative Cardiac Dysrhythmias: Mechanisms, Recognition, Management has undergone extensive revision for this second edition. Much of the text has been rewritten and new material added, particularly in sections dealing with autonomic involvement in dysrhythmogenesis and nonpharmacologic approaches to dysrhythmia management. Some illustrations appearing in the first edition have been removed, and many new ones have been added. The list of references is far more extensive for most chapters. Those familiar with the first edition will note that Chapters 6 (Recognition of Perioperative Dysrhythmias) and 9 (Management of Specific Cardiac Dysrhythmias) are now combined into a single chapter (Recognition and Management of Specific Dysrhythmias) and that Chapter 8 (Cardiac Pacemakers and Direct-Current Cardioversion) has been incorporated elsewhere. For example, discussion relating to pacemaker terminology, design, and function appears in Chapter 4 under Pacemaker Electrocardiography. The indications for pacing and direct-current cardioversion, as well as their use in perioperative dysrhythmia management, are now found in Chapter 6 under Nonpharmacologic Dysrhythmia Management. Among other changes, these are some of the more important.

By and large, the purposes of the book remain the same. However, it is hoped that an additional objective can be met, which is, to provide a comprehensive source of information for cardiologists asked to consult in management of cardiac dysrhythmias in operating or recovery rooms, or in surgical intensive care units. Often, at least in my experience, cardiologists are not fully aware of how commonplace cardiac dysrhythmias are in these settings, how deleterious and difficult to treat they can be at times, or what their potential causes are. Chief among the causes for dysrhythmias in perioperative settings are autonomic imbalance and acutely altered physiologic states, in contrast to coronary artery disease, prodysrhythmia, and cardiomyopathies, which are more familiar to cardiologists. The material presented in Chapter 5 (Causes for Perioperative Dysrhythmias) should adequately support this contention. In addition, I note that anesthesiologists and intensivists are more focused on short-term aspects of management. It is hoped that with this assembled body of information our colleagues in cardiology can assist us with difficult aspects of perioperative dysrhythmia prevention and management.

Finally, I note with some sense of satisfaction that there seems to be renewed interest among anesthesiologists and investigators working in the field in the problem of cardiac dysrhythmias. Whether the first edition of this book had anything to do with this I cannot know, but I would like to think so. Regardless, I have tried in this revision to include as much material as possible, as well as relevant citations, in a book of reasonable length to further productive research in this field.

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Preface to First Edition

Anesthesiologists are frequently called upon to care for patients who are being managed with drugs, pacemakers, or both for preexisting cardiac dysrhythmias. Alternatively, patients who are otherwise healthy may develop cardiac rhythm disturbances when subjected to the stress of anesthesia and surgery. While cardiac dysrhythmias are commonplace in the perioperative setting and often relatively benign, they are serious when they compromise hemodynamic function, upset a favorable myocardial oxygen balance, or predispose to life-threatening dysrhythmias.

Reviews on the subject of cardiac dysrhythmias in the perioperative setting have appeared in the anesthesia literature from time to time, but there have been none for nearly 15 years. Within that time span, significant advances have been made by cardiologists, physiologists, pharmacologists, and engineers to further the understanding of mechanisms, enhance detection and differential diagnosis, and improve the treatment of cardiac dysrhythmias. While these advances have led to effective prevention and management of many cardiac dysrhythmias, most anesthesiologists have only a passing familiarity with them and consequently, are unable to employ them effectively in the management of their patients. Futhermore, the management of dysrhythmias in the perioperative environment brings into play a whole host of additional and potentially confounding variables.

The purposes of this book are several: (1) to explore recent advances made in the field of cardiovascular medicine with respect to the mechanisms, recognition, and management of cardiac dysrhythmias; (2) to review what is known about the effects of anesthesia and ancillary drugs on the electrical properties of the heart, and to relate these to the genesis of dysrhythmias; (3) to present those aspects of electrocardiography pertinent to the recognition of dysrhythmias in the perioperative setting; (4) to discuss the pharmacology of the drugs used in the acute as well as chronic treatment of dysrhythmias; (5) to review the current status of pacemaker technology, and to discuss the place of pacemakers and direct-current cardioversion in the management of cardiac dysrhythmias; and finally (6) to present a management scheme for cardiac dysrhythmias in the perioperative setting. By bringing together in one volume the many aspects of the problem of cardiac dysrhythmias, particularly as they relate to the circumstances of anesthesia and surgery and post-anesthetic recovery, it is hoped that anesthesiologists will be better able to manage patients entrusted to their care.

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Acknowledgment

A book of this sort cannot be written without the help and support of colleagues. In particular I express appreciation to the chairmen at the University of Wisconsin—Madison, Dr. S. Craighead Alexander, and the Medical College of Wisconsin in Milwaukee, Dr. John P. Kampine, for providing me the time and resources during the last 3 months of 1988 to complete the task of rewriting this book for its second edition. My sincere appreciation is also extended to colleagues, faculty and residents alike, in the Department of Anesthesiology at the University of Wisconsin—Madison, who provided either suggestions or rhythm strips illustrating dysrhythmias for the preparation of this edition; special thanks in this regard go to Drs. Ben Rusy, Mark Rice, Scott Springman, Steven Croy, Ed Violante, and Harvey Woehlck. Also, I wish to thank Dr. Wayne Grogan of the Division of Cardiology at the University of Wisconsin—Madison for his many valuable consultations and for providing some of the illustrations used in Chapter 7.

Four individuals are especially deserving of recognition for their valuable contributions to this edition of the book: Patricia Jacobson, Pamela Licht, Denise Roberts, and Edith Sulzer for preparation of the manuscript and for obtaining permissions, and Patricia Dvorak for preparation of illustrations and design of the book cover. Thank you Trish, Pam, Denise, Edith, and Trisha! Also, as was true for the first edition, I am especially grateful for the pleasant and efficient working relationship I have had with the editorial and production staffs of Year Book Medical Publishers. In particular, I wish to single out my sponsoring editor, Kevin Kelly, and editing manager, Fran Perveiler, for special thanks.

Finally, I wish to express my sincere thanks to my wife, Barbara, and children, Sarah and Johnny, for their support and encouragement during the difficult and time-consuming task of rewriting this book.

John L. Atlee, III, M.D.

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Perioperative Cardiac Dysrhythmias in Perspective

HISTORICAL INSIGHTS

The First Case

The first recorded death under anesthesia, that in 1848 of Hannah Greener, has been attributed to cardiac dysrhythmia. The dysrhythmia probably was ventricular fibrillation caused by the sensitizing action of chloroform to endogenously released catecholamines. Greener's death, which occurred just 15 months after Morton's public demonstration of the efficacy of ether anesthesia at Massachusett's General Hospital and just 2 months after Simpson's introduction of chloroform as an anesthetic, created great interest because it was the first death recognized as due to anesthesia. The following account of the events is taken from John Snow's[1] description of fatal cases of chloroform inhalation:

> The first death from chloroform was that of Hannah Greener, which occurred at Winlaton, near Newcastle, on the 28th of January, 1848. The patient was a girl of 15, who required to have the nail of the great toe removed. A similar operation had been performed on the other foot, in the previous November, in the Newcastle Infirmary, when ether was administered with a satisfactory result. The following is the account of the accident by Dr. Meggison, who administered the chloroform: "She appeared to dread the operation, and fretted a good deal: in fact, she commenced sobbing on our entering the house, and continued so until seated in the operating chair, and commencing the inhalation, which was done from a handkerchief on which a teaspoonful of chloroform had been poured. After drawing her breath twice, she pulled my hand from her mouth. I told her to put her hands on her knees, and breathe quietly, which she did. In about half a minute, seeing no change in breathing, or alteration of pulse, I lifted her arm, which I found rigid. I looked at the pupil and pinched her cheek, and, finding her insensible, requested Mr. Lloyd to begin the operation. At the termination of the semilunar incision she gave a kick or twitch, which caused me to think the chloroform had not sufficient effect. I was proceeding to apply more to the handkerchief, when her lips, which had been previously a good colour, became suddenly blanched, and she spluttered at the mouth, as if in epilepsy. I threw down the handkerchief, dashed cold water in her face, and gave her some internally, followed by brandy, without, however, the least effect, not the slightest attempt at a rally being made. We laid her on the floor, opened a vein in her arm, and the jugular vein, but no blood flowed. The whole process of inhalation, operation, venesection, and death, could not, I should say, have occupied more than two minutes."*

An autopsy was performed the day after death by Sir John Fife and Dr. Glover. The findings included "a very high state of" pulmonary congestion; dark fluid blood in both chambers of the heart, but very little in the left; more than usual congestion in the brain; and a stomach distended

**Lond Med Gazette* 1848; 41:255.

with food. Fife and Glover expressed the opinion that chloroform caused death by producing congestion of the lungs.

Snow,[1] who reviewed an additional 49 cases of fatal chloroform inhalation, commented on the mode of death in "chloroform syncope":

> In all cases in which the symptoms which occurred at the time of death are reported, there is every reason to conclude, as shown above, that death took place by cardiac syncope, or arrest of the action of the heart. In forty of these cases the symptoms of danger appeared to arise entirely from cardiac syncope, and were not complicated by the over-action of the chloroform on the brain. It was only in four cases that the breathing appeared to be embarrassed and arrested by the effect of chloroform on the brain and medulla oblongata, at the time when the action of the heart was arrested by it; and only in one of these cases (No. 42) that the breathing was distinctly arrested by the effect of the chloroform, a few seconds before the agent also arrested the action of the heart.

In reviewing these cases, as well as 109 cases collected by the Chloroform Committee of the Royal Medical and Chirurgical Society and published in 1864, it is worth noting the few instances of fatal chloroform inhalation where the administrators detected an irregular or rapid pulse prior to their detection of an absent pulse. However, knowledge of cardiac dysrhythmias, including means for detection, was lacking at that time, and hence the failure to recognize the probable cause of death as ventricular fibrillation and cardiac arrest.

Advent of ECG Monitoring

Ventricular fibrillation was first described by Erichsen[2] in 1842, induced electrically by Hoffa and Ludwig[3] in 1850, depicted vividly by MacWilliam[4] in 1887, and induced by the injection of adrenal extract into a dog anesthetized with chloroform by Oliver and Schafer[5] in 1895; it was not until 1912 that the first electrocardiogram (ECG) showing human ventricular fibrillation was published, by August Hoffman.[6] Kraus and Nicolai[7] first recorded ventricular extrasystoles with the ECG in 1908. Although the ECG is the sine qua non for dysrhythmia detection, it should not be assumed that prior to the inception of electrocardiography in 1903, by Einthoven, only ignorance existed in the realm of cardiac dysrhythmias. In 1902, James Mackenzie published *The Study of the Pulse,*[8] in which he compiled his studies of arterial and venous pulsations using his improved, clinical ink-writing polygraph. The reliable tracings of Mackenzie's polygraph were used to correct the inexact interpretations of ECGs in the early days of Einthoven[9] and Lewis.[10] Indeed, in the research of A. Goodman Levy,[11] first summarized in his presentation to the Section on Anaesthetics of the Royal Society of Medicine in 1914 and later published as a monograph, the arterial pulsations inscribed on a polygraph were the only recordings of ventricular dysrhythmias produced by varying means of stimulation in the cat. Levy, in his presentation to the Royal Society, was the first to provide an explanation of the mode of death in chloroform syncope (i.e., ventricular fibrillation), and this more than 50 years after Hannah Greener's death. However, much of our present understanding of the mechanisms for ventricular fibrillation under chloroform, as well as for ventricular dysrhythmias with other anesthetics, came from the pioneering work on anesthetic sensitization by Meek and co-workers[12] at the University of Wisconsin—Madison.

Anesthetic Sensitization in Perspective

Potentiation of epinephrine-induced ventricular dysrhythmias by the hydrocarbon inhalation anesthetics ("sensitization"*) has long been regarded as synonymous (or nearly so) with the problem of cardiac dysrhythmias occurring in the setting of anesthesia and surgery. This is not to suggest that other potential causes for dysrhythmias in the perioperative setting (e.g., hypoxia, hypercarbia, digitalis) were not also recognized. However, the compatibility of anesthetics and anesthetic adjuncts with epinephrine (see Chapter 5) has been and still is of some concern to anesthesiologists. Indeed, "sensitizing" anesthetics could not be introduced into clinical anesthetic practice today, and among the indicated preclinical testing would be determination of the agent's sensitizing properties. Even so, it has become necessary for anesthesiologists and intensivists to recognize other potentially important causes for cardiac dysrhythmias in perioperative settings. Many of these are unique to such settings (e.g., autonomic imbalance, reflex reactions, drug interactions; that is, they are different from the causes most familiar to internists and cardiolo-

*Katz and Bigger[13] regarded the term *sensitization* as inaccurate.

gists (e.g., myocardial ischemia or infarction, prodysrhythmic actions of antidysrhythmic drugs, acquired valvular or congenital heart disease). Consideration of the foregoing provides a justification of sorts for the existence of this book, which is to provide a reasonably complete source of information that focuses on the problem of perioperative dysrhythmias for physicians and investigators interested in the mechanisms, causes, recognition, and management of such dysrhythmias.

INCIDENCE

Reliable data for the incidence of cardiac dysrhythmias in perioperative settings (particularly, anesthesia and surgery) are limited to those studies where the ECG was *continuously monitored and recorded* ("complete" studies). Because of difficulties inherent in examining the copious data generated in complete studies (even with computer-aided dysrhythmia detection),[14, 15] it is not surprising that they are limited in size (fewer than 200 patients) and often to groups of patients undergoing selected surgical procedures. Representative studies in which continuous ECG recordings were used include dental procedures[14–18]; ear, nose, and throat surgery in children[19, 20]; and cystoscopy in men.[21]

Several studies have dealt with the incidence and types of dysrhythmia in large numbers of patients, including those of Dodd and colleagues[22] in 1962 (569 patients), Reinikainen and Pöntinen[23] in 1966 (1,196 patients), and Vanik and Davis[24] in 1968 (5,013 patients). In these studies the ECG was *continuously monitored* and displayed ("incomplete" studies). An overall incidence was given for the first and third of these studies, 29.9% and 17.9%, respectively. These incidence figures are lower then those found in complete studies, with continuously recorded ECG data,[14–21] probably because some lesser dysrhythmias (e.g., infrequent extrasystoles, atrioventricular [AV] junctional rhythms, wandering atrial pacemaker) might escape detection by even the most careful observer. Although incomplete studies underestimate the incidence of dysrhythmias, they still provide valuable information concerning types of dysrhythmia, relative incidence, predisposing and causative factors, and the relation to type of anesthetic or surgical procedure.

Incomplete Studies

Dodd and co-workers[22] found that supraventricular dysrhythmias (excluding sinus tachycardia) accounted for 68% and ventricular premature contractions 42% of observed dysrhythmias. Eighty percent of the supraventricular dysrhythmias were categorized as AV dissociation (including atrial rhythm, nodal rhythm, wandering pacemaker, and sinus arrest with supraventricular escape beats), and 17% as atrial premature contractions. The single most important factor in the development of dysrhythmia was *preexisting heart disease.* In patients without heart disease, neither advancing age nor primary inhalation anesthetic agent (including either, cyclopropane, halothane, and nitrous oxide [N_2O]) were related to the incidence of dysrhythmia in this study. Dysrhythmias occurred less frequently when thiopental was the primary anesthetic agent used. The principal findings of this study, as well as those of the other two incomplete studies described below, are shown in Table 1–1.

Reinikainen and Pöntinen[23] found a 24% incidence of dysrhythmia in 111 patients undergoing operations (mostly strabismus surgery in children) under local anesthesia, and the same incidence in 85 patients undergoing operations (major urologic and gynecologic surgery) under epidural anesthesia. With halothane, the incidence of dysrhythmia during maintenance was 13% in 582 patients, 362 of whom had ocular surgery. The overall incidence of dysrhythmia in 418 patients under neuroleptanalgesia (by inference, mostly ocular surgery) was 39%. In these same patients, incidence of dysrhythmia was 50% if atropine was included in the premedication and 31% if it was not. In addition, these authors found a twofold reduction in dysrhythmias caused by the oculocardiac reflex when respirations were controlled rather than spontaneous. Finally, these authors examined dysrhythmias during intubation in a series of 1,075 patients. In patients under halothane, dysrhythmias developed in 29% after intubation without relaxants and in 54% after intubation with succinylcholine. With the exception of ventricular extrasystoles in one patient and supraventricular tachycardia in two others, all dysrhythmias were of the slow supraventricular type (e.g., sinus bradycardia and arrest, isorhythmic AV dissociation,* AV nodal rhythm). With

* AV dissociation is not a true dysrhythmia but a phenomenon associated with certain types of dysrhythmias (see Chapter 4).

TABLE 1–1.
Dysrhythmia Incidence Studies in Which the ECG Was Continuously Monitored But Not Continuously Recorded: "Incomplete" Studies

Author (Yr)	No. of Patients	Overall Incidence	Anesthesia Effect on Incidence	Surgery Effect on Incidence	Other Findings
Dodd et al.[22] (1961)	569	29.9% (S, 68%, V, 42%)	Incidence lower when thiopental primary agent, but similar among other inhalation agents and with spinal anesthesia	Intra-abdominal (31.6%), extraabdominal (16.7%)	Incidence of dysrhythmias higher in patients with preexisting heart disease
Reinikainen and Pöntinen[23] (1966)	1,196	Not stated	*Local*, stabismus surgery in children (24%); *epidural*, urologic procedures (24%); *neucleptanalgesia*, ocular surgery (39%); *thiopental and SCh* for intubation (17%); *halothane and SCh* for intubation (54%); *halothane alone* for intubation (29%)	Not examined	Increased incidence of dysrhythmias with oculocardiac reflex when atropine included in premedication and with spontaneous respirations; most dysrhythmias related to intubation of the slow supraventricular type, except with thiopental and SCh, where 50% were of ventricular origin
Vanik and Davis[24] (1968)	5,013	17.9% (Serious, 0.9%)	Dysrhythmias of about equal incidence with halothane (16.9%), regional (19.1)%, or other (18.6%) anesthetic	Not examined	Higher incidence of new dysrhythmias in patients with heart disease or preexisting dysrhythmias; far greater incidence of serious dysrhythmias in patients with heart disease (3.9%), compared with those without (0.7%) or with preexisting dysrhythmias (0.4%); higher incidence with intubation: incidence of dysrhythmias higher with digitalis (43.2%) than without digitalis (17.3%)

SCh = succinylcholine; S = supraventricular; V = ventricular.