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**ARTICLE**

Personal and Public Safety Issues Related to Arrhythmias That May Affect Consciousness: Implications for Regulation and Physician Recommendations

A Medical/Scientific Statement From the American Heart Association and the North American Society of Pacing and Electrophysiology

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Patients with arrhythmias may experience complete or partial loss of consciousness, and questions about activities that are safe for them arise every day. Probably the most common question concerns the advisability of driving, because the safety of both patients and others may be threatened when personal or professional activities are performed by persons with arrhythmias that may impair consciousness. In view of the magnitude of this problem, the American Heart Association and the North American Society of Pacing and Electrophysiology (NASPE) cosponsored two conferences on medical and regulatory issues related to arrhythmias in the context of driving and other activities. The first conference, "Driving and Arrhythmias: Medical Aspects," was held at the NASPE Scientific Session on May 10, 1994, in Nashville, Tenn. The conference was chaired by William M. Miles, MD, and cochaired by Gregory A. Kidwell, MD, and Elizabeth J. Darling, MSN, RN. The second conference, "Personal and Public Safety Issues Related to Arrhythmias That May Affect Consciousness: Implications for Regulation and Physician Recommendations," was held January 12 through 13, 1995, in Washington, DC, and was chaired by Andrew E. Epstein, MD. The second conference had a broader focus and addressed regulatory and legal issues related to activities in patients with arrhythmias that may result in impaired consciousness. This statement is based on the proceedings of the two conferences.

Although ventricular arrhythmias are the most common cause of sudden cardiac death, both bradyarrhythmias and the usually more benign supraventricular arrhythmias can lead to syncope or sudden death. Furthermore, arrhythmia therapies themselves can interfere with day-to-day functioning as a consequence of reactions to drugs, shocks from implantable devices, and physical limitations after arrhythmia operations. Some arrhythmias can be cured, and others can only be

palliated. There are risks to both personal and public safety when patients with arrhythmias that may impair consciousness drive or perform public service functions such as commercial driving and commercial or military flying. Thus, efficacy of treatment and the implications of arrhythmia recurrence are especially important, regardless of the treatment approach, whether by drug, device, surgery, or catheterization.

It must be recognized that the goal of a zero-percent risk is unattainable. Indeed, society already accepts certain degrees of risk by allowing the young and elderly to drive.¹ What constitutes an acceptable risk is a function of the setting and activity in question. For example, although a person may have a quantifiable risk for arrhythmia recurrence, the consequences of such a recurrence will differ, depending on whether the driver is alone on a deserted country road or driving a school bus full of children. Where the welfare of the public is concerned, society has legislated and will continue to define what it sees as an acceptable risk. Sometimes the degree of risk lies on a continuum. For example, "threshold limit values," the legally allowable concentrations of toxic substances to which people can be exposed, are classified on the basis of assessments of what constitutes an undue risk to health. Similarly, measurable degrees of visual impairment exclude some persons from obtaining a driver's license. On the other hand, some medical problems, such as arrhythmias, occur paroxysmally, and a simple, single measurement, such as determination of the blood alcohol concentration, cannot be used to assign the risk of an event either at the instant of assessment or over time.

The rights of individuals, including acceptance of personal risk, compete with society's right to legislate the level of risk it considers acceptable for performance of certain activities by people who may cause harm to others. Any such policy must be fair to all persons, recognizing that restrictions may limit personal freedoms, job security, and feelings of well-being. Attendees at the two policy conferences appreciated these concerns.

Attendees also felt constrained by limitations of the available data with which to make recommendations. For example, reporting problems on morbidity and mortality related to traffic accidents plague the literature. Undoubtedly injuries to not only the drivers but also passengers and persons not involved in the accident vehicle are underreported. There is often a degree of uncertainty as to what, if any, medical problem contributed to an accident. Many times deaths that are a consequence of an accident are delayed and are not necessarily recorded in mortality statistics. Another limitation of the available data is that even if the risk of recurrent arrhythmias can be determined for a particular group of patients, the operational environments of specialized activities may increase or decrease the estimated risk. For example, military pilots are exposed to temperature extremes, acceleration forces during takeoffs and landings, hypoxia, and altered sympathetic states. Similarly, commercial drivers have irregular schedules and dietary habits, the stress of monotonous driving, and, depending on the requirements of their job, may lead relatively sedentary lifestyles.

Even though "cures" for a variety of arrhythmias are available through surgery and ablative techniques, many patients remain disabled because of insurance, liability, and work-related restrictions. To enable these people to reenter the work force, it is necessary to educate patients, employers, and the public. Although some therapies may be viewed as markers that identify patients for whom activities should be limited (for example, implantable cardioverter-defibrillators in patients who want to drive), the problem of impaired consciousness is at the crux of problems of regulation and patient management. Thus, this statement addresses the consequences of impaired mentation due to any arrhythmia (ventricular tachyarrhythmia, supraventricular tachyarrhythmia, or

bradyarrhythmia), because any may lead to impaired consciousness. *The objectives of this document are to (1) provide data to help estimate the risk of injury (to patients and others) attributable to arrhythmias and (2) provide recommendations about acceptable activities for patients with arrhythmias that may impair consciousness to protect both patients and the public from injury.*

In the sometimes subjective assessment of risk, the following issues were carefully considered: What is the particular rhythm diagnosis? Does the arrhythmia actually correlate with the symptoms? What is the probability of recurrence of arrhythmia? In the event of recurrence, what is the chance for syncope, incapacity, or an accident? What is the risk over time? How can patients at risk for arrhythmia recurrence be identified prospectively? Recognizing these limitations, this statement represents the best judgments and interpretations of the literature that attendees of the policy conferences could make. This statement is intended to represent a set of *guidelines*, not *practice standards*. However, it is intended to help standardize the assessment of patients with arrhythmias who perform potentially dangerous activities and to foster consistent use of the guidelines so that they can be revised as data become available in the future. This statement is intended to encourage not only safety but also vocations and avocations that are important to patients and society. It is hoped that a balance can be struck between arrhythmias, other diseases, occupations, and the consequences of impaired consciousness. Some flexibility must be retained, because the risks associated with arrhythmia recurrence must be placed on a continuum within the context of vocational, personal, and societal needs.

The consensus conference considered such issues as ethics of regulation; current regulations related to driving and flying; arrhythmias and recurrence rates; recommendations for allowing patients to return to driving, flying, or other potentially hazardous activities; and responsibilities of various interested parties in formulating public policy. Each of these aspects is summarized below.

ETHICS OF REGULATION

Ethics is a branch of philosophy that systematically and formally examines the rightness and wrongness of human acts, the logic used in ethical arguments, and the assumptions on which ethical decisions are based. Medical ethics deals with these issues as they relate to medical practice in the care of patients and their families, medical research, and setting public policies related to medical issues that impact society and its culture. Because of the tremendous impact of regulation of activities on individuals, attention is given to ethical issues related to regulation of patients with arrhythmias.

The virtuous individual does not want to harm another. Thus, if a person has a medical condition that could cause unconsciousness at any time, and if this risk cannot be controlled or eliminated by medical treatment, it is not unethical to enact guidelines or regulations preventing that person from engaging in such activities. Thus, an affected pilot may be prevented by regulation from piloting a plane without backup by a copilot free of such a medical condition. The same holds for the driver of a vehicle in a public transportation system. Indeed, the virtuous pilot or bus driver would automatically remove himself/herself from such work. Furthermore, it is also ethical for society to take action to prevent a person from putting others at undue risk of harm.

Just as all citizens in a society have certain claims and rights, so too do patients with arrhythmias, including pilots and bus drivers. From the standpoint of justice, these claims and rights must certainly equal those of all other persons with arrhythmias. However, groups do differ in their rights and claims in the social contract. Thus, anyone as an autonomous individual member of a society ethically has

the right to freely choose work that he or she wishes to do, to prove his or her competence in such work, and to be given the opportunity by society to do that work if such work is needed and he or she is physically able to do it. Although these are claims and natural rights of all autonomous citizens, society also has the right to protect itself from harm that might be done by those persons in their line of work. Individual autonomy does not supersede the right of society to protect itself from harm.

Justice demands that all individuals be treated alike. For instance, society is obligated to point out to a person who wishes to be a pilot that certain medical requirements must be met to perform the work. Society has the obligation to do this before that person enters a training program for pilots. Society is also obligated to make it known to all persons who wish to enter such programs that certain regulations govern physical requirements for continuing in such work. Conversely, a regulation to prevent persons with a defined medical condition, such as an arrhythmia that may impair consciousness, from performing certain activities must be applied to all persons with that condition. Because arrhythmias may develop after a person has entered his or her profession or field of work, the person who is removed from his or her job (piloting or driving, for instance) must be treated with justice. The employer who terminates a position for medical reasons must make every effort to minimize that individual's loss of economic power as well as emotional stress and pain.

Knowledge of a patient's condition is held in confidence in the doctor-patient relationship. Medical information may be released to a third party only when the patient has authorized it. If an employer does not use its own medical staff to evaluate an employee's fitness for certain activities, the physician may release information only with the permission of the patient. However, if the patient wishes to withhold medical information from his or her employer to avoid losing his or her job, the physician who knows the patient's condition, the responsibilities of the patient's job, and danger to other parties if the patient remains on the job may not ethically withhold this information. The physician must try to persuade the patient to give permission to release such information, but if the patient does not, it is ethical for the physician to break the rule of confidentiality if he or she knows that grave harm could result from his or her silence. While breaking the rule of confidentiality may lead to legal action by the patient against the physician, the ethical principles of beneficence and nonmaleficence outweigh the principle of confidentiality in such a situation. The responsible medical ethical action for the physician is to release the information to the proper authority with full disclosure to the patient.

REGULATIONS PERTAINING TO DRIVING AND FLYING

Driving

In the United States approximately 40 000 deaths occur annually as a result of motor vehicle accidents, a toll far greater than that associated with any other form of transportation. The frequency with which medical causes contribute to motor vehicle accidents in the United States is not known. However, the proportion is believed to be small (see "Arrhythmias and Recurrence Rates"), based on the Ontario, Canada, experience alluded to in the Conference on Cardiac Disorders and Commercial Drivers.² The likelihood of obtaining more precise data on arrhythmias as a cause of motor vehicle accidents is remote, due to the difficulty of documenting such events in the general population.

Commercial Driving Approximately 78% of all freight is hauled by US commercial trucking companies. In 1992 commercial trucking accounted for 288 billion road miles in the United States.

Despite increasing commercial traffic, fatal truck accidents fell by 50% per driven mile from 1983 to 1993. Forty-six percent of deaths from accidents are unrelated to collisions. It is likely that the US experience is comparable to that of Canada, given the similarity of equipment and environment. In Ontario, police records of the province suggest that approximately 50% of accidents involving commercial vehicles can be attributed to failure to follow the rules of the road (eg, speeding, failure to yield the right-of-way, etc.). An additional 15% to 20% are due to mechanical problems. Ten percent are the result of load shifting and 5% the result of other road users. Only 5% appeared to be due to loss of control such as might occur with an acute medical condition. However, even in the latter case, factors such as fatigue³ and possibly alcohol and drugs probably account for a large proportion, with medical causes infrequent.⁴

Experience in Europe also suggests that the impact of arrhythmia-induced loss of consciousness is a relatively minor factor in road accidents. Approximately 0.1% of reportable road accidents are attributed to medical causes, and only 10% to 25% of these are due to cardiological events. License revocation is recommended for commercial drivers for an arrhythmia that has caused syncope within the past 2 years or is deemed likely to cause sudden impairment of consciousness or distraction. A board (eg, the Driver and Vehicle Licensing Agency in Great Britain) can assess these standards as they apply to individual cases.

In the United States licensing of drivers and most aspects of road safety (except interstate commerce) are the responsibility of the states. In some cases the federal government attempts to influence state actions by withholding federal highway funds (eg, setting the legal age for drinking at 21 years or older, restricting driving speeds to 65 mph, implementing seat belt and helmet laws), or alternatively by using incentives (safety grant programs). With respect to interstate commerce, the US Department of Transportation through the Federal Motor Carrier Safety Regulation sets performance standards and attempts to define medical conditions that may keep a driver from safely operating a commercial motor vehicle. In regard to cardiac diseases, "It is the intent of the Federal Motor Carrier Safety Regulation to disqualify a driver who has a current cardiovascular disease which is accompanied by and/or likely to cause symptoms of syncope, dyspnea, collapse, or congestive heart failure. . . ." However, "the subjective decision of whether . . . an individual's condition will likely cause symptoms . . . rests with the medical examiner and the motor carrier."⁵

Commercial trucking is a highly distributed business in the United States, with more than 270 000 companies in operation. As a result, the medical examination process is of necessity diffuse. The skill level of examiners is variable, and in many cases the physicians performing examinations may not appreciate or fully understand each operator's job requirements. For instance, whether the driver is responsible for loading and/or unloading the vehicle as well as driving it may influence the impact of a medical condition on driving fitness. Additionally, heat, humidity, and fatigue are variables considered important by medical experts associated with the motor transport industry but may not be considered by many examining physicians.

Medical standards for US interstate commercial drivers with cardiovascular disease were last reviewed in detail approximately 10 years ago.² During a 2-day conference to update the approach to cardiological examinations (first adopted by the US Department of Transportation in 1970), cardiologists, occupational health physicians, and motor carrier industry experts met to review and propose modifications to the cardiovascular regulations and recommended test procedures. (See reference 2, section IV, "Dysrhythmias, Sudden Death, and Pacemakers" and section V,

“Cardiovascular Pharmacological Agents.”) In both cases the recommendations are somewhat vague, but a brief summary of the key points is provided in the Appendix.

Personal Driving Information on the frequency with which cardiac arrhythmias contribute to automobile accidents is sparse. Furthermore, given the inherent difficulty of determining an arrhythmic cause of accidents, available data can only be considered a rough estimate. For example, among 3000 cases surveyed from the records of the Medical Control Unit of the Virginia Commonwealth Department of Motor Vehicles, only 6% were deemed related to cardiovascular disease, and of these, only a fraction (14%) appeared to be associated with cardiac arrhythmias.⁶ If these numbers can be verified in other jurisdictions, it would appear that cardiac arrhythmias are infrequent contributors to motor vehicle accidents. Whether high-risk population subsets (eg, patients with implantable cardioverter-defibrillators) are responsible for a higher frequency of arrhythmia-related automobile accidents is as yet unproved.

In the United States medical criteria for driving fitness are determined by the individual states. No established criteria are widely applicable at present. Recommendations from the 1992 Consensus Conference of the Canadian Cardiovascular Society⁷ provide a basis for formulating recommendations for commercial and personal driving.

Nonmilitary Flying

In the United States the Federal Aviation Administration (FAA) is responsible for medical certification of more than 650 000 aviators with US licenses. Given the frequency with which medical certification is required (every 6 months for first class, annually for second and third class), the FAA Aeromedical Certification Division processes 1900 new or renewal applications daily. Fewer than 1% are denied for failure to meet medical standards and are not eligible under special issuance provisions. In Europe the aviation environment is regulated through the Joint Aviation Authorities (JAA), an international agency encompassing 26 countries. A comprehensive medical standard has been developed. The Joint Aviation Requirements became effective early in 1996.

According to findings from the Aeromedical Office of the Airline Pilots Association, each year approximately 42 persons with rhythm disturbances contact the office. Just over one half of these persons have experienced syncopal episodes, with 5 to 10 in-cockpit syncopes per year. In a review of 102 syncopes over 5 years, less than half were believed due to ventricular arrhythmias. The majority of individuals with ventricular arrhythmias were permanently disqualified from flying. On the other hand, most individuals with syncope believed to be bradyarrhythmic returned to flying after evaluation.

United States (FAA) Arrhythmia Standards The most frequently reported arrhythmias in air personnel are ventricular and atrial premature beats, atrial fibrillation, and paroxysmal supraventricular tachycardia. Other disturbances, including abnormalities of intraventricular conduction, preexcitation, and sinus node dysfunction are less prevalent. Recognizing that a comprehensive aeromedical appeals process exists for cases in which certification is denied for medical reasons, the following summarizes the current FAA view on eligibility for flying with a detected arrhythmia:

Paroxysmal reentrant and nonparoxysmal supraventricular tachycardias are not considered a problem when treated by acceptable means. An exception is multifocal atrial tachycardia, which is

considered a cause for disqualification because of its close association with significant underlying cardiac or noncardiac disease.

Atrial fibrillation and atrial flutter are not disqualifying conditions if the individual is considered to be at low risk for embolic events.

Complex ventricular arrhythmias may be acceptable if there is no evidence of underlying heart disease.

Sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) are considered disqualifying conditions regardless of treatment.

Right bundle branch block (RBBB) of recent onset requires exclusion of underlying structural heart disease.

Fascicular blocks and left bundle branch block (LBBB) are disqualifying conditions unless structural heart disease is excluded.

Ventricular preexcitation (eg, Wolff-Parkinson-White syndrome) is a disqualifying condition if there is a preceding history of arrhythmia or evidence of structural heart disease.

As a rule, medical certification of air personnel is possible after thorough evaluation and demonstration of successful treatment during close follow-up.

European (JAA) Arrhythmia Standards Cardiovascular causes are the most common cause of loss of flying license in Western Europe, and cardiac arrhythmia is the main disqualifier in a substantial proportion of these (eg, 48 of the 69 Class 1 problem cases reviewed by the Medical Advisory Council in the United Kingdom between 1984 and 1992). Frequent ventricular premature beats, nonsustained VT, and paroxysmal atrial fibrillation were the most common problem arrhythmias.

European experience indicates that the best commercial airlines using the latest model aircraft achieve more than 2 million (2×10^6) flying hours between fatal accidents, whereas the worst have such an accident every 250 000 flying hours.⁸ Accidents due to medical causes are uncommon; 80% of all accidents and 60% of fatal accidents are due to human error, including design problems.⁸ On the other hand, transient medical incapacitation during flight is relatively common; a survey by the International Federation of Airline Pilots summarized by Bennett⁸ indicates that one third of pilots have had such an experience (59% due to gastrointestinal problems), but in only 3% might there have been a safety hazard. In the vast majority of cases sufficient warning was available to permit the copilot to take over. A review by the International Air Transport Association of 36 000 pilots at risk over 10 years found 26 cardiovascular or neurological medical events that could have jeopardized safety had they occurred at a critical time (ie, takeoff or approach), but none did.

The European approach to medical standards for flying fitness became uniform in 1996 and incorporates an attempt to define acceptable risk (ie, a "safe enough" criterion, given the obvious inability to eliminate all risk from flying).⁹ The risk criterion states that for multicrew operations (ie, largely commercial, JAA Class I) the fatal accident rate should not exceed one event in 10^7 flying hours, and that medical causes of such events should not exceed 1% of the total (ie, "the one-percent rule"; medical causes of fatal events would not exceed one every 10^8 to 10^9 hours). The estimate takes into account the cardiovascular mortality of middle-aged men and is based on duration of critical segments of flight operation (ie, takeoff, descent, approach) being about 10% of total flight duration. In the 5 years before the report (1992) the fatal accident rate averaged 1 every 6×10^5 hours. The cardiological-cause accident rate was approximately 1 every 2.5×10^8 hours.

For single-crew operation, in which the fatal accident rate is likely to equal the serious incapacitation rate, the observed accident rate is 1 event in 10^5 flying hours. By extrapolation the medically related accident risk should not exceed 1 event in 10^7 flying hours. This latter goal is apparently being achieved currently.

The medical standards for private flying (JAA Class II) are also considered. At the time of the report the fatal accident rate in this category was 1 every 40 000 to 50 000 hours flown, approximately 10 to 20 times worse than for scheduled airlines.^{8 9} It was recommended that medical causes be responsible for no more than 1 in 25 to 50 fatal accidents (compared with 1 in 100 for Class I licenses).

The following provides an overview of the Joint Aviation Requirements standard^{9 10} as it applies to arrhythmias and cardiac conduction disturbances. An appeals process is detailed to review problem or disputed cases on an individual basis:

Atrial arrhythmias. The standard disqualifies applicants with significant persistent or paroxysmal atrial rhythm disturbances pending cardiological assessment. In the case of atrial fibrillation, evaluation guidelines are specific. Recertification requires a normal echocardiogram, satisfactory completion of at least three stages of a Bruce protocol graded exercise test (with no other arrhythmia or conduction disturbance), completion of several ambulatory electrocardiographic recordings (Holter type) in which, if atrial fibrillation is present, there are no RR intervals less than 300 ms or greater than 3 seconds. In the case of a single episode of atrial fibrillation with an apparent cause, restricted recertification is permitted with arrangements for regular medical assessment. If there are no further attacks, full certification is permitted only after 2 years. Additional attacks would result in restricted certification only if suppressed or asymptomatic.

Sinoatrial disease. Applicants with asymptomatic sinus bradycardia and tachycardia may be assessed as fit in the absence of significant underlying pathology. Evidence of sinoatrial disease requires a thorough standard evaluation.

Ventricular arrhythmias. Asymptomatic isolated uniform ventricular ectopic beats are not disqualifying. However, frequent or complex forms require cardiological evaluation. A ventricular rhythm disturbance should not exceed 2% of the total QRS count. Coronary angiography is recommended if there are any doubts about structural heart disease.

Conduction disturbances. Incomplete bundle branch block or stable left-axis deviation is not disqualifying in the absence of other abnormalities. Applicants with complete RBBB or LBBB require complete cardiological evaluation.

Preexcitation syndromes. Preexcitation syndromes are considered disqualifying pending medical assessment.

Patients with pacemakers. Pacemaker patients are considered unfit unless specific medical evaluation provides evidence to the contrary. Specific guidelines are incorporated into the standard.

Military Flying

The US military services own and operate “public use” aircraft, a designation that is distinct from commercial or private aircraft. By law, agencies owning and operating public use aircraft—not the FAA—are responsible for aircrew medical certification. In regard to arrhythmias and fitness for military flying, the US Army and Air Force have a joint policy that strives to take into account the particularly uncertain and stressful nature of operational circumstances, along with concerns regarding potential

environmental extremes, physical stress, circadian disturbances, and the possible subjection of individuals to substantial acceleration forces.

The principal objective of screening for cardiovascular disease and arrhythmias in the military situation is detection before symptoms are manifested. As a result, abnormal test results, not clinical symptoms, are often the main concern.

Standards Related to Specific Arrhythmias^{11 12}

Bradyarrhythmias Sinus bradycardia is common in this selected physically fit population. Nonetheless, persons with a heart rate of 40 beats per minute (bpm) or lower undergo noninvasive cardiac evaluation, and a selected group may undergo electrophysiological testing, although the need for the latter is rare. Aircrew members with normal exercise heart rate response and no evidence of sinus node dysfunction are permitted to fly. Those with sinus node dysfunction and those requiring pacemakers are restricted from flying.

Detection of sinus pauses is followed by Holter monitoring and exercise testing. Asymptomatic and infrequent pauses of less than 4 seconds' duration are not disqualifying.

Atrial Arrhythmias Detection of premature atrial contractions leads to noninvasive cardiac assessment, including ambulatory electrocardiographic monitoring and possibly echocardiography and exercise testing. Aggravating factors such as thyroid disease, nicotine, or caffeine are also addressed. Persons with benign findings are permitted to fly. Others are considered on a case-by-case basis.

Treatment of atrial fibrillation, atrial flutter, and multifocal atrial tachycardia is similar to the supraventricular tachycardias described below. In military aviation experience multifocal atrial tachycardia is usually nonsustained and benign, and consequently it is treated the same as other supraventricular tachycardias, depending on the presence of underlying structural heart or pulmonary disease.

Supraventricular Tachycardias Three or more consecutive supraventricular tachycardia (SVT) beats result in restriction from flying, pending investigation. Those with a single episode (3 to 10 beats in duration) can return to flying if they have three normal Holter recordings over a 3-month period, normal thyroid function, and normal echocardiogram and exercise tests. A greater than 10-beat run of SVT triggers more detailed evaluation that may include angiographic and electrophysiological studies. As a general rule, current policies disqualify only those aviators with hemodynamic symptoms associated with SVT, recurrent sustained SVT, or SVT with ventricular preexcitation. Aircrew with SVT in the setting of cardiac disease or preexcitation or who have hemodynamically unstable SVT or recurrent sustained SVT are disqualified from flying duties.

Aircrew with preexcitation syndromes (eg, Wolff-Parkinson-White syndrome) who have undergone successful radiofrequency (RF) ablation for standard medical indications may return to flying duties. Radiofrequency ablation is not recommended for aeromedical status alone in the absence of a standard medical indication. Success must be documented by multiple normal Holter tracings over a 6-month period as well as a normal follow-up electrophysiological study. It should be noted that this policy applies only when arrhythmias are detected in persons who have already been trained. When preexcitation is found before training, such persons are not accepted for flying duties.

Premature Ventricular Contractions Holter studies are conducted when premature ventricular contractions (PVCs) are found. If frequent, noninvasive assessment follows. Those considered at risk for ischemic heart disease also undergo angiography. In general, infrequent PVCs are not disqualifying in the absence of evident cardiac disease. Others are assessed on an individual basis. Recent reevaluation of the medical literature has led to the recommendation that frequent ventricular ectopy or couplets are not cause for disqualification.

Ventricular Tachycardia Ventricular tachycardia, defined as three or more consecutive ventricular complexes at a rate of 100 bpm or greater, is cause for restriction from flying pending investigation. Those with only a single run of nonsustained VT, based on three Holter recordings over a 3-month period and no evident cardiac disease (including normal echocardiogram and exercise study and normal angiography in persons older than 36 years) may return to low-performance flying. Those with recurrent VT, sustained VT (30 seconds or longer), or VT in the presence of cardiac disease are disqualified.

Nonsustained VT presents a particular dilemma in the military flying environment. Currently the prognosis of these patients from a mortality perspective is considered benign with respect to persons with more serious disease. However, the risk of syncope and presyncope is largely unknown (yet relevant for fliers), and the mortality risk compared with a healthy young control group (ie, other military aviator candidates) is unclear.

Conduction Disturbances First-degree atrioventricular (AV) block that normalizes with exercise is not disqualifying, and aircrew members with this condition are permitted to return to flying. Mobitz type I AV block is also considered a normal variant and does not affect flying duties. Flying duties are restricted in the presence of Mobitz type II or third-degree AV block.

Right bundle branch block with a normal echocardiogram does not exclude entry into flight training. Similarly a pilot with this finding is returned to duty if a noninvasive examination including echocardiography, exercise testing, and Holter monitor recording is otherwise normal. Left bundle branch block results in restriction from flying pending the outcome of noninvasive studies, coronary angiography, and electrophysiological testing. Normal results of these studies can lead to resumption of flying duties; abnormal findings result in restriction from flying.

ARRHYTHMIAS AND RECURRENCE RATES

Overview of Risk

Various arrhythmias may be associated with syncope. If the person is driving a motor vehicle or flying an airplane, loss of consciousness could result in death or injury to one or many people. Because the data regarding such risk for specific arrhythmias are meager, a general overview is presented on the incidence of traffic fatalities due to cardiac and other causes.

The available data do not support the contention that sudden cardiac death while driving is a significant public safety issue. In 1960 Norman¹³ noted that in 220 000 driver years, of 46 drivers who lost consciousness at the wheel, only 14 did so due to a cardiac cause. In 12 of the 14 cases the vehicle was moving, but only three accidents occurred, and no one other than the driver died. These findings were supported by Trapnell and Groff,¹⁴ who reported 50 cases of myocardial infarction in truck drivers. Infarction recurred in five, including four recurrences during driving that resulted in death. However, no accidents occurred, and each of the four persons who died had stopped their

vehicles beforehand for a variety of reasons. Herner et al¹⁵ reported that 41 of 44 255 road accidents in one region of Sweden during the years 1959 to 1963 were probably caused by sudden illness in the driver. Of these 41 events, 10 were attributed to epilepsy, seven to myocardial infarction, and the remainder to a variety of other causes. Notably, eight of the drivers died at the wheel as a result of disease, including all seven with myocardial infarction. However, in the latter cases, no one else was injured; there were four instances of property damage. From 1 to 3 per 1000 accidents are caused by driver impairment.¹⁵ There are reports of driver-related deaths resulting in the death of others.¹⁶

In 1964 Myerburg and Davis¹⁷ reported 1348 incidents of death in Florida due to coronary artery disease. One hundred twenty-two patients were designated as having a hazardous occupation, including 75 persons (61.4%) responsible for ground transportation (truck drivers, 30.3%; taxi drivers, 16.4%; bus and streetcar operators, 7.4%; and various others, including railroad workers, ambulance drivers, and chauffeurs). Thirteen (10.7%) were on sea crews, and 8 (6.6%) were on aircrews (airline transport pilots, 2.5%; Air Force pilots, 2.5%; airline flight engineers, 0.8%; and commercial pilots, 0.8%). Of 52 persons who died while driving private automobiles, 32 were able to drive their vehicle to the side of the road without an accident. In 15 cases there were minor accidents that caused only property damage but no bodily injury. There was no incidence of major property damage, major bodily injury, or fatalities to others in this series. Similarly there were only two minor accidents among the 15 men who died while driving trucks. Of two pilots who died in flight, one was a commercial pilot; the aircraft was landed by the crew without incident. The other was a young student pilot in a single-engine plane who died probably because of myocardial infarction. The notable conclusions were that of 122 persons who died suddenly of coronary artery disease and who held occupations that involved public transportation (9.1% of all sudden deaths), no serious accidents occurred as a consequence. Likewise, among the 101 persons (7.5% of the population) who were performing a hazardous activity at the time of death, including 52 who were driving cars, no serious injury to others occurred.

Similar observations have been made by others.^{18 19 20 21 22} Peterson and Petty¹⁹ analyzed all driver fatalities examined at the Office of the Chief Medical Examiner in Baltimore, Md, during the 4-year period from 1956 to 1960. Of 81 cases (80 drivers and 1 passenger), 36 drivers collapsed at the wheel and had an accident as a consequence of an earlier medical illness. The remaining 45 were dead or moribund at the wheel of an automobile that had not been in an accident. Of the drivers with accidents, 28 of 36 deaths were due to heart disease (others were attributed to dissecting aortic aneurysm [2], ruptured aortic aneurysm [1], and stroke [5]). Of the drivers without accidents, 42 of 44 were due to heart disease (2 were due to stroke). The 1 passenger death was due to aspiration. Thirty-six deaths occurred in drivers, and 21 resulted in damage to fixed objects or parked automobiles. In 13 cases moving vehicles were hit. In the remaining 2 cases the automobile left the road and hit no other object. In none of these 36 cases was a person other than the driver injured.

In 1974 Hossack²⁰ reported that 11 of 102 drivers who underwent autopsy died of natural causes at the wheel of their automobile. Five of the drivers were able to stop their automobiles before they died, thereby preventing injury. The other 6 accidents were minor with no injury to others. Ostrom and Eriksson²¹ reported 126 cases of sudden death in persons driving in Sweden. That number included persons riding bicycles and motorcycles. The majority (112 of 126) had ischemic heart disease. Among 31 persons at risk during the driver's loss of consciousness, 2 suffered minor injuries, and no other road user was hurt.

Although the occurrence of death while performing a dangerous activity appears dramatic, these reviews suggest that the incidence of injury to others is very low. Furthermore, these injuries must be

placed in the context of the overall risk attributable to driving. In 1968 Grattan and Jeffcoate²³ concluded that chronic disease contributed to only 0.5% of 593 injury-related traffic accidents. In 1986 Parsons³ analyzed 131 press reports and the records of 92 patients of a neurological clinic from the perspective of loss of consciousness while driving. According to press accounts of persons who collapsed at the wheel, 50% of the events occurred in patients with coronary artery disease, and 29% simply fell asleep at the wheel without any evidence of an organic cause for loss of consciousness. Notably this resulted in 0.05 and 1.55 traumatic deaths per incidence, respectively, attesting to the fact that falling asleep and fatigue represent a much greater risk for injury than does sudden death due to arrhythmias or coronary artery disease.

Alcohol is also a major contributor to sudden death during driving. West et al¹⁶ reported in 1968 that 648 of 871 drivers (74%) who died as a result of an accident had a blood alcohol level of 0.19 g/100 mL. The authors extended these initial observations and analyzed the 155 drivers (15% of 1026) who died at the wheel due to natural causes and from which accidents resulted. Of the 96 drivers whose blood alcohol levels were measured, 20 had positive levels, 18 of which were more than 0.1 g/100 mL. It was further observed that high blood alcohol levels in the driver increased the risk of death to others. In the 155 accidents 1 passenger was killed and 18 were injured, 4 seriously. Four of 5 drivers in whose vehicles passengers were killed or injured had high blood alcohol levels.

Age is another factor. Larsen et al²⁴ observed that in Oregon, persons 16 to 19 years old had 0.9 accidents per 100 drivers per month compared with survivors of VT and VF who had event rates of 1.8% and average monthly risks of 1.1%, respectively, 1 year after discharge from the hospital. Eight to 12 months after discharge the respective rates were only 0.8% and 0.4%, rates similar to those of all licensed Oregon drivers (0.4%) and not different from the risk that society tolerates for teenagers behind the wheel. Conversely, an increased risk for death among the elderly has been recognized for years. In 1967 Waller²⁵ reported that healthy persons aged 30 to 59 years had 9.1 accidents per 1 million miles driven compared with 36.2 accidents per 1 million miles driven in persons aged 60 or older with cardiovascular changes and senility. Rigdon¹ noted a similar distribution of increased fatalities for the young.

In summary, sudden death while performing dangerous activities is rare and seldom results in injury to others. Risk depends somewhat on specific cardiac arrhythmias as noted below.

Ventricular Arrhythmias

The risk that patients with life-threatening ventricular arrhythmias might pose if allowed to drive must be addressed. The principal factors that determine the magnitude of this risk are the likelihood that patients, once treated, will experience a recurrence of their arrhythmia, the likelihood that such a recurrence will impair consciousness sufficiently to interfere with their ability to operate a motor vehicle, the probability that such an event will result in an accident, and the probability that such an accident will result in death or injury to other road users or innocent bystanders.

In patients with a history of sustained monomorphic VT or VF who are treated with an implantable cardioverter-defibrillator (ICD), the likelihood of experiencing a recurrent episode of arrhythmia can be estimated by examining the frequency and time course of appropriate shocks delivered by the device after implantation. "Appropriate" shocks are usually defined as shocks delivered during electrocardiographically documented sustained VT or VF or shocks delivered during an episode of syncope or presyncope that result in restoration of consciousness. When analyzed by actuarial methods, the proportion of patients experiencing an appropriate shock during several years of follow-

up approaches 70%.^{26 27} Most such shocks occur in the first year after implantation, after which hazard rates calculated for consecutive 12-month intervals decline markedly.²⁷ In patients who experience a shock during follow-up, approximately 10% have syncope associated with the shock,²⁸ and an additional 10% have presyncope severe enough to impair or prevent voluntary motor activities (E. Berger, unpublished observation, 1994). Importantly, persons who experience syncope associated with ICD discharge cannot be reliably identified prospectively by any clinical criteria, including etiology of heart disease, severity of ventricular dysfunction, presence or absence of syncope with presenting arrhythmia, or cycle length of VT induced at the time of electrophysiological testing.²⁸ Furthermore, in patients who experience multiple ICD shocks, the absence of syncope in association with the first shock does not predict freedom from syncope during subsequent shocks.²⁸

The probability that patients will experience recurrence of a ventricular arrhythmia severe enough to impair their ability to operate a motor vehicle in the year after initiation of therapy for the arrhythmia has recently been studied in detail.²⁴ Outcome events, which included syncope, sudden death, recurrent VF, recurrent hemodynamically compromising VT, or ICD discharge were analyzed by actuarial methods in a group of 501 patients admitted to a hospital after resuscitation from sustained VT or VF. At the end of 1 year of follow-up, 17% of patients had experienced an outcome event. Analysis of the monthly hazard rates during this first year of follow-up indicated that the highest hazard rate was seen in the first month after discharge from the hospital.²⁴ Hazard rates for months 2 through 7 were moderate, after which they declined substantially.²⁴ Because only 8% of the entire group was treated with an ICD, these results predominantly reflect the results of antiarrhythmic drug therapy.

It has been suggested that the yearly risk of harm to other road users or innocent bystanders caused by a patient who develops a hemodynamically compromising ventricular arrhythmia while driving can be expressed mathematically.⁷ The equation for risk of harm (RH) is $RH = (TD)(V)(SCI)(Ac)$, where TD equals the time the patient spends driving during the year; V, a constant based on the type of vehicle driven, eg, 1.0 for a commercial heavy truck and 0.28 for a standard-size passenger car; SCI, the risk of sudden death or incapacity during the year; and Ac, the probability that sudden death or incapacity while driving will result in death or injury to other road users or innocent bystanders. It has been estimated that most noncommercial drivers spend only 4% of their time driving a car.⁷ If it is assumed that approximately 50% of patients with an ICD will experience a discharge of the device in the first year after implant and that 20% of patients who experience an ICD discharge will have either syncope or presyncope with the discharge, then SCI in the equation equals 0.10. It is difficult to estimate a value for Ac in this equation, but most of the available data suggest that it is quite small. Studies of patients without an ICD who die suddenly while driving suggest that fewer than 2% of such incidents result in death or injury to other road users or innocent bystanders.^{17 23 29 30} This is supported by more recent data from a study of patients with ICDs that stated approximately 10% of ICD discharges that occur while patients are driving result in an accident and that injury or death of other road users or innocent bystanders as a consequence of such accidents is rare.³¹ Substituting these values (TD=0.04, V=0.28, SCI=0.10, Ac=0.02) into the equation above yields an annual risk of harm to other road users or innocent bystanders of 0.0000224 or 1 per 45 000. The risk of harm caused by patients with ventricular arrhythmias who are taking antiarrhythmic drugs and are allowed to drive is probably equivalent or only slightly higher.²⁴

One can identify groups of patients with heart disease who have never had an episode of VT or VF in whom the risk of sudden death or incapacity approaches or even exceeds the risk in patients

with ventricular arrhythmia being treated with an ICD or an antiarrhythmic drug.³² Ambulatory patients with Class III or IV heart failure awaiting cardiac transplantation or patients with a recent myocardial infarction, severe left ventricular dysfunction, and reduced heart rate variability are two such examples.

Atrioventricular Node Reentry and Wolff-Parkinson-White–Related Tachycardias

A regular, relatively rapid, narrow QRS complex tachycardia is the most frequent presenting arrhythmia in patients who have AV node reentry, Wolff-Parkinson-White (WPW) syndrome, or concealed accessory connections.³³ Both AV node and AV reentry can degenerate into atrial fibrillation,^{34 35 36} which in patients with WPW rarely leads to an extremely rapid preexcited ventricular response causing VF and subsequent sudden cardiac death.³⁷ Both pharmacological and nonpharmacological therapy can be used to treat patients with AV and AV node reentry. The widespread availability and success of RF catheter ablation to cure these arrhythmias has made it a first-tier treatment option.

Atrioventricular Node Reentry Patients with AV node reentry compared with AV reentry statistically are older and more commonly women (R. Chamberlain-Webber, et al, unpublished data, 1995). However, in at least one study there was no difference in tachycardia rate (179 bpm) (R. Chamberlain-Webber, et al, unpublished data, 1995). Symptoms during spontaneous episodes of AV node reentry are quite variable, ranging from palpitations to syncope. The incidence of syncope is difficult to ascertain for at least two reasons: patient symptoms are infrequently detailed in published studies,^{38 39 40 41 42 43 44 45 46} and referral bias that may give an overestimation of syncope is likely. Nevertheless, syncope was reported to occur in 33% to 39% of patients with AV node reentry.^{38 39 41} Atrioventricular node reentry was reportedly the cause of cardiac arrest in two patients.^{38 39} In this regard, degeneration of AV node reentry to polymorphic VT has been reported in a patient with no evidence of structural heart disease.⁴⁷ However, this is a rare event.^{48 49}

The technique of RF catheter ablation to cure AV node reentry has undergone substantial modification over the past few years,^{39 40 41 42 43 44 45 46} and several publications have described more than one technique. Thus, a summary of the data may not accurately reflect current results. The eight publications include 454 patients.^{39 40 41 42 43 44 45 46} Successful ablation of AV node reentry ranged from 79% to 100%. Clinical recurrence of arrhythmia or inducibility at follow-up electrophysiological study was noted in 0% to 18% of patients, but only three studies reported a recurrence rate greater than 10%.^{39 43 45} Most experienced laboratories now achieve a success rate greater than 90% using the posterior (or slow pathway) approach.

In summary, patients with syncope due to AV node reentry require therapy. Effective RF ablation or antiarrhythmic drug therapy can prevent recurrence of syncope. Prediction of spontaneous recurrences after initiation of empiric antiarrhythmic therapy may be difficult due to spontaneous variability in arrhythmia frequency.⁵⁰

Wolff-Parkinson-White Syndrome Patients with accessory pathways are susceptible to a variety of arrhythmias.³³ In some cases the pathway forms a critical part of the tachycardia circuit, but in other instances the accessory pathway functions as a transporter of electrical impulses from the atria to ventricles. This latter function can be pernicious and on rare occasions results in sudden death in patients with atrial fibrillation.³⁷ Regardless of the mechanism of tachycardia, loss of conduction over

the accessory pathway ameliorates the situation. As with AV node reentry, there are few data concerning the incidence of syncope.^{51 52 53 54 55} These groups^{52 53 54} reported that syncope occurred in 11% to 29% of patients, and 1% to 8% had a history of cardiac arrest.

Results of RF catheter ablation of accessory pathways have been reported for 693 patients.^{51 52 53 54 55} Successful elimination of accessory pathway conduction occurred in 86% to 99% of patients. Recurrence rates were quite variable among investigators, ranging from 4% to 17%. Successful ablation of the accessory pathway removes the risk of syncope or cardiac arrest. If patients are treated with antiarrhythmic agents in lieu of RF ablation, their risk will depend on the ability of the drug to affect conduction over the accessory pathway, which should be evaluated.

The approach to asymptomatic patients with ventricular preexcitation is a conundrum. No therapy will make them feel better, but some patients may be at risk for sudden cardiac death. However, the incidence of sudden death in these individuals is exceedingly low, probably 1 per 1000 years of patient follow-up or less.⁵⁶ Furthermore, the prevalence of a rapid preexcited ventricular response during atrial fibrillation, the marker of potential risk for VF, is too high in asymptomatic patients to provide any meaningful data regarding specificity.⁵⁶ Thus, as a general rule, mass screening to identify patients at potential risk for sudden death is not recommended. However, risk stratification may be appropriate in select subgroups such as airline pilots, commercial drivers, and competitive athletes.

Atrial Tachycardia in Young Patients

Atrial tachyarrhythmias in young patients are more common than ventricular tachyarrhythmias. While the exact mechanism for reentrant arrhythmias confined to the atria is not always clear, it is likely that atrial flutter is the most frequent. This arrhythmia occurs most often in young patients with congenital heart disease. In a study of 380 young patients with atrial flutter, the most common diagnoses were postoperative Mustard or Senning operation for transposition (21%), complex congenital heart disease (18%), and postoperative atrial septal defect (12%). Overall, 60% had postoperative congenital heart disease. To examine the problem with respect to prevalence within each type of congenital heart disease, with average follow-up extended 12 years, approximately 25% of patients have atrial flutter after the Mustard or Senning operation, compared with approximately 10% of patients after repair of atrial septal defect during longer term follow-up.⁵⁷ After the Fontan operation, with an average follow-up of 7 years, approximately half of patients with atrial arrhythmias have atrial flutter, 25% have an atrial reentrant tachycardia that does not have the morphology of atrial flutter, and 25% have an automatic tachycardia.⁵⁸ In a given patient with Fontan the type of atrial arrhythmia may change over time from one mechanism to another. It is interesting that atrial fibrillation is rare in persons younger than 25 years. However, it does seem that as younger patients pass the age of 30, those who had atrial flutter in their twenties begin to develop atrial flutter-fibrillation and eventually atrial fibrillation.

It is not commonly known that more young patients die from atrial tachycardia than VT.⁵⁹ The reasons for this are twofold. First, the AV node of the young patient conducts extremely well. Newborn babies can conduct at atrial rates of 500 bpm, and young adults can conduct readily at 300 bpm. Rapid conduction may occur paroxysmally and is usually associated with an intense increase in catecholamines, such as those found during sports or driving. For this reason, the young patient with a chronic atrial tachycardia such as atrial flutter with a “controlled” ventricular response is at risk for 1:1 conduction. Among 380 young patients with atrial flutter followed for an average of 6 years, the

risk of sudden death was 20% in those with continued episodes of flutter despite a controlled ventricular response, compared with 5% among those whose episodes were thought to be controlled.⁶⁰ The second reason that atrial tachycardias are lethal is that they are found most often in young patients with an abnormal heart; among 380 young patients, 93% had an abnormal heart. As in older patients with ventricular tachyarrhythmias, severely abnormal hemodynamics are a risk factor for sudden death.

In summary, it seems reasonable to conclude that at times activities such as driving may involve catecholamine surges similar to those that occur during competitive sports. In young patients, because there is a real danger of 1:1 conduction, attempts should be made to eliminate episodes of atrial flutter as well as other atrial tachyarrhythmias.

Bradyarrhythmias

Bradyarrhythmias may cause transient cerebral hypoperfusion with consequent complete or partial loss of consciousness and/or postural tone (ie, syncope or presyncope). The effectiveness with which symptoms can be prevented by treatment is predicated on both establishing an etiologic diagnosis with a high degree of certainty and the availability of satisfactory therapeutic options. Among treated patients whose syncopal symptoms are due to bradyarrhythmias secondary to structural conduction system disease, cardiac pacing is highly effective in preventing recurrences. Similarly, among individuals in whom syncope is closely associated with neurally mediated bradyarrhythmic or vasodepressor syndromes (eg, carotid sinus syndrome, vasovagal syncope), the real risk of syncope while driving seems to be small (K. Lurie, et al, unpublished data, 1995).

The bradyarrhythmias of interest in this section may be divided into two general classes, although it is recognized that elements of the two are often intermingled: (1) arrhythmias caused by disease of the sinoatrial node and/or cardiac conduction system, and (2) arrhythmias associated with neural-cardiovascular interactions. The former includes intrinsic sinoatrial dysfunction and acquired disease of the specialized cardiac conduction system. The latter includes a broad range of neurally mediated bradycardia-vasodepressor syndromes, the most frequent of which are the vasovagal faint, carotid sinus syndrome, and postmicturition syncope.

Establishing the etiologic basis for syncopal and presyncopal episodes begins with a detailed medical history. Syncope associated with abrupt severe bradycardia due to sinus node dysfunction (eg, sinus arrest) or conduction system disease (eg, high-grade AV block) may be unassociated with premonitory symptoms, especially in elderly patients. Furthermore, in such cases loss of consciousness may be independent of posture, although upright posture (standing, occasionally sitting) is usually implicated. Similarly, in certain forms of neurally mediated syncope (eg, carotid sinus syndrome), onset may be abrupt and also without warning. In the latter case a history of symptoms associated with head movement may be helpful in establishing the diagnosis, although there is still considerable variability. On the other hand, in patients with other forms of neurally mediated syncope (eg, vasovagal syncope or cough syncope), suggestive warning symptoms and signs (eg, preceding sense of apprehension and nausea, pallor, and diaphoresis) and the situation in which the faint occurred are often helpful diagnostically. However, once again there are no reported warning symptoms in many cases. The latter seems particularly true in the older patient.⁶¹ Consequently, while the medical history of a syncopal event is important, it cannot be relied on routinely to identify the etiology of syncopal spells. Careful diagnostic testing is usually warranted regarding both the basis of symptoms and the likely effectiveness of the selected treatment. In this context the most

important first step after obtaining a thorough medical history (including that obtained from bystanders), is to determine whether structural heart disease is present. If so, electrocardiographic studies (eg, cardiac monitoring, signal-averaged electrocardiography) and conventional electrophysiological testing are generally the next step. In the absence of structural heart disease, autonomic function testing (usually incorporating tilt-table testing) is indicated.

Bradyarrhythmias Caused by Disease of the Sinoatrial Node or Cardiac Conduction System Cardiac arrhythmias associated with intrinsic sinus node dysfunction and/or disease of the specialized cardiac conduction system are among the most frequent causes of syncope. In such cases, however, the true basis of syncope may be multifactorial, including not only the severity of the bradyarrhythmia but also postural factors, status of left ventricular function, magnitude of concomitant cerebrovascular disease, and adequacy of vascular compensation to maintain systemic pressure (ie, an example of the impact of neural reflex effects).

Clinical manifestations of sinoatrial disease vary widely. Syncope is a relatively common reason for such patients to be referred to cardiologists, yet such severe symptoms are probably an uncommon manifestation of the disease, given the high frequency with which it occurs in the population. Nonetheless, syncope or dizziness in this setting may be manifestations of either bradyarrhythmias or tachyarrhythmias. Establishing the arrhythmia at fault is essential for devising an effective treatment strategy.

Acquired disease of the cardiac conduction system is also quite common. As a result, electrocardiographic evidence of conduction system disease is a relatively nonspecific finding with respect to syncopal or presyncopal symptoms. Therefore, in the absence of well-documented second- or third-degree AV block, the role of conduction system disease in the patient with syncope requires careful assessment, often including electrophysiological study. Importantly, the pathology of the conduction system in patients exhibiting long-term conduction system disease tends toward widespread fibrosis and exists with regional or generalized myocardial disease. Thus, these patients share the potential for other arrhythmias, particularly ventricular tachyarrhythmias, and not infrequently it is tachyarrhythmias rather than bradycardia that account for symptoms.^{62 63}

As a rule, for patients with structural heart disease associated with symptomatic bradyarrhythmias, physiological cardiac pacing therapy (ie, implantation of a pulse generator system that eliminates both risk of bradycardia and reasonably mimics normal AV sequence and chronotropic response) essentially eliminates subsequent risk of syncope. The probability of abrupt pacemaker system failure is remote, and in patients with recurrent symptoms it is unlikely to be due to pacemaker system malfunction.

Neurally Mediated Syncopal Syndromes Neurally mediated syncopal syndromes are the most common cause of syncope.^{61 64} The afferent signals that initiate these events may originate from the central nervous system directly (as in syncope associated with fear or anxiety) or from any of a variety of peripheral receptors that respond to various stimuli (eg, carotid sinus syncope or postmicturition syncope). Subsequent electrophysiological and hemodynamic events are variable. Certain patients exhibit a predominantly cardioinhibitory state (ie, cardiac slowing primarily parasympathetically mediated via the vagus nerve) with an extended period of bradycardia (marked sinus bradycardia, sinus arrest, paroxysmal AV block, or various combinations) being the principal cause of the faint. Most patients, however, present a mixed vasodepressor and cardioinhibitory

response. The mechanism of the vasodepression is believed to be predominantly the result of abrupt peripheral sympathetic neural withdrawal. Consequently, effective treatment cannot usually be restricted to elimination of susceptibility to bradyarrhythmias alone.

In recent years tilt-table testing has become an accepted tool for establishing susceptibility to neurally mediated syncope and thereby is a useful element in diagnostic evaluation of patients with syncope. In the absence of a true diagnostic gold standard, the sensitivity of head-up tilt testing can only be estimated to range from 60% to 80%. Specificity ranges from 80% to 90%.^{65 66} The results of tilt testing appear to be reasonably reproducible within a 6-week time period,⁶⁷ but reproducibility of findings over a longer time period, ie, months to years, has not been reported. Therapeutic options for vasovagal syncope include β -adrenergic blockers, anticholinergic agents, theophylline, disopyramide, volume expansion (NaCl, fludrocortisone acetate), antidepressants (for their autonomic effects), and in some cases permanent pacing. Although tilt testing is used with increasing frequency in the diagnosis of vasovagal syncope, its value in guiding therapy remains controversial. Combined therapies are often necessary, and a trial-and-error strategy may be required to find the most effective treatment regimen. Recurrences may be unpredictable, and spontaneous variability as well as spontaneous remissions may occur.

To evaluate current expert thought regarding driving recommendations in patients with suspected or diagnosed neurally mediated syncope, 66 cardiac electrophysiology centers in 22 US states and nine countries were surveyed (K. Lurie, et al, unpublished data, 1995). Respondents reported treating more than 11 500 patients with syncope. Eighty-nine percent of respondents indicated that none of their treated patients were involved in a subsequent motor vehicle accident. Furthermore, of the 11% of respondents who reported at least one treated patient involved in an accident, no respondent had more than two such events. Based on the number of patients treated by respondents, the incidence of subsequent motor vehicle accidents was very low (0.1% to 0.2%).

RECOMMENDATIONS

Using the principles discussed above, the available data, and the medical judgment of the authors, the following recommendations were formulated. These represent guidelines and not practice standards. They are not applicable to every patient in every situation, and physicians are encouraged to use their own judgment in making a recommendation for any given patient. It must be emphasized that many of the recommendations are based on limited data; thus, these recommendations may change as more information becomes available.

Although an ICD requires special consideration with respect to allowing a patient to participate in potentially hazardous endeavors (see below), the recommendations for ventricular tachyarrhythmias do not specifically target patients with ICDs. Rather, they target the risk of arrhythmia recurrence and, in the event of recurrence, risk of syncope or loss of control. In addition, if an arrhythmia is thought to be caused or exacerbated by a reversible cause (such as acute ischemia, electrolyte imbalance, or drug toxicity), reversal of the precipitating factors is important. Once these factors have been corrected, the patient's risk of recurrent arrhythmia in the absence of these precipitating factors and/or the risk of having these precipitating factors recur should be considered.

Once a therapy for prevention of arrhythmia recurrence has been undertaken, there are two general methods for demonstrating its efficacy. First, a test result (possible comparison with a pretherapy test result) may be used to evaluate efficacy, for example, prolonged electrocardiographic monitoring or electrophysiological testing before and after drug therapy for VT, programmed

stimulation for induction of an arrhythmia after attempted ablation of the arrhythmia, or testing the antitachycardia pacing efficacy of an ICD for sustained monomorphic VT. The second method of documenting adequate arrhythmia suppression is to follow the patient for a defined period of time after the therapeutic intervention without recurrence of arrhythmia. Throughout the recommendations one or both of these general criteria to confirm arrhythmia suppression may be used.

Criteria for adequate suppression of arrhythmias are controversial, differing greatly between arrhythmias and patients and depending heavily on the physician's judgment. Each physician must decide for each patient whether the test selected to evaluate arrhythmia suppression is reliable in that particular situation. For example, an electrophysiology study 6 weeks after ablation of an accessory pathway documenting no accessory pathway conduction would be highly reliable in predicting future freedom from arrhythmias. On the other hand, the value of a negative tilt-table test while the patient is receiving β blockers at predicting future freedom from syncope is more controversial; in this situation a time period for observation should be used to judge whether the patient should return to a potentially hazardous activity.

Recommendations are grouped into the following categories: Class A, Class B, and Class C. Patients in the Class A category should have no restrictions. Patients in the Class B category are restricted for a defined time without arrhythmia recurrence, usually after a therapeutic intervention. This time period (in months) is expressed as a subscript (eg, B₃). Patients in the Class C category should have total restriction of potentially hazardous activities.

There was extensive discussion at the consensus conference about the level of restriction for patients performing only personal driving (noncommercial) versus either commercial driving or flying privileges. A wide variety of potential hazards exist within each of these two categories. For example, the patient who drives a few blocks to church on Sunday and to the grocery store twice weekly may present less risk to himself and others than the private driver who travels 12 miles on an interstate highway to go to work every day. Likewise, the commercial driver who drives a larger vehicle many hours on the highway daily may present a greater hazard than the commercial driver who drives a floral delivery truck short distances intermittently throughout the day. Loss of consciousness in patients who drive multiple-occupancy vehicles such as school buses may result in particularly dangerous outcomes. The hazard of flying to both pilot and others varies, depending on whether the craft is a one-pilot private plane, a commercial airliner with two or more pilots, low-performance military aircraft (such as bombers or transport aircraft), or high-performance military fighter jets. Despite these differences, which must be considered in any individual case, the conference consensus was that restrictions should be divided into only two categories: personal or noncommercial driving and commercial driving/flying. *In general, unless stated otherwise, recommendations for flying are the same as those for commercial drivers.* The Canadian Cardiovascular Society Consensus Conference⁷ defined criteria to distinguish a private driver from a commercial driver on the basis of number of kilometers driven per year, hours per year behind the wheel, weight of the vehicle, and whether or not the vehicle is used to earn a living. (Specifically, a private driver was defined as one who drives less than 36 000 kilometers per year or spends less than 720 hours behind the wheel per year, drives a vehicle weighing less than 11 000 kg, and does not earn a living by driving. A commercial driver was defined as any licensed driver who does not fulfill the definition of a private driver.) The conference participants encourage physicians to use their personal judgment in deciding whether a particular patient's situation warrants inclusion in the private driving or commercial driving category.

The commercial recommendations may also be applied to potentially hazardous occupations other than driving or flying in which both patient and others may be endangered should loss of consciousness occur (for example, operation of heavy equipment in a factory). The definition of which occupations/activities are covered by these guidelines involves a judgment by employer, patient, and physician. Activities that place only the patient at risk (for example, competitive athletic events) are beyond the scope of this statement.

Special Considerations for Patients With Implantable Cardioverter-Defibrillators

OverviewThe ICD is effective therapy for terminating ventricular arrhythmias and preventing sudden cardiac death.^{68 69 70 71} However, despite its effectiveness, identifying the appropriate time (if any) for the patient to return to driving may be difficult. First, patients with ICDs may occasionally experience severe symptoms or even hemodynamic collapse before termination of the arrhythmia.^{28 72} Symptoms before delivery of ICD therapy are related to hemodynamic tolerance of the arrhythmias, which is related to rate, duration of arrhythmia before delivery of therapy, and status of the underlying myocardial function.^{73 74 75} Furthermore, prediction of the timing and frequency of ventricular arrhythmia recurrence and the rate and the time of recurrence may be difficult.^{70 71 76 77} ⁷⁸ Patients may also experience an arrhythmia that actually results from ICD therapy or arrhythmia instability and acceleration with hemodynamic collapse.^{79 80 81} Finally, ICD shock therapy may cause discomfort and frighten or startle the patient, which could lead to loss of control of a vehicle when driving, even in the absence of symptoms due to the arrhythmia.

Symptoms and Arrhythmias Before TherapyMost prior reports on the frequency of VT/VF recurrence in patients with ICDs are based primarily on the development of symptoms before shock therapy.^{68 69 76 77 78} However, it has been well documented that symptoms in general are not a reliable indicator of the type of arrhythmia leading to ICD therapy, although severe symptoms are usually associated with sustained ventricular arrhythmias.^{72 82} The incidence of frank syncope preceding ICD therapy is indeed small. Kou and colleagues²⁸ documented a cumulative incidence of less than 10%. Of note, most patients who experience sustained ventricular arrhythmias before ICD shock experience few or no symptoms.^{72 82} The availability of advanced diagnostic information in the form of RR intervals and stored electrograms will undoubtedly improve the ability to identify the frequency of sustained ventricular arrhythmia recurrence in patients with ICDs.^{83 84 85 86} Information derived from stored electrogram analysis suggests that in the absence of severe symptoms, approximately 25% of arrhythmia episodes leading to device therapy are due to supraventricular tachycardia.⁸⁴ Furthermore, approximately 40% of patients with an ICD will experience an ICD discharge in the absence of severe symptoms for a rhythm of nonventricular origin.^{84 85}

Predictors of Arrhythmia Recurrence After ImplantationThe actuarial incidence of ICD shocks varies somewhat from study to study and may be influenced by the study population receiving the ICD.^{70 71 87 88 89} However, the 5-year actuarial incidence of ICD shocks that are deemed appropriate (ie, documented electrocardiographically to be VT or preceded by severe symptoms) typically ranges between 60% and 70%.^{70 71} Factors documented to be independently associated with the time to an ICD shock after implantation include left ventricular ejection fraction, inducible rapid polymorphic VT or fibrillation at the time of electrophysiological testing, amiodarone therapy, and cardiac arrest.^{70 71} Patients with depressed left ventricular function appear to have an actuarial incidence of the first ICD

discharge that is at least two to three times greater than that of patients with normal LV function.^{70 71}
⁷⁸ Other factors that may be associated with the frequency of and time to ICD shock include the presence of coronary artery disease, primary electrical disease, previous coronary artery bypass graft surgery, and β -blocker therapy. Factors not associated with time to ICD shock include patient age and gender.

Clearly the absence of ICD therapy during the first year after implantation has been associated with a lower risk of arrhythmia recurrence and subsequent ICD discharge. This risk ranges from 0% in a report by Anderson and Camm⁸⁹ to 10% to 20% annual actuarial incidence of first ICD shock therapy if no preceding ICD therapy has been received.^{27 89 90 91} Just as importantly, those patients who experience an ICD shock are at an increased risk for requiring further ICD therapies during subsequent years. This actuarial incidence of further ICD therapy has been estimated to be at least 33%.

Tiered-Therapy Implantable Cardioverter-Defibrillator Devices: Implications for DrivingThe use of tiered-therapy devices for managing life-threatening ventricular arrhythmias has extended the use of ICD treatment to a lower range of VT rates and has eliminated shocks in the absence of symptoms for many patients with ventricular arrhythmias.^{80 81} The effectiveness of antitachycardia pacing has been well documented for patients with sustained VT and may also be of benefit even for selected patients with a cardiac arrest and inducible sustained VT.²⁷ One downside related to the use of tiered-therapy devices is increased use of the ICD in the patient population with few or no symptoms related to VT. This therapy can also now be used in patients with more frequent VT episodes, increasing the chance that the patient will experience an episode that is accelerated and associated with hemodynamic embarrassment. A potentially longer duration of the VT episode required to deliver effective tiered therapy and possible acceleration of the arrhythmia with antitachycardia pacing may occur in patients with tiered-therapy devices. In addition, VT with slower rates that overlap with supraventricular tachycardia can potentially initiate VT that may be poorly tolerated hemodynamically. Thus, although tiered therapy does prevent shocks in many patients who receive an ICD for VT, it may actually increase the hazards that may occur during driving in selected patients with ICD devices.⁷⁹

Use of Adjunctive Antiarrhythmia Drug or Ablative TherapyThe use of adjunctive therapy such as antiarrhythmic drugs or ablative therapy may be advantageous in decreasing the frequency of arrhythmia recurrences and symptomatic ICD shocks. Antiarrhythmic drugs may prevent episodes of VT, slow the tachycardia rate, and make it more likely to be hemodynamically tolerated and amenable to termination with pacing. Selected drugs may reduce the energy requirements for defibrillation and prevent supraventricular tachycardia that can trigger inappropriate ICD therapy.^{92 93} The use of antiarrhythmic drugs, however, must be approached with caution. Proarrhythmic effects may result in an increased frequency of arrhythmias that may be less amenable to ICD therapy and may increase energy requirements for defibrillation.^{94 95 96} Slowing of VT rates may result in an increased overlap with rates of supraventricular rhythms achieved with stress or activity.

Catheter ablation may provide arrhythmia control without risk of proarrhythmia. Preliminary reports in highly selected patients have suggested a potential cure rate as indexed by noninducibility of VT and short-term arrhythmia control in up to 75% of patients with sustained VT who undergo catheter ablation.^{97 98 99} Thus, catheter ablative therapy may have promise in selected patients with coronary artery disease who have frequent or incessant VT episodes before or after planned ICD therapy.

Summary For patients who drive after implantation of an ICD, drug or ablative therapy may be instituted to prevent frequent recurrences. Episodes of supraventricular tachycardia should be managed aggressively by appropriate programming and drug or ablative therapy. Reliability of ICD therapy programming and/or effectiveness of preventive or curative therapy should be assessed not only in the electrophysiology laboratory before hospital discharge but during a defined period of clinical follow-up (Table 1). This period of follow-up will take into account factors such as clinical arrhythmia presentation and frequency, left ventricular function, use of adjunctive drug and/or ablative therapy, and observed response at the time of electrophysiological testing. Larsen et al²⁴ suggest that patients without arrhythmia events for a duration of 6 months fall into a sufficiently low-risk group to permit resumption of driving.

Ventricular Tachycardia (Table 1)

There is no reason a priori to make different recommendations for patients with VT or VF whose primary treatment is antiarrhythmic drug therapy rather than ICD therapy. Patients with sustained VT or VF who are treated with antiarrhythmic drugs should be prohibited from noncommercial driving for the first 6 months after initiation of therapy to document an arrhythmia-free interval before they are allowed to resume driving. If a second episode of ventricular arrhythmia occurs after resuming driving, then another 6-month period of abstinence from driving should be recommended to provide sufficient time to judge whether the changes in medical therapy have adequately suppressed arrhythmia recurrences. As with ICD-treated patients, it is recommended that all commercial driving be permanently prohibited in patients with VT or VF who are treated primarily with antiarrhythmic drugs.

It is probably reasonable to make exceptions to the above recommendations for patients who have idiopathic VT and who do not have symptoms of impaired consciousness with their presenting arrhythmia. Such patients should be shown by extensive evaluation to have normal ventricular function, normal coronary arteries, no evidence of hypertrophic cardiomyopathy, and no evidence of right ventricular dysplasia. Because such patients do not have obstructive coronary artery disease, they are unlikely to develop enough myocardial ischemia during their arrhythmia to render the tachycardia electrically unstable; degeneration of VT to VF in such patients is rare. Accordingly, it is likely that if the patient has not had symptoms of impaired consciousness with the presenting arrhythmia, he or she will tolerate future episodes equally well. Thus, in selected individuals, a shorter period of driving restriction may be appropriate once medical therapy has been initiated (Table 1).

Special mention should be made of patients who have the long QT syndrome, which is classified as acquired or congenital. The acquired forms are due either wholly or in part to reversible factors, such as drugs that prolong the QT interval or electrolyte abnormalities such as hypokalemia and hypomagnesemia. Most patients can be allowed to drive after correction of these reversible factors. The inherited disorders are associated with ventricular tachyarrhythmias, particularly torsade de pointes VT, that can produce syncope or death. Arrhythmias and syncope occur most often during physical exertion or emotional stress. Treatment effectively prevents symptoms in the vast majority of patients, and symptoms decrease in frequency over time, particularly during the second to fourth decades. They are uncommon after the fourth decade. Drugs that prolong the QT interval should be avoided in these patients. Patients who have symptomatic long QT syndrome should not have driving privileges, but patients with long QT syndrome who are asymptomatic or who have a history of symptoms but are asymptomatic on treatment should receive driving privileges after a 6-month symptom-free interval.

Among patients treated with an ICD because of a previously documented episode of VT or VF, approximately 10% will experience syncope or presyncope in association with ICD discharge in the first year after implantation. No clinical variable or combination of variables can predict which patients will experience syncope or presyncope in association with ICD discharge. The period of greatest risk appears to be the first 6 months after implantation. Beyond this period risk falls to a much lower level, remaining relatively constant up to 4 years after implantation. However, there is no period beyond which the risk of syncope or presyncope is zero. For noncommercial drivers who spend only a small fraction of their time driving and who usually drive relatively light-weight vehicles, the risk of causing harm to other road users because of syncope or presyncope that might occur in association with an ICD discharge is extremely small. This risk, in fact, is of the same order of magnitude as the risk for certain other groups of cardiac patients with no history of ventricular arrhythmia who presently are not prohibited from driving (for example, the post-myocardial infarction population).²⁴

In view of these considerations, noncommercial drivers should be prohibited from all driving for the first 6 months after ICD implantation. After 6 months, if ICD discharge has not occurred, patients may resume driving. However, when highway (high-speed) or long-distance travel is anticipated, patients should be encouraged to have an adult companion drive; cruise-control driving should be avoided. If ICD discharge occurs after implantation, either with or without associated syncope or presyncope, patients should be advised not to drive for the next 6 months. This period of time should be adequate to determine the nature and tempo of the rhythm responsible for triggering the ICD and to take appropriate corrective action. For commercial drivers, eg, truck drivers, bus drivers, or delivery people who spend a much greater proportion of their time driving or who tend to drive much heavier vehicles, the risk of causing harm to other road users as a consequence of syncope or presyncope in association with ICD discharge is substantially increased. It is recommended that all commercial driving be prohibited permanently after ICD implantation.

There may be an unusual circumstance in which a patient with an ICD has multiple episodes of asymptomatic VT on follow-up that are repeatedly terminated by antitachycardia pacing with no episodes of acceleration¹⁰⁰; in this situation the rules prohibiting driving may be adjusted on a very selective individual basis.

On rare occasions an ICD may be implanted in a patient who has never had a sustained ventricular tachyarrhythmia. Examples include patients with hypertrophic cardiomyopathy or long QT syndrome and a strong family history of sudden death or patients with nonsustained VT and left ventricular dysfunction enrolled in research protocols that may include ICD implantation. If these patients have had no symptoms of hemodynamic compromise, they should not be prohibited from private driving, although commercial driving should still be restricted.

Regardless of the consensus recommendations presented in this document, liability and other considerations may take precedence. For example, school boards will not likely assume responsibility for an individual with an ICD to drive a school bus. Trucking companies may be unwilling to assume any risk for a driver when others without potentially dangerous medical problems can be hired. Finally, the FAA and the military already have guidelines governing pilots that will take precedence over recommendations of a lay body.

Supraventricular Tachycardia (Table 2)

The supraventricular tachycardias comprise the atrial tachycardias, including focal atrial tachycardias, atrial fibrillation, atrial flutter, AV reentry, and AV node reentry. Although the general prognosis in

patients with Wolff-Parkinson-White syndrome and other supraventricular tachycardia is generally quite favorable, at least a single episode of syncope is reported in approximately 25% of patients referred to electrophysiology laboratories for assessment.^{100 101 102 103} Sudden death is rare as the first manifestation of Wolff-Parkinson-White syndrome and is seen only sporadically in asymptomatic individuals with ventricular preexcitation.¹⁰⁴ Atrial tachyarrhythmias in young patients following repair of congenital heart disease can be associated with severe symptoms.^{57 58 59 60}

Atrial fibrillation is the most common supraventricular arrhythmia.¹⁰⁵ Syncope related to atrial fibrillation is readily recognized in most instances as resulting from intermittent bradycardia or, conversely, a rapid ventricular rate. There are no data documenting the frequency with which syncope related to supraventricular tachycardia causes motor vehicle accidents, but it is probably rare. A wide range of catheter ablative, operative, and medical therapies offer definitive cure or excellent control of symptoms with supraventricular tachycardias (Table 2). Patients with supraventricular tachycardias that appear to be successfully ablated may drive after recovery from the procedure, because the risk of arrhythmia recurrence and risk of injury from recurrence are low.

Bradycardia With and Without Cardiac Pacing (Table 3)

The safety of a patient driving with the diagnosis of a bradyarrhythmia is dependent on the presence of syncope before placement of the pacemaker, the reliability of the pacing system, and the underlying cardiac pathology.^{106 107 108} Bradyarrhythmias can be divided into those with and those without syncope or presyncope. Patients with symptoms require cardiac pacing to drive. Those without symptoms do not need a pacemaker and can drive as long as they are symptom free.^{7 109 110 111}

Patients who have cardiac pacemakers are unlikely to have further symptomatic bradycardia, and if symptoms recur they are unlikely to be due to pacemaker malfunction. The risk of further driving is dependent on the underlying cardiac pathology and is more likely to be related to tachyarrhythmias than bradyarrhythmias.¹¹² Pacemakers implanted for neurally mediated syncope may fail to prevent recurrent syncope if there is a prominent vasodepressor component. Frequently pacemakers are implanted to increase cardiac output above resting levels but not to prevent syncope. It is inappropriate to restrict the driving of persons with a pacemaker. Indirect support for this position lies in the absence of problems observed. Indeed, when pacemakers were first used, concerns about driving were raised but disappeared when no problem was seen.

For patients who have lost consciousness due to bradyarrhythmias, a period of time should pass to ensure stable lead function before they return to driving; this should be 1 week for noncommercial drivers and 4 weeks for commercial drivers, because commercial drivers drive larger vehicles for a longer time duration, and they may also have greater physical demands (for example, loading and unloading a truck). Pacemaker patients followed according to the published follow-up guidelines accepted by NASPE¹¹³ with pacemakers programmed to an appropriate pacing safety margin provide adequate assurance that the pacemaker will protect the patient from syncope due to bradycardia.

Neurally Mediated Syncope and Presyncope (Table 4)

Vasovagal syncope and carotid sinus syncope can both result in an abrupt loss of consciousness caused by reflexive overactivity of the autonomic nervous system. Although these disorders can produce isolated hypotension or bradycardia, a mixed response is commonly observed. The decision

to allow a patient to return to driving after an episode of neurally mediated syncope or presyncope is complicated by the variable nature of the illness. Many adults have experienced a single syncopal episode without recurrence and have not sought medical attention. In contrast, some patients suffer from a severe form of the syndrome with multiple recurrences of syncope despite aggressive medical management. Common faints are most likely vasovagal in etiology and generally have recognizable precipitating events (prolonged standing, a hot room, pain, fear, the sight of blood) and warning symptoms (flushing, nausea, diaphoresis, lightheadedness). Consequently the history of the syncopal event often provides strong diagnostic clues. However, the head-up tilt test is being used with increasing frequency to support the diagnosis of vasovagal syncope.

The natural history of carotid sinus syncope suggests a low incidence of recurrent syncope in treated patients.¹¹⁴ A recent prospective randomized trial in patients with carotid sinus syncope supports an important role for permanent pacing in these patients.¹¹⁵ During a 3-year follow-up, syncope recurred in 57% of patients without pacemakers (16 of 28) and in 9% of patients with pacemakers (3 of 32).

The decision to allow a patient to resume driving should be based on the severity and nature of the presenting event. Mild vasovagal syncope or carotid sinus syncope is characterized by mild symptoms (usually without syncope), occurs with warning and usually only with standing, has clear precipitating causes, and is infrequent. By distinction, severe vasovagal syncope or carotid sinus syncope is characterized by severe symptoms (usually syncope), occurs without warning and in any position, has no clear precipitating causes, and/or occurs frequently.

Arrhythmias Associated With Congenital Heart Disease

Patients with congenital heart disease and associated arrhythmias generally fall into one or more of the following four categories: (1) those with bradycardia secondary to sinoatrial node disease, primarily postoperative; (2) those with second-degree (type II) or third-degree AV block related to congenital heart disease; (3) those having supraventricular tachyarrhythmias; and (4) those having ventricular tachyarrhythmias, generally secondary to having surgery on one or both ventricles.

Symptomatic persons who have bradycardia due to sinoatrial node dysfunction should receive pacemakers according to the ACC/AHA guidelines.¹¹⁶ If normal pacemaker function is documented, these persons may be permitted to drive. Due to the frequent coexistence of paroxysmal tachyarrhythmias with bradycardia, evaluation should exclude atrial flutter or tachycardia with a rapid ventricular response as the potential cause of symptoms. Persons with atrial tachyarrhythmias and a rapid ventricular response rate associated with congenital heart defects require appropriate therapy, whether medical, ablative, or device based. Those who have undergone a Mustard or Senning operation, in particular, should have a 6-month period with no clinical recurrences after therapy before being permitted to drive. At the conclusion of that 6-month period, there should be a 24-hour ambulatory electrocardiogram with no evidence of tachycardia recurrence. In the patient with type II second-degree or third-degree AV block after congenital heart surgery, a well-functioning pacemaker must be in place. Criteria for driving in patients who have a pacemaker would then be in effect.

Patients who have L-transposition of the great arteries and whose natural history includes potential acquisition of complete AV block may drive unless they have syncopal or presyncopal symptoms. In that case there should be adequate evaluation to exclude intermittent or permanent AV block with appropriate therapy before the patient may drive again. For patients with congenital heart lesions associated with ventricular ectopy, the decision to treat may be very controversial. If the decision to

treat is made in the absence of symptoms due to coexistent poor hemodynamic status or other considerations, a successful clinical end point must be present before driving should be permitted; it has been traditional in pediatric electrophysiology to accept end points including negative programmed ventricular stimulation for VT in the absence of drugs, a positive study followed by a negative study on drugs, or medical suppression of ectopy during follow-up noninvasive study. In patients with episodes of sustained or nonsustained VT associated with symptoms of cerebrovascular ischemia, successful therapy must be established by the methods just described, and the patient must be symptom free for 6 months before driving is permitted.

SPECIAL CONSIDERATIONS AND RESPONSIBILITY IN FORMULATING PUBLIC POLICY

The Problem and the Law

In 1992 there were more than 170 million licensed drivers, more than 143 million registered automobiles, 44.5 million trucks, and 600 000 interstate buses operating in the United States.¹¹⁷ The annual fatality rate has plateaued at about 40 000 deaths per year; over 3 million people are injured on America's highways each year, and the total lifetime cost of these tragedies is more than \$48 billion. Consequently the public expects and demands to be protected from unreasonable threats to their safety on the road. In contrast to accidents that occur in aviation, there is an underreporting of accidents that occur as a consequence of medical conditions while driving. Federal and state regulators are responsible for restricting licensing in patients with medical conditions that are clearly related to driving and flying.

People who share the road or any other part of the environment with patients do not in general have the choice of avoiding contact. They must rely on patients being honest and willing to follow sound medical advice, physicians practicing good diagnostics and treatment, employers refusing to allow certain employees to operate potentially dangerous equipment, and manufacturers making reliable products. Any weak link in the chain can lead to injury or death to patients and others as well as economic loss.

Laws regarding the use of private motor vehicles are in general made at the state level. However, there are, as discussed earlier, instances where federal legislation impacts state law, such as the 65 mph speed limit, seat belt and helmet laws, and the restriction on drinking before age 21. In contrast, commercial transportation and interstate commerce are regulated at the federal level. Notably, when large trucks are involved in accidents, occupants of other involved vehicles are more than twice as likely to die as the truck driver.⁷

Licensing agencies have relied on physicians' input to meet their statutory obligation to refuse or revoke licensure. This medical input ranges from individual medical reports to review by medical boards whose decisions are based on established policies and procedures. When rules are drafted, not only data but also physician recommendations, as presented in this document, are taken into consideration.

Patients, Families, and Friends

Families and friends face difficult personal decisions when interacting with loved ones who have heart disease. On the one hand, patients need support when limitations are prescribed by physicians or imposed by the disease process. On the other hand, patients need encouragement to overcome disabilities and physical limitations. Unfortunately denial often complicates the ability of patients to

accept limitations and make sensible and responsible decisions about activities that can be safely pursued. Regardless of the origin of legislation that regulates personal activity, there will be a potential tension between the regulations and the Americans With Disabilities Act. Some of the ethical considerations related to the responsibilities of individuals and regulators are addressed in the section “Ethics of Regulation.”

Role of the Physician

In the context of arrhythmia management, physicians carry the responsibility of providing data and best estimates of risk for the occurrence of first or repeated medical events that may lead to impaired consciousness. Often, prospective data are not available, and clinical observation and best judgments must be used to make recommendations. For example, although most patients with ICDs have been told not to drive (for at least some portion of their postimplantation courses), many have driven, and there is a paucity of reports that document accidents attributable to defibrillator therapy. As emphasized earlier, there is no zero risk in medicine, and all risks must be considered within the context of what society has previously considered to be a tolerable level of risk (eg, teenage driving, threshold limit values discussed above, etc.).

Ultimately physicians have the responsibility to define or estimate the risk of arrhythmia recurrence and loss of consciousness, recognizing that risk assessment will be used to make recommendations about their perception of the advisability of individual patients to perform tasks that may lead to injury if consciousness is impaired. State and federal regulators then have the responsibility to translate these data and recommendations into law or to propose guidelines. Physicians are obligated to become knowledgeable about the specific medical job standards for their patients when asked to render opinions regarding fitness to drive or fly. For fairness to patients, any regulation must be applied with consistency in a standardized, predictable fashion. The tension in regard to confidentiality that exists between the responsibilities physicians have to society and to patients was discussed above.

Responsibilities of Industry

Device manufacturers have the responsibility of designing and producing medical instruments that are reasonably free of defects. This does not mean that a device will never fail. Even if the design could be taken to the extreme without consideration of cost, an absolute guarantee against failure can never be provided. In this effort manufacturers are required to adhere to good manufacturing practices with detailed documentation, testing, and safeguards built into the product and its design and construction. Compliance with good manufacturing practices is monitored by the Food and Drug Administration. All new products must be approved by the agency before commercial release is authorized.

If a person has a recurrence of symptoms, the differential diagnosis must include a failure of the device. It is extremely uncommon for a symptomatic episode to occur while a patient is being monitored. Furthermore, a recurrence of symptoms is not necessarily due to a recurrence of the arrhythmia or a problem with its therapy. In a recent report, documented bradycardia device failures were only rarely heralded by major symptoms such as syncope.¹¹⁸

Although general product liability law is very broad with respect to device defects, prescription items such as medical devices come under the doctrine of an unavoidably unsafe product. Like drugs, devices have known side effects. The prescribing physician is supposed to be aware of

theoretical adverse effects of a therapy, whether drug, ablating device, or implantable antiarrhythmia device, and weigh its potential benefits against its anticipated risks. While prescribing details, implantation technique, and programming are out of manufacturers' control, these and others factors directly affect the function of the implanted system. The law protects the manufacturer of an unavoidably unsafe product from liability for defects in design that can only be identified in retrospect but not for manufacturing flaws or negligence in adhering to approved procedures for its manufacture. Manufacturers are not licensed to provide direct care to patients; as such they have no role in regulating the activities of patients with arrhythmias. Rather their responsibility is to make the products as safe as possible.

Manufacturers also have the responsibility to warn (counsel) the medical community about potential risks associated with implantable devices. This task has traditionally been performed through technical manuals and package inserts that accompany implantable devices and drugs. Furthermore, when a potential systematic problem is identified after a drug or device is commercially released, it is brought to the attention of the medical community through "Dear Doctor" letters, safety alerts, and technical memoranda issued by the manufacturer. When device malfunction is at issue, the warning should include some estimate of the likelihood of problems. The fact that a safety alert or recall has been issued only means that a product is not performing in accord with either the expectations of the manufacturer or the Food and Drug Administration. It is not a guarantee that the device in question will fail. Once the physician has been appraised of a potential problem, he or she then bears the responsibility for its use in subsequent treatment of the specific patient.

Just as the manufacturer has a responsibility to keep the medical community informed, the medical community has a reciprocal responsibility. Manufacturers are not likely to know about a problem unless they are notified when adverse events occur (ie, drug reactions, cardiac perforations with ablating devices, lead failures, antiarrhythmia device malfunction, etc.).

Dilemmas Faced by Employers

The employer must balance a complex group of sometimes competing responsibilities. First, health and disability insurance, retirement benefits, and compensation in the event of injury on the job must be provided. Employers also carry responsibility for workers compensation for other workers who may be injured on the job by another worker (the patient). Second, employers may face an economic loss for damage caused by a suddenly incapacitated worker. Third, the employer may be liable for personal injury and property losses of third parties, people who may be injured as a result of acts or omissions arising from an employee's medical condition. To minimize risk and maximize the possibility for employment, compliance with myriad laws is required.

Insurance and Insurers

When patients are covered, heart disease will trigger one or more kinds of insurance coverage: health, disability, life, workers compensation, automobile, general liability, and product liability, to name a few. Physicians are also affected by medical malpractice. Whether insurance benefits are paid will be determined by the regulations and factors discussed here. Ultimately all of society is affected by these issues from financial and moral vantage points.

Future Needs

Finally, the following recommendations are made concerning the future. First, a database must be established to obtain prospective data that defines the risk of accidents in patients who are or were being treated for arrhythmias. This will resolve the currently unavoidable problem of underreporting. A database will also permit analysis of subgroups of patients with particular arrhythmias, presentations, and therapies. Current guidelines could then be updated and revised as appropriate. In the future it may be possible to replace standards that impose absolute prohibitions on licensure with performance standards that permit individualized treatment. This is a desirable but as yet theoretical goal. As new information on medical conditions can be joined with basic research and technological advances, performance-based standards may evolve. There must be continued interaction between physicians and regulators in these areas.

APPENDIX

The following is a summary of key points from the US Department of Transportation, Federal Highway Administration Conference on Cardiac Disorders and Commercial Drivers, Bethesda, Md, 1986 (Reference 2).

Dysrhythmias, Sudden Death, and Pacemakers

1. Sinus and Primary Atrial Arrhythmias. Sinus arrhythmia was considered a normal variant of no consequence.

b. Symptomatic sinus bradycardia or tachycardia is abnormal and requires a search for its cause.

c. Asymptomatic sinus bradycardia or tachycardia in the absence of underlying relevant diseases should not be disqualifying.

d. Isolated atrial premature beats (symptomatic or not) and not requiring therapy were not considered disqualifying.

e. Atrial fibrillation and flutter are usually associated with disease states and should preclude commercial driving until adequately evaluated and treated.

f. Multifocal atrial tachycardia is usually associated with serious underlying metabolic or pulmonary disease. Patients with this arrhythmia should not be considered fit for driving.

2. Junctional Rhythms and Paroxysmal Supraventricular Tachycardia. Nonparoxysmal junctional tachycardia was considered to be closely associated with disease states and digitalis toxicity. Medical evaluation is required and driving permitted only if the arrhythmia is asymptomatic and cardiac disease states are excluded.

b. Paroxysmal supraventricular tachycardia (SVT) was considered acceptable if well controlled with any acceptable medical regimen. The same recommendation was provided for patients with ventricular preexcitation (eg, Wolff-Parkinson-White syndrome), although an undefined symptomatic subset will require detailed investigation, and this subset is not qualified for commercial motor vehicle operation.

3. Ventricular Dysrhythmias. Lown grade 3 and above (ie, multiform premature complexes, couplets, three or more consecutively, and R-on-T phenomenon) ventricular arrhythmias were disqualifying unless cleared by a cardiologist on a case-by-case basis.

b. Sustained or nonsustained ventricular tachycardia, whether symptomatic or not, was considered disqualifying.

4. Heart Block. First- and second-degree type I atrioventricular (AV) block were not considered problematic. The implication of narrow versus wide QRS complexes in the setting of second-degree type I AV block was not addressed.

b. Second-degree type II and third-degree AV block were considered disqualifying. Congenital AV block was not addressed.

c. Bundle-branch blocks and fascicular blocks should prompt a search for evidence of intrinsic cardiac abnormalities, but in the absence of such diseases they should not be disqualifying.

5. PacemakersThe majority opinion concluded that insertion of a pacemaker should not in itself be disqualifying, although specialized follow-up was advised. However, a dissenting minority opinion recommending disqualification was included in the report. This opinion recommended disqualification due to total lack of certainty for pacemaker operation. The concept of pacemaker dependency was not addressed.

6. Sudden DeathFor patients who have survived cardiac arrest, the consensus of the conference was that they still carry a substantial risk of additional episodes and should not be considered fit for commercial driving irrespective of the success of subsequent therapy. The impact of ICD treatment was not a consideration at the time of the report.

7. Cardiovascular Pharmacological AgentsFor patients receiving β blockers, concern was raised regarding impaired mental alertness and development of depression and somnolence. Case-by-case decisions with respect to driving qualification were implied. In general, calcium channel blockers were accorded a higher level of safety with respect to driving risk.

In regard to Vaughn Williams Class I and III agents, the conference provided a timely warning against use of antiarrhythmic agents for common benign arrhythmias, pointing out that they often provided no benefit and often cause a worsening of ectopy. They recommended that patients who require such drugs undergo a comparative evaluation before and afterward. The specifics of the recommended evaluations would now be considered inadequate and therefore are not included here.

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Table 1. Ventricular Tachycardia (Table view)

Arrhythmia Type	Private	Commercial
Nonsustained ventricular tachycardia	B ₃ ,* A†	B ₆ ,* A†
Sustained ventricular tachycardia	B ₆ , B ₃ ‡	C, B ₆ ‡
Ventricular fibrillation	B ₆	C

A indicates no restrictions; B, restriction for defined period of months, indicated as subscript to document an arrhythmia-free interval after initiation of therapy (whether implantable cardioverter-defibrillator, antiarrhythmic drug, or both); C, total restriction with presenting arrhythmia.

*Symptoms of impaired consciousness with arrhythmia (before treatment).

†No impairment of consciousness with arrhythmia.

‡Idiopathic ventricular tachycardia (normal coronary arteries, normal ventricular function) and no impairment of consciousness.

Arrhythmia type refers to arrhythmias occurring with no apparent reversible cause; for example, ventricular fibrillation in the setting of acute myocardial infarction, severe electrolyte abnormality, drug toxicity, or electrocution would be excluded.

Table 2. Supraventricular Tachycardia (Table view)

Condition	Private or Commercial
Asymptomatic or minimally symptomatic SVT (including WPW syndrome)	A
Symptomatic* SVT	B†
Atrial fibrillation treated by catheter ablation of AV node	B (see guidelines for pacing)
SVT with uncontrolled symptoms	C

SVT indicates supraventricular tachycardia; WPW, Wolff-Parkinson-White; AV, atrioventricular; A, no restrictions; B, restriction for defined period of months, indicated as subscript to document an arrhythmia-free interval after initiation of therapy (whether implantable cardioverter-defibrillator, antiarrhythmic drug, or both); C, total restriction with presenting arrhythmia.

*Symptomatic: evidence of hemodynamic compromise such as syncope, presyncope, chest pain, or dyspnea; palpitations alone are not sufficient.

†Restrictions until after initiation of therapy that eliminates symptoms. Patients with supraventricular tachycardia that appears to be successfully ablated can drive after recovery from the procedure. Patients treated with drug therapy should as a minimum have a 1-month symptom-free period before resuming driving, depending on pretherapy frequency of tachycardia.

Table 3. Bradycardia With and Without Cardiac Pacing (Table view)

Condition	Noncommercial	Commercial
No symptoms No pacemaker	A	A
Syncope or near syncope No pacemaker	C	C
Not pacemaker-dependent* Yes, pacemaker	A	A
Pacemaker-dependent* Yes, pacemaker	B—1 wk	B—4 wk

A indicates no driving restrictions; B, driving permitted after controlled arrhythmia is documented for a specified period of time and an adequate pacemaker follow-up regimen is followed; C, driving completely prohibited.

*For these purposes, “pacemaker-dependent” is defined as patients who have lost consciousness in the past due to bradyarrhythmias. It may also include patients immediately after atrioventricular junction ablation or any other patient in whom sudden pacemaker failure would be likely to result in alteration of consciousness.

Table 4. Neurally Mediated Syncope and Presyncope (Table view)

	Private	Commercial
VVS mild	A	B ₁
VVS severe		
Treated	B ₃	B ₆
Untreated	C	C
CSS mild	A	A
CSS severe		
Treated with control	B ₁	B ₁
Treated with uncertain control	B ₃	B ₆
Untreated	C	C

VVS indicates vasovagal syncope or presyncope; CSS, carotid sinus syncope or presyncope (mild and severe forms are defined in text); control, pause less than 3 sec with decrease in systolic blood pressure ≤10 mm Hg with carotid sinus massage after therapy; A, no driving restrictions; B, driving permitted after controlled arrhythmia is documented for a specified period of time and an adequate pacemaker follow-up regimen is followed; C, driving completely prohibited.

ARTICLE INFORMATION

Affiliations

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REFERENCES

1. Rigdon JE. Rise in older drivers poses safety risk. *The Wall Street Journal*, October 29, 1993:A8.
2. US Department of Transportation, Federal Highway Administration. *Conference on Cardiac Disorders and Commercial Drivers*. Bethesda, Md; December 1987. Publication No. FHWA-MC-88-040.
3. Parsons M. Fits and other causes of loss of consciousness while driving. *Q J Med.* 1986;58:295-303. [PubMed](#).
4. Shephard RJ. The cardiac patient and driving: the Ontario experience. In: *Conference on Cardiac Disorders and Commercial Drivers*. Bethesda, Md: US Department of Transportation, Federal Highway Administration; December 1987:85-94.
5. Medical regulatory criteria for evaluation. Section 391.41(b)(4). *Federal Register*. November 23, 1977; amended October 1983.
6. McCue H. Cardiac arrhythmias in relation to automobile driving. Presented at the Scientific Conference on Personal and Public Safety Issues Related to Arrhythmias; January 12-13, 1995; Washington, DC.
7. Consensus Conference, Canadian Cardiovascular Society. Assessment of the cardiac patient for fitness to drive. *Can J Cardiol.* 1992;8:406-412.
8. Bennett G. Medical-cause accidents in commercial aviation. *Eur Heart J.* 1992;13(suppl H):13-15.
9. Joy M. Cardiological aspects of aviation safety: the new European perspective. In: The First European Workshop in Aviation Cardiology. *Eur Heart J.* 1992;13(suppl H):21-26.
10. Joy M. Class I cardiovascular standards. In: *European Manual of Civil Aviation Medicine*. Neuilly-sur-Seine, France: Joint Aviation Authority. In press.
11. Mason KT. Military flying and cardiac arrhythmia policy. Presented at the Scientific Conference on Personal and Public Safety Issues Related to Arrhythmias; January 12-13, 1995; Washington, DC.
12. Kruyer WB. Military aviation and specific cardiac arrhythmias. Presented at the Scientific Conference on Personal and Public Safety Issues Related to Arrhythmias; January 12-13, 1995; Washington, DC.
13. Norman LG. Medical aspects of road safety. *Lancet.* 1960;1:989-994, 1039-1045. [PubMed](#).
14. Trapnell JM, Groff HD. Myocardial infarction in commercial drivers. *J Occup Med.* 1963;5:182-184. [PubMed](#).
15. Herner B, Smedby B, Ysander L. Sudden illness as a cause of motor-vehicle accidents. *Br J Ind Med.*

- 1966;23:37-41.
16. West I, Nielsen GL, Gilmore AE, Ryan JR. Natural death at the wheel. *JAMA*. 1968;205:266-271. [Crossref](#). [PubMed](#).
 17. Myerburg RJ, Davis JH. The medical ecology of public safety: I. sudden death due to coronary heart disease. *Am Heart J*. 1964;68:586-595. [Crossref](#). [PubMed](#).
 18. Kerwin AJ. Sudden death while driving. *Can Med Assoc J*. 1984;131:312-314. [PubMed](#).
 19. Peterson BJ, Petty CS. Sudden natural death among automobile drivers. *J Forensic Sci*. 1962;7:274-285.
 20. Hossack DS. Death at the wheel: a consideration of cardiovascular disease as a contributory factor to road accidents. *Med J Aust*. 1974;1:164-166.
 21. Ostrom M, Eriksson A. Natural death while driving. *J Forensic Sci*. 1987;32:988-998. [PubMed](#).
 22. Christian MS. Incidence and implications of natural deaths of road users. *Br Med J*. 1988;297:1021-1024. [Crossref](#). [PubMed](#).
 23. Grattan E, Jeffcoate GO. Medical factors and road accidents. *Br Med J*. 1968;1:75-79. [Crossref](#). [PubMed](#).
 24. Larsen GC, Stupey MR, Walance CG, Griffith KK, Cutler JE, Kron J, McAnulty JH. Recurrent cardiac events in survivors of ventricular fibrillation or tachycardia: implications for driving restrictions. *JAMA*. 1994;271:1335-1339. [Crossref](#). [PubMed](#).
 25. Waller JA. Cardiovascular disease, aging, and traffic accidents. *J Chronic Dis*. 1967;20:615-620. [Crossref](#). [PubMed](#).
 26. Fogoros RN, Elson JJ, Bonnet CA. Actuarial incidence and pattern of occurrence of shocks following implantation of the automatic implantable cardioverter defibrillator. *PACE Pacing Clin Electrophysiol*. 1989;12:1465-1473.
 27. Tchou P, Axtell K, Anderson AJ, Keim S, Sra J, Troup P, Jazayeri M, Avitall B, Akhtar M. When is it safe not to replace an implantable cardioverter defibrillator generator? *PACE Pacing Clin Electrophysiol*. 1991;14:1875-1880. [Crossref](#). [PubMed](#).
 28. Kou WH, Calkins H, Lewis RR, Bolling SF, Kirsch MM, Langberg JJ, de Buitelir M, Sousa J, el-Atassi R, Morady F. Incidence of loss of consciousness during automatic implantable cardioverter-defibrillator shocks. *Ann Intern Med*. 1991;115:942-945. [Crossref](#). [PubMed](#).
 29. Baker SP, Spitz WU. An evaluation of the hazard created by natural death at the wheel. *N Engl J Med*. 1970;283:405-409. [Crossref](#). [PubMed](#).
 30. Antecol DH, Roberts WC. Sudden death behind the wheel from natural disease in drivers of four-wheeled motorized vehicles. *Am J Cardiol*. 1990;66:1329-1335. [Crossref](#). [PubMed](#).
 31. Curtis AB, Conti JB, Tucker KJ, Kubilis PS, Reilly RE, Woodard DA. Motor vehicle accidents in patients with an implantable cardioverter-defibrillator. *J Am Coll Cardiol*. 1995;26:180-184. [Crossref](#). [PubMed](#).
 32. Packer M. Sudden unexpected death in patients with congestive heart failure: a second frontier. *Circulation*. 1985;72:681-685. [Crossref](#). [PubMed](#).
 33. Prystowsky EN, Klein GJ. *Cardiac Arrhythmias: An Integrated Approach for the Clinician*. New York, NY: McGraw-Hill; 1994.
 34. Hurwitz JL, German LD, Packer DL, Wharton JM, McCarthy EA, Wilkinson WE, Prystowsky EN, Pritchett EL. Occurrence of atrial fibrillation in patients with paroxysmal supraventricular tachycardia due to atrioventricular nodal reentry. *PACE Pacing Clin Electrophysiol*. 1990;13:705-710. [Crossref](#). [PubMed](#).
 35. Campbell RW, Smith RA, Gallagher JJ, Pritchett EL, Wallace AG. Atrial fibrillation in the preexcitation syndrome. *Am J Cardiol*. 1977;40:515-520.
 36. Prystowsky EN. Tachycardia-induced tachycardia: a mechanism of initiation of atrial fibrillation. In: DiMarco

- JP, Prystowsky EN, eds. *Atrial Arrhythmias: State of the Art*. Armonk, NY: Futura Publishing Co; 1995:81-95.
37. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med*. 1979;301:1080-1085.
 38. Epstein LM, Scheinman MM, Langberg JJ, Chilson D, Goldberg HR, Griffin JC. Percutaneous catheter modification of the atrioventricular node: a potential cure for atrioventricular nodal reentrant tachycardia. *Circulation*. 1989;80:757-768. [Crossref](#). [PubMed](#).
 39. Lee MA, Morady F, Kadish A, Schamp DJ, Chin MC, Scheinman MM, Griffin JC, Lesh MD, Pederson D, Goldberger J, Calkins H, de Buitelir M, Kou WH, Rosenheck S, Sousa J, Langberg JJ. Catheter modification of the atrioventricular junction with radiofrequency energy for control of atrioventricular nodal reentry tachycardia. *Circulation*. 1991;83:827-835. [Crossref](#). [PubMed](#).
 40. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Selective radiofrequency ablation of the slow pathway for the treatment of atrioventricular nodal reentrant tachycardia: evidence for involvement of perinodal myocardium within the reentrant circuit. *Circulation*. 1992;85:1675-1688. [Crossref](#). [PubMed](#).
 41. Jackman WM, Beckman KJ, McClelland JH, Wang X, Friday KJ, Roman CA, Moulton KP, Twidale N, Hazlitt HA, Prior MI, Oren J, Overholt ED, Lazzara R. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter ablation of slow-pathway conduction. *N Engl J Med*. 1992;327:313-318.
 42. Jazayeri MR, Hempe SL, Sra JS, Dhala AA, Blanck Z, Deshpande SS, Avitall B, Krum DP, Gilbert CJ, Akhtar M. Selective transcatheter ablation of the fast and slow pathways using radiofrequency energy in patients with atrioventricular nodal reentrant tachycardia. *Circulation*. 1992;85:1318-1328. [Crossref](#). [PubMed](#).
 43. Mitrani RD, Klein LS, Hackett FK, Zipes DP, Miles WM. Radiofrequency ablation for atrioventricular node reentrant tachycardia: comparison between fast (anterior) and slow (posterior) pathway ablation. *J Am Coll Cardiol*. 1993;21:432-441. [Crossref](#). [PubMed](#).
 44. Lindsay BD, Chung MK, Gamache MC, Luke RA, Schechtman KB, Osborn JL, Cain ME. Therapeutic end points for the treatment of atrioventricular node reentrant tachycardia by catheter-guided radiofrequency current. *J Am Coll Cardiol*. 1993;22:733-740. [Crossref](#). [PubMed](#).
 45. Li HG, Klein GJ, Stites HW, Zardini M, Morillo CA, Thakur RK, Yee R. Elimination of slow pathway conduction: an accurate indicator of clinical success after radiofrequency atrioventricular node modification. *J Am Coll Cardiol*. 1993;22:1849-1853. [Crossref](#). [PubMed](#).
 46. Chen SA, Chiang CE, Tsang WP, Hsia CP, Wang DC, Yeh HI, Ting CT, Chuen WC, Yang CJ, Cheng CC, Wang SP, Chiang BN, Chang MS. Selective radiofrequency catheter ablation of fast and slow pathways in 100 patients with atrioventricular nodal reentrant tachycardia. *Am Heart J*. 1993;125:1-10. [Crossref](#). [PubMed](#).
 47. Bennett DH, Coyle C. Development of multiform ventricular tachycardia during atrioventricular nodal reentrant tachycardia. *Br Heart J*. 1989;62:220-221. [Crossref](#). [PubMed](#).
 48. Hays LJ, Lerman BB, DiMarco JP. Nonventricular arrhythmias as precursors of ventricular fibrillation in patients with out-of-hospital cardiac arrest. *Am Heart J*. 1989;118:53-57. [Crossref](#). [PubMed](#).
 49. Wang YS, Scheinman MM, Chien WW, Cohen TJ, Lesh MD, Griffin JC. Patients with supraventricular tachycardia presenting with aborted sudden death: incidence, mechanism and long-term follow-up. *J Am Coll Cardiol*. 1991;18:1711-1719. [Crossref](#). [PubMed](#).
 50. Pritchett EL, McCarthy EA, Lee KL. Clinical behavior of paroxysmal atrial tachycardia. *Am J Cardiol*. 1988;62:3D-9D. [PubMed](#).
 51. Jackman WM, Wang XZ, Friday KJ, Roman CA, Moulton KP, Beckman KJ, McClelland JH, Twidale N,

- Hazlitt HA, Prior MI, Margolis PD, Calame JD, Overholt ED, Lazarra R. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. *N Engl J Med.* 1991;324:1605-1611.
52. Schluter M, Geiger M, Siebels J, Duckeck W, Kuck KH. Catheter ablation using radiofrequency current to cure symptomatic patients with tachyarrhythmias related to an accessory atrioventricular pathway. *Circulation.* 1991;84:1644-1661. [Crossref](#). [PubMed](#).
 53. Calkins H, Langberg J, Sousa J, el-Atassi R, Leon A, Kou W, Kalbfleisch S, Morady F. Radiofrequency catheter ablation of accessory atrioventricular connections in 250 patients: abbreviated therapeutic approach to Wolff-Parkinson-White syndrome. *Circulation.* 1992;85:1337-1346. [Crossref](#). [PubMed](#).
 54. Swartz JF, Tracy CM, Fletcher RD. Radiofrequency endocardial catheter ablation of accessory atrioventricular pathway atrial insertion sites. *Circulation.* 1993;87:487-499. [Crossref](#). [PubMed](#).
 55. Saul JP, Hulse JE, De W, Weber AT, Rhodes LA, Lock JE, Walsh EP. Catheter ablation of accessory atrioventricular pathways in young patients: use of long vascular sheaths, the transseptal approach and a retrograde left posterior parallel approach. *J Am Coll Cardiol.* 1993;21:571-583. [Crossref](#). [PubMed](#).
 56. Klein GJ, Prystowsky EN, Yee R, Sharma AD, Laupacis A. Asymptomatic Wolff-Parkinson-White: should we intervene? *Circulation.* 1989;80:1902-1905. [Crossref](#). [PubMed](#).
 57. Bink-Boelkens M. Dysrhythmias after atrial surgery in children. *Am Heart J.* 1983;106:125-129. [Crossref](#). [PubMed](#).
 58. Porter CJ, Battiste CE, Humes RA, Offord KP, Puga FJ, Schaff HV, Danielson GK. Risk factors for supraventricular arrhythmias after Fontan procedure for tricuspid atresia. *Am Heart J.* 1986;112:645. Abstract.
 59. Garson A. Electrophysiology and arrhythmias in young patients: considerations for antiarrhythmic drug regulation. *J Cardiovasc Electrophysiol.* 1991;2:450-455. [Crossref](#).
 60. Garson A Jr, Bink-Boelkens M, Hesslein PS, Hordof AJ, Keane JF, Neches WH, Porter CJ, and other investigators of the Pediatric Electrophysiology Society. Atrial flutter in the young: a collaborative study of 380 cases. *J Am Coll Cardiol.* 1985;6:871-878. [Crossref](#). [PubMed](#).
 61. Fitzpatrick A, Theodorakis G, Vardas P, Kenny RA, Travill CM, Ingram A, Sutton R. The incidence of malignant vasovagal syndrome in patients with recurrent syncope. *Eur Heart J.* 1991;12:389-394.
 62. Dhingra RC, Denes P, Wu D, Chuquimia R, Amat-y-Leon F, Wyndham C, Rosen KM. Syncope in patients with chronic bifascicular block: significance, causative mechanisms, and clinical implications. *Ann Intern Med.* 1974;81:302-306. [Crossref](#). [PubMed](#).
 63. Ezri M, Lerman BB, Marchlinski FE, Buxton AE, Josephson ME. Electrophysiologic evaluation of syncope in patients with bifascicular block. *Am Heart J.* 1983;106:693-697. [Crossref](#). [PubMed](#).
 64. Benditt DG, Sakaguchi S, Shultz JJ, Remole S, Adler S, Lurie KG. Syncope: diagnostic considerations and the role of tilt table testing. *Cardiol Rev.* 1993;1:146-156.
 65. Almquist A, Goldenberg IF, Milstein S, Chen MY, Chen XC, Hansen R, Gornick CC, Benditt DG. Provocation of bradycardia and hypotension by isoproterenol and upright posture in patients with unexplained syncope. *N Engl J Med.* 1989;320:346-351. [Crossref](#). [PubMed](#).
 66. Fouad FM, Sitthisook S, Vanerio G, Maloney J III, Okabe M, Jaeger F, Schluchter M, Maloney JD. Sensitivity and specificity of the tilt table test in young patients with unexplained syncope. *PACE Pacing Clin Electrophysiol.* 1993;16:394-400. [Crossref](#). [PubMed](#).
 67. Sheldon R, Splawinski J, Killam S. Reproducibility of isoproterenol tilt-table tests in patients with syncope. *Am J Cardiol.* 1992;69:1300-1305. [Crossref](#). [PubMed](#).
 68. Winkle RA, Mead RH, Ruder MA, Gaudiani VA, Smith NA, Buch WS, Schmidt P, Shipman T. Long-term

- outcome with the automatic implantable cardioverter-defibrillator. *J Am Coll Cardiol*. 1989;13:1353-1361.
69. Fogoros RN, Elson JJ, Bonnet CA. Survival of patients who have received appropriate shocks from their implantable defibrillators. *PACE Pacing Clin Electrophysiol*. 1991;94:1842-1845.
 70. Grimm W, Flores BT, Marchlinski FE. Shock occurrence and survival in 241 patients with implantable cardioverter-defibrillator therapy. *Circulation*. 1993;87:1880-1888.
 71. Levine JH, Mellits ED, Baumgardner RA, Veltri EP, Mower M, Grunwald L, Guarnieri T, Aarons D, Griffith LS. Predictors of first discharge and subsequent survival in patients with automatic implantable cardioverter-defibrillators. *Circulation*. 1991;84:558-566. [Crossref](#). [PubMed](#).
 72. Grimm W, Flores BF, Marchlinski FE. Symptoms and electrocardiographically documented rhythm preceding spontaneous shocks in patients with implantable cardioverter-defibrillator. *Am J Cardiol*. 1993;71:1415-1418. [Crossref](#). [PubMed](#).
 73. Anderson MH, Katritsis D, Gibson SA, Ross DJ, Pickering A. Automobile driving and ventricular arrhythmias: implications of a study of motor performance during rapid ventricular pacing. *Circulation*. 1992;86(suppl I):I-313. Abstract.
 74. Steinbach KK, Merl O, Frohner K, Hief C, Nurnberg M, Kaltenbrunner W, Podczek A, Wessely E. Hemodynamics during ventricular tachyarrhythmias. *Am Heart J*. 1994;127:1102-1106. [Crossref](#). [PubMed](#).
 75. Steinbeck G, Dorwarth U, Mattke S, Hoffmann E, Markewitz A, Kaulbach H, Tassani P. Hemodynamic deterioration during ICD implant: predictors of high-risk patients. *Am Heart J*. 1994;127:1064-1067. [Crossref](#). [PubMed](#).
 76. Myerburg RJ, Luceri RM, Thurer R, Cooper DK, Zaman L, Interian A, Fernandez P, Cox M, Glicksman F, Castellanos A. Time to first shock and clinical outcome in patients receiving an automatic implantable cardioverter-defibrillator. *J Am Coll Cardiol*. 1989;14:508-514. [Crossref](#). [PubMed](#).
 77. Kelly PA, Cannom DS, Garan H, Mirabal GS, Harthorne JW, Hurvitz RJ, Vlahakes GJ, Jacobs ML, Ilvento JP, Buckley MJ, Ruskin JN. The automatic implantable cardioverter-defibrillator: efficacy, complications and survival in patients with malignant ventricular arrhythmias. *J Am Coll Cardiol*. 1988;11:1278-1286. [Crossref](#). [PubMed](#).
 78. Curtis JJ, Walls JT, Boley TM, Stephenson HE, Schmaltz RA, Nawarawong W, Flaker GC. Time to first pulse after automatic implantable cardioverter defibrillator implantation. *Ann Thorac Surg*. 1992;53:984-987. [Crossref](#). [PubMed](#).
 79. Johnson NJ, Marchlinski FE. Arrhythmias induced by device antitachycardia therapy due to diagnostic nonspecificity. *J Am Coll Cardiol*. 1991;18:1418-1422. [Crossref](#). [PubMed](#).
 80. Marchlinski RE, Kleinman RB, Hook BG, Pachulski RT, Ratcliffe M. Advances in implantable cardioverter defibrillator therapy. In: Josephson ME, Wellens HJJ, eds. *Tachycardias: Mechanisms and Management*. Mt. Kisco, NY: Futura Publishing Co; 1993:405-420.
 81. Fromer M, Brachmann J, Block M, Siebels J, Hoffmann E, Almendral J, Ohm OJ, den Dulk K, Coumel P, Camm AJ, Toubol P. Efficacy of automatic multimodal device therapy for ventricular tachyarrhythmias as delivered by a new implantable pacing cardioverter-defibrillator: results of a European multicenter study of 102 implants. *Circulation*. 1992;86:363-374. [Crossref](#). [PubMed](#).
 82. Maloney J, Masterson M, Khoury D, Trohman R, Wilkoff B, Simmons T, Morant V, Castle L. Clinical performance of the implantable cardioverter defibrillator: electrocardiographic documentation of 101 spontaneous discharges. *PACE Pacing Clin Electrophysiol*. 1991;14:280-285. [Crossref](#). [PubMed](#).
 83. Hook BG, Marchlinski FE. Value of ventricular electrogram recordings in the diagnosis of arrhythmias precipitating electrical device shock therapy. *J Am Coll Cardiol*. 1991;17:985-990. [Crossref](#). [PubMed](#).
 84. Hook BG, Callans DJ, Kleiman RB, Flores BT, Marchlinski FE. Implantable cardioverter-defibrillator therapy

- in the absence of significant symptoms: rhythm diagnosis and management aided by stored electrogram analysis. *Circulation*. 1993;87:1897-1906. [Crossref](#). [PubMed](#).
85. Grimm W, Flores BT, Marchlinski FE. Electrocardiographically documented unnecessary, spontaneous shocks in 241 patients with implantable cardioverter defibrillators. *PACE Pacing Clin Electrophysiol*. 1992;15:1667-1673. [Crossref](#). [PubMed](#).
86. Marchlinski FE, Gottlieb CD, Sarter B, Finkle J, Hook B, Callans D, Schwartzman D. ICD data storage: value in arrhythmia management. *PACE Pacing Clin Electrophysiol*. 1993;16:527-534. [Crossref](#). [PubMed](#).
87. Meissner MD, Lehmann MH, Steinman RT, Mosteller RD, Akhtar M, Calkins H, Cannom DS, Epstein AE, Fogoros RN, Liem LB, Marchlinski FE, Myerburg RD, Veltri EP. Ventricular fibrillation in patients without significant structural heart disease: a multicenter experience with implantable cardioverter-defibrillator therapy. *J Am Coll Cardiol*. 1993;21:1406-1412. [Crossref](#). [PubMed](#).
88. Lessmeier TJ, Lehmann MH, Steinman RT, Fromm B, Akhtar M, Calkins H, DiMarco JP, Epstein AE, Estes NA, Fogoros RN, Marchlinski FE, Wilber D. Outcome with implantable cardioverter-defibrillator therapy for survivors of ventricular fibrillation secondary to idiopathic dilated cardiomyopathy or coronary artery disease without myocardial infarction. *Am J Cardiol*. 1993;72:911-915. [Crossref](#). [PubMed](#).
89. Anderson MH, Camm AJ. Legal and ethical aspects of driving and working in patients with an implantable cardioverter defibrillator. *Am Heart J*. 1994;127:1185-1193.
90. Grimm W, Marchlinski FE. Shock occurrence in patients with implantable cardioverter-defibrillator without spontaneous shocks before first generator replacement for battery depletion. *Am J Cardiol*. 1994;73:969-970. [Crossref](#). [PubMed](#).
91. Gross JN, Song SL, Buckingham T, Furman S. Influence of clinical characteristics and shock occurrence on ICD patient outcome: a multicenter report—The Bilitch Registry Group. *PACE Pacing Clin Electrophysiol*. 1991;14:1881-1886. [Crossref](#). [PubMed](#).
92. Mitra RL, Hsia HH, Hook BG, Callans DJ, Flores BT, Miller JM, Josephson ME, Marchlinski FE. Implantable defibrillators with antitachycardia pacing reduce the number of shocks in cardiac arrest patients who have inducible sustained ventricular tachycardia. *J Am Coll Cardiol*. 1993;21:126A.
93. Roy D, Waxman HL, Buxton AE, Marchlinski FE, Cain ME, Gardner MJ, Josephson ME. Termination of ventricular tachycardia: role of tachycardia cycle length. *Am J Cardiol*. 1982;50:1346-1350. [Crossref](#). [PubMed](#).
94. Gottlieb CD, Horowitz LN. Potential interactions between antiarrhythmic medication and the automatic implantable cardioverter defibrillator. *PACE Pacing Clin Electrophysiol*. 1991;14:898-904. [Crossref](#). [PubMed](#).
95. Wang M, Dorian P. DL and D sotalol decrease defibrillation energy requirements. *PACE Pacing Clin Electrophysiol*. 1989;12:1522-1529. [Crossref](#). [PubMed](#).
96. Natale A, Jones DL, Kim Y, Klein GJ. Effects of lidocaine on the defibrillation threshold in the pig: evidence of anesthesia-related increase. *PACE Pacing Clin Electrophysiol*. 1991;14:1239-1244. [Crossref](#). [PubMed](#).
97. Willems S, Borggrefe M, Shenasa M, Chen X, Hindricks G, Haverkamp W, Wietholt D, Block M, Breithardt G. Radiofrequency catheter ablation of ventricular tachycardia following implantation of an automatic cardioverter defibrillator. *PACE Pacing Clin Electrophysiol*. 1993;16:1684-1692. [Crossref](#). [PubMed](#).
98. Morady F, Harvey M, Kalbfleisch SJ, el-Atassi R, Calkins H, Langberg JJ. Radiofrequency catheter ablation of ventricular tachycardia in patients with coronary artery disease. *Circulation*. 1993;87:363-372. [Crossref](#). [PubMed](#).
99. Stevenson WG, Khan H, Sager P, Saxon LA, Middlekauff HR, Natterson PD, Wiener I. Identification of reentry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. *Circulation*. 1993;88:1647-1670. [Crossref](#). [PubMed](#).

100. Higgins JR. Automatic burst extrastimulus pacemaker to treat recurrent ventricular-tachycardia in a patient with mitral-valve prolapse. *J Am Coll Cardiol.* 1986;8:446-450. [Crossref](#). [PubMed](#).
101. Paul T, Guccione P, Garson A Jr. Relation of syncope in young patients with Wolff-Parkinson-White syndrome to rapid ventricular response during atrial fibrillation. *Am J Cardiol.* 1990;65:318-321. [Crossref](#). [PubMed](#).
102. Leitch JW, Klein GJ, Yee R, Leather RA, Kim YH. Syncope associated with supraventricular tachycardia: an expression of tachycardia rate or vasomotor response? *Circulation.* 1992;85:1064-1071. [Crossref](#). [PubMed](#).
103. Fitzpatrick AP, Theodorakis G, Vardas P, Sutton R. Methodology of head-up tilt testing in patients with unexplained syncope. *J Am Coll Cardiol.* 1991;17:125-130. [Crossref](#). [PubMed](#).
104. Yee R, Klein GJ. Syncope in the Wolff-Parkinson-White syndrome: incidence and electrophysiologic correlates. *PACE Pacing Clin Electrophysiol.* 1984;7:381-388. [Crossref](#). [PubMed](#).
105. Leitch JW, Klein GJ, Yee R, Murdock C. Prognostic value of electrophysiology testing in asymptomatic patients with Wolff-Parkinson-White pattern [erratum *Circulation* 1991;83:1124]. *Circulation.* 1990;82:1718-1723. [Crossref](#). [PubMed](#).
106. Kannel WB, Wolf PA. Epidemiology of atrial fibrillation. In: Falk RH, Podrid PJ, eds. *Atrial Fibrillation: Mechanisms and Management.* New York, NY: Raven Press; 1992:81-92.
107. Pavlovic SU, Kocovic D, Djordjevic M, Blekic K, Kostic D, Velimirovic D. The etiology of syncope in pacemaker patients. *PACE Pacing Clin Electrophysiol.* 1991;14:2086-2091.
108. Sgarbossa EB, Pinski SL, Jaeger FJ, Trohman RG, Maloney JD. Incidence and predictors of syncope in paced patients with sick sinus syndrome. *PACE Pacing Clin Electrophysiol.* 1992;15:2055-2060. [Crossref](#). [PubMed](#).
109. Edhag O, Wedelin EM. Long-term cardiac pacing: experience of fixed-rate pacing with an endocardial electrode in 260 patients, 13. Rehabilitation of paced patients. *Acta Med Scand.* 1969;502(suppl):81-92.
110. Brandaleone H. Motor vehicle driving and cardiac pacemakers. *Ann Intern Med.* 1974;81:548-550. [Crossref](#). [PubMed](#).
111. Sowton E. Driving licenses for patients with cardiac pacemakers. *Br Heart J.* 1972;34:977-980. [Crossref](#). [PubMed](#).
112. Sowton E. Use of cardiac pacemakers in Britain. *Br Med J.* 1976;2:1182-1184. [Crossref](#). [PubMed](#).
113. Jelic V, Belkic K, Djordjevic M, Kocovic D. Survival in 1,431 pacemaker patients: prognostic factors and comparison with the general population. *PACE Pacing Clin Electrophysiol.* 1992;15:141-147. [Crossref](#). [PubMed](#).
114. Levine PA. Proceedings of the policy conference of the North American Society of Pacing and Electrophysiology on programmability and pacemaker follow-up programs. *Clin Prog Pacing Electrophysiol.* 1984;2:145-191. [Crossref](#).
115. Huang SK, Ezri MD, Hauser RG, Denes P. Carotid sinus hypersensitivity in patients with unexplained syncope: clinical, electrophysiologic, and long-term follow-up observations. *Am Heart J.* 1988;116:989-996. [Crossref](#). [PubMed](#).
116. Brignole M, Menozzi C, Lolli G, Bottoni N, Gaggioli G. Long-term outcome of paced and nonpaced patients with severe carotid sinus syndrome. *Am J Cardiol.* 1992;69:1039-1043. [Crossref](#). [PubMed](#).
117. Dreifus LS, Fisch C, Griffin JC, Gillette PC, Mason JW, Parsonnet V. Guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Committee on Pacemaker Implantation). *J Am Coll Cardiol.* 1991;18:1-13. [Crossref](#). [PubMed](#).

118. *Highway Statistics 1992*. Bethesda, Md: US Department of Transportation, Federal Highway Administration; 1993. Publication No. FHWA-PL-93-03.
119. Saliba WI, Rizo-Patron C, El-Souki R, et al. Pacing lead survival of three safety alert models: a second look. *Circulation*. 1994;90(suppl I):I-70. Abstract.