**U.S. Department
Of Transportation**

**Federal Motor Carrier
Safety Administration**

Cardiovascular Advisory Panel Guidelines for the Medical Examination of Commercial Motor Vehicle Drivers

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EXECUTIVE SUMMARY

Cardiovascular disease (CVD) is the leading cause of medical illness and sudden death in commercial motor vehicle drivers (CMV). CVD will have an increasingly powerful impact on the health and safety of CMV drivers because of its prevalence in the population, its progressive nature, the aging work force, and recent advances in diagnosis and therapy.

The Federal Motor Carrier Safety Administration (FMCSA) administers the Federal Motor Carrier Safety Regulations (FMCSRs) concerning the medical qualifications of commercial drivers in interstate commerce. While only a small percentage of crashes are caused by cardiovascular disease, they are responsible for significant mortality and morbidity.

The Department of Transportation (DOT) examination is an essential part of assuring a healthy CMV driver workforce. The guidelines assist medical examiners in the evaluation and certification of each person on whom they perform a DOT examination. The last DOT review of its cardiac guidelines for CMV drivers was published in December 1987.

In fall, 2001, the FMCSA convened a Cardiovascular Medical Advisory Panel to develop new guidelines to reflect the medical advances that have occurred over the last 15 years. Panel members submitted medical review papers on their topics. The papers reviewed the currently accepted scientific opinion on the risks, diagnoses and treatments of numerous cardiovascular diseases. For easier use, the recommendations are summarized and placed in table format at the end of each paper.

Each Panel member's topic is intended to assist medical examiners in determining if the commercial vehicle driver's cardiovascular condition increases his/her risk of sudden death or incapacitation that the driver endangers their health and safety and the health and safety of the public sharing the road with them. The level of risk must be considered within the context of the setting and activity in question and what society considers acceptable. Determining “acceptable risk” becomes a matter of public policy and the decision to certify or disqualify a commercial driver is both a medical and a societal decision.

SUMMARY OF TOPICS

Ischemic Heart Disease Dr. Roger Blumenthal and Dr. Joel Braunstein

Findings
Almost 12% of those over age 40 have coronary heart disease (CHD). In the general population, the initial presentation of CHD is catastrophic in over two-thirds of cases.

Risk factor identification and treatment is the key strategy in decreasing the mortality and morbidity of CHD. Commercial drivers have an increased prevalence of cardiovascular risk factors relative to other occupations; specific work-related factors further elevate the risk of CVD.

Recommendations
There is some evidentiary utility for using the exercise tolerance test (ETT) to assess CMV drivers who have risk factors but no symptoms or signs of CHD. Because of its lack of specificity and sensitivity and its unknown cost effectiveness when used as a screening tool in this population, the Panel is not able to recommend for or against this strategy.

Medical examiners have been provided recommendation tables to assist in deciding whether to certify commercial drivers with coronary artery risk factors, with known CHD, following a cardiac event, or following a cardiac procedure.

Hypertension Dr. Roger Blumenthal and Dr. Joel Braunstein

Findings
An estimated 50 million Americans have hypertension. Commercial drivers have an increased propensity for the development of hypertension that exceeds the risk seen in other professions. Long-term data show increased rates of cerebral, cardiac, and renal complications in patients with elevated blood pressure. Hypertension is progressive in nature if uncontrolled and requires regular follow-up. The effect of hypertension on target organs also increases the risk of sudden incapacitation.

Recommendations
The Panel adopted the sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 6). Certification, disqualification, and follow-up are based on the blood pressure. A blood pressure exceeding 140/90 mmHg is considered elevated for most individuals who have no other cardiovascular risk factors.

Valvular Disease and Myocardial Disease Dr. Bernard J. Gersh

Findings
Consistent with other CVD, improved diagnostic testing and treatment can increase the number of CMV drivers with valvular or myocardial disease who seek certification. Valvular and myocardial diseases are often progressive and require long-term follow-up. This section reviews the risks of these diseases and the effect of prosthetic valves on certification.

Recommendations
Due to the complexity of this area, a cardiologist's assessment is often recommended. Because of the progressive nature of these diseases, there is a need for follow-up evaluations of these drivers.

This comprehensive review includes the current recommendations from the American College of Cardiology and the American Heart Association. The use of these guidelines makes assessment of the driver more uniform and provides accepted medical standards for medical examiners.

Arrhythmias Dr. Andrew Epstein

Findings
Arrhythmia is the most likely cause of sudden death or driver incapacity, with CHD as its underlying etiology. Arrhythmia, depending on the type, location and classification fall along the spectrum from harmless to instantaneously fatal. Certain arrhythmias are more likely to produce conditions that most threaten the safety of the public and the driver: syncope (fainting), collapse, sudden death or other sudden incapacitation. Moreover, the more serious arrhythmias often occur in those with no prior knowledge or diagnosis of heart disease. The review includes arrhythmias that can produce hemodynamic compromise, pacemakers, and implantable cardioverter-defibrillators.

Recommendations
In addition to risk-identification and management of arrhythmia, treatment of the underlying heart disease (if present) is of paramount importance.

Congenital Heart Disease Dr. Heidi Connolly

Findings
Heart failure and sudden death are the major causes of death among patients with congenital heart disease. Because of advances in surgical and medical management, over 85% of infants born with congenital heart diseases are expected to survive to adult life. The number of individuals with congenital heart disease requesting commercial driver certification is expected to rise proportional to the increasing patient population. Applicants for certification are likely to be those with milder forms of congenital heart disease or those who have had surgical repair.

Recommendations
To maintain certification, it is recommended that these drivers have regular, ongoing follow-up by a cardiologist knowledgeable in adult congenital heart disease.

Diseases of the Arteries and Veins Dr. Ellison Wittels

Findings

Arterial disease is most often secondary to atherosclerosis. The diagnosis of arterial disease should trigger an evaluation for the presence of other cardiovascular diseases. Rupture is the most feared complication of an abdominal aortic aneurysm (AAA), and is related to the size of the aneurysm. Intermittent claudication is the primary symptom of peripheral vascular disease (PVD) of the lower extremities, usually a slowly progressive disease. Deep venous thrombosis (DVT) can be the source of acute pulmonary emboli or lead to long-term venous problems.

Recommendations

AAA requires ongoing follow-up because of its high mortality rate (78-94%) upon rupture. PVD can require surgical revascularization, angioplasty or amputation. Acute DVT disqualifies the commercial driver until adequately treated. Varicose veins do not medically disqualify the CMV driver.

Recommendations of the Advisory Panel

In the United States, CVD is the fastest growing and most prevalent chronic, progressive condition. Paradoxically, advances in diagnosis and management insure that a larger number of commercial drivers will be able to continue to work even though they have been diagnosed with CVD. The inherent progressive nature of CVD however, will necessitate the ongoing updating of medical guidelines and increasingly active participation and consultation from cardiac specialists.

The Panel provided specific recommendations designed to:

1. Improve database research;
2. Increase support of Medical Examiners;
3. Systematically review guidelines and examinations;
4. Conduct specific research; and
5. Establish a standing medical advisory panel.

Ellison H. Wittels, MD, FACP; Chairman, Cardiovascular Advisory Panel

INTRODUCTION

Background

The growing size of the commercial driver population coupled with the prevalence of cardiovascular disease (CVD) in the United States makes certain that heart-related illness will have an increasingly powerful impact on the health and safety of commercial motor vehicle (CMV) drivers in specific, and the traveling public in general.

In the United States, heart disease ranks first and stroke third as the leading causes of death (1, 2). According to current estimates, one in five persons has some form of CVD, including approximately 50 million with hypertension, more than 3 million who have survived a stroke, and 12.4 million with coronary heart disease (CHD) (3, 4).

There are nearly 9 million U.S. drivers who hold a commercial driver's license (CDL), and another 2 million drivers with non-CDL commercial licenses. Nearly all of these CMV license holders are required to meet federal medical standards as a condition of employment.

The Federal Motor Carrier Safety Administration (FMCSA) is responsible for the establishment and enforcement of the Federal Motor Carrier Safety Regulations (FMCSRs), including the medical qualifications of commercial motor vehicle drivers. The current medical standard covering commercial drivers with CVD has been in effect since 1970, and permits qualification of individuals to operate CMVs if they "have no current clinical diagnosis of myocardial infarction, angina pectoris, coronary insufficiency, thrombosis, or any other cardiovascular disease of a variety known to be accompanied by syncope, dyspnea, collapse, or congestive cardiac failure." The decision whether the nature and severity of a driver's condition will cause sudden incapacitation is on an individual basis and rests with the medical examiner.

FMCSR Guidelines

To assist medical examiners, FMCSA periodically convenes expert medical panels to review its guidelines and recommend qualification criteria, test procedures and decision matrices that reflect current medical knowledge and technology. Medical guidelines help standardize the certification process and decrease the risk of medically unfit commercial drivers receiving certification. FMCSA's goal is *not* to prevent drivers from working, but to help ensure that the roads and highways are safer for the commercial driver and those who share the road with the driver.

In 1995, the agency convened an expert panel to update its qualification criteria for anticoagulation treatment of commercial drivers. The final report was submitted on April 1, 1996.

The last major review of the cardiovascular guidance materials, a two-day conference, was held in October 1986. The final report, "Conference on Cardiac Disorders and Commercial Drivers," was published in December 1987, and is the basis for FMCSA's current guidelines on CVD and commercial drivers (5).

2001 Cardiovascular Medical Advisory Panel and Topics

In October 2001, the FMCSA convened a medical advisory panel to update the cardiovascular guidelines.

Panel Members	Cardiovascular Disease Topic
Dr. Roger Blumenthal and Dr. Joel Braunstein, Johns Hopkins University Medical Center	Ischemic Heart Disease, Hypertension
Dr. Heidi Connolly, Mayo Clinic	Congenital Heart Disease
Dr. Andrew Epstein, University of Alabama at Birmingham	Arrhythmias, Sudden Death, Pacemakers
Dr. Bernard J. Gersh, Mayo Clinic	Valvular Disease, Myocardial Disease
Dr. Ellison H. Wittels, Chairman Concentra Medical Centers	Peripheral Vascular Disease, Transplantation

Ms. Sandra Zywokarte, MPH, Office of Bus and Truck Standards and Operations, Federal Motor Carrier Safety Administration, has served as the Program Technical Representative. Mr. John Sheridan of Cherry Engineering Support Services (CESSI), provided administrative support.

FMCSA Directives to the Cardiovascular Advisory Panel

The panel was asked to conduct a systematic review of the cardiovascular guidelines to ensure that they reflect current medical knowledge and technology and to recommend qualification criteria, test procedures and decisions matrices to assist medical examiners.

The Panel was requested to provide findings and recommendations that:

1. Identify potential risk factors (complications or medical factors) that might be associated with driving impairment;

2. Discuss how identified risk factors may affect driving ability;
3. Identify treatment technology that can be used to control each risk factor or medical condition; and
4. Update the specific recommendations from the 1987 Conference on Cardiac Conditions and Commercial Drivers.

Resources Used in the Development of the New Guidelines

The Panel members used a number of resources. The Medical Review is based on:

1. Current literature on the natural history, symptoms, signs, testing and treatment of cardiovascular disease;
2. A review of the literature on driving and cardiovascular disease;
3. Federal Motor Carrier Safety Regulations, Section 391.41(b)(4) and (6);
4. Federal Aviation Administration Guide for Aviation Medical Examiners;
5. Guidelines from other countries; References (6), (9), (26) and (34);
6. The 1987 Office of Motor Carriers Conference Report on Cardiac Disorders and Commercial Drivers; and
7. Input and synthesis of the Cardiovascular Advisory Panel members.

Process Used by the Panel

The Panel members combined their academic backgrounds with their clinical experience in diagnosing and treating CVD. Each Panel member not only submitted a medical review paper with recommendations for certification, disqualification, testing and re-certification, but also reviewed other Panel members' papers. When necessary, additional questions were posed to Panel members. Conclusions, along with dissents, are noted. The final report was presented to the FMCSA. As part of their commitment, Panel members will be available over the next several years to answer questions that may arise about the guidelines.

Format of Papers

Each of the CVD topics has a medical review and specific recommendations based on the medical review. Cardiovascular pharmacological agents and their effects are reviewed under the appropriate section.

Medical Review
Each medical review is designed to provide the medical examiner information on the anatomy and physiology of cardiovascular diseases. Because most cardiovascular diseases are progressive over time, their natural history has been well documented. In addition, much attention has been paid to the testing required to assess the cardiovascular condition of the driver. It is not likely that medical examiners will conduct the testing

outlined in this section. However, review of the testing requirements provides the examiner the information to assess whether the driver has been adequately evaluated and to classify the severity of the disease as it relates to the driver's general health and ability to be medically certified.

Recommendation Tables
The Recommendation Tables are based on the medical review and are found at the end of each paper. The tables have been written to make the recommendations clear, concise and easier to access. Guidelines that are too complex or too long will not be read (6).

Guidelines Versus Standards

The medical examiner should distinguish between the medical standard (49 CFR 391.41), and the medical guidelines. The standard must be followed. Guidelines are recommendations that the medical examiner should follow. While not law, the guidelines are intended as standards of practice for medical examiners. Although the medical examiner is responsible for determining if the commercial driver is medically qualified under the FMCSRs, these guidelines have been issued by FMCSA and are based on the medical literature. If the medical examiner chooses not to follow the guidelines, the reason(s) for the variation should be documented.

Guideline Limitations

The medical examiner is not expected to act as the commercial driver's primary physician. The guidelines are not intended to decide medical or surgical treatment. However, the Panel did minimize variation from appropriate clinical practice. For example, the Panel did not wish to require extensive testing that would not otherwise be required to assess the driver's medical condition (7).

There are times when the medical assessment and the guidelines may yield different conclusions about the severity of the condition. A driver could have a benign underlying medical problem with an excellent prognosis, but still not be medically qualified as a commercial driver. For example, if a benign supraventricular arrhythmia causes syncope, the person cannot be medically certified until the problem has been corrected (8).

Because atherosclerosis can affect different vascular beds, the presence of clinical disease in one vascular bed may be a clue that there is significant atherosclerosis in other parts of the body. While the CVD topics have been presented separately, several cardiovascular diseases may be present at the same time. While these guidelines are comprehensive, it is not possible to review all combinations of medical diseases (9).

If a driver has more than one cardiac problem (e.g., valvular heart disease plus chronic atrial fibrillation), the criteria for each should be satisfied and the driver then assessed to determine how the conditions occurring together affect certification to drive.

Overview of Medical Illness and Motor Vehicle Crashes

Acute medical illness is responsible for a small percentage of motor vehicle crashes, with estimates ranging from less than 0.1% to 3% (10-12). In the United States, commercial driver illness and blackouts were recorded in 0.3% of crashes (13). While the incidence of crashes is low, they are responsible for significant morbidity and mortality. Most studies have shown that CVD is the major cause of acute medical illness that results in motor vehicle crashes (11,14-17).

Establishing Risk for Commercial Drivers

Risk is an expression of the probability of an event occurring over a certain period of time (18). The level of risk must be considered within the context of the setting and activity in question and what society considers acceptable. Determining "acceptable risk" becomes a matter of public policy. Therefore, the decision to certify or disqualify a commercial driver is both a medical and a societal decision (19).

Common sense and a well-researched literature make it clear that there is no zero risk in certifying commercial drivers, including those in whom a diagnosis of CVD has not yet been made. Focusing more on societies' concerns and risk avoidance makes licensing more restrictive. Focusing more on the driver's right to earn a living in their occupation of choice makes licensing less restrictive. The right of the individual to pursue his/her desired occupation and to earn a living should not be unreasonably denied; however, there are times when the commercial driver with cardiovascular (or other) disease may not be medically safe to drive. Given the complex demands of operating a large truck or bus, coupled with the high fatality risk for occupants of the other vehicle in crashes involving CMVs, a conservative approach is required.

The fundamental question when deciding if a driver should be certified is whether the CMV driver has a cardiovascular disease that so increases his/her risk of sudden death or incapacitation that the driver endangers his/her health and safety and the health and safety of the public sharing the road with them.

Qualifications and Duties of Medical Examiners

For many years in the United States, only Doctors of Medicine and Doctors of Osteopathy were designated to perform medical certification examinations for CMV drivers. In 1992, the FMCSRs were amended to allow physician assistants, advanced practice nurses and Doctors of Chiropractic to perform certification examinations, if their

state license allows them to do so. This has expanded the pool of medical examiners and provided easier access to the medical certification process.

Medical examiners are not required to have any specific training and do not need to demonstrate any special competence to medically certify commercial drivers. The FMCSA does not certify or regulate medical examiners. However, examiners are expected to exercise good medical judgment during the evaluation and may be open to litigation in the case of an undesirable outcome. The medical examiner cannot shed his/her responsibility to evaluate carefully each person on whom they perform a physical examination (20).

The medical examiner must:

1. Have some familiarity with the physical demands and the mental and emotional responsibilities of a CMV driver;
2. Be familiar with the requirements in 49 CFR 391.41 and the medical guidelines; and
3. Record accurately the information required on the examination and certification forms (49 CFR 391.43).

Consideration of Job Demands

The demands on the driver vary greatly with the type of vehicle and the type of driving required. Commercial drivers usually cannot choose their work hours or routes. Overall, CMV drivers have a multitude of job demands. For example, a commercial trucker's duties may include loading and unloading, making multiple stops, driving cross-country or in heavy city traffic, working with load securement devices, or changing tires. A commercial bus driver has responsibilities that are different from the commercial trucker (21).

To improve health and safety, the FMCSA requires not only that CMV drivers meet higher medical standards for driving, but also requires that drivers are medically suitable to perform the (potential) physical demands of commercial driving. The medical examiner must either certify or not certify a driver; the examiner cannot place additional restrictions or accommodations other than those listed on the certification form. In granting medical certification, the medical examiner is certifying that the person is able to perform any job duty required of a commercial driver, not just his/her current CMV job duties.

Medical Evaluation

The physical examination is an essential part of assuring a healthy commercial driver work force (7,22-24). The medical assessment is based on information provided by the driver (History), objective data (Physical), and additional testing requested by the medical examiner. In the vast majority of instances, evidence of CVD is found when the

history is honestly given and carefully reviewed and when the physical examination is done thoroughly (25). However, the Canadian Cardiovascular Society warned, "symptoms may change when some privilege or economic benefit is involved" (26). The demands of driving commercial vehicles reflect *physical, psychological and environmental factors.* The medical examiner needs to consider each of these three factors (27-30).

Consideration of Non-Cardiovascular Factors

CVD and its effects on driving cannot be considered in isolation; the effect of heart disease on driving has to be viewed in relation to the general health of the individual. There are also times when other medical conditions may exacerbate a cardiovascular condition. Medical certification to drive depends on a comprehensive medical assessment of overall health and informed medical judgment about the impact of single or multiple conditions on the whole person.

The New DOT Medical Examination Form

The medical form has been revised for the first time since 1970. FMCSA published its final rule (65 FR 59363) for the new examination form on October 5, 2000 (31). Use of the new form has been required since November 2001 (32). The medical examination form is found on FMCSA's web site at www.fmcsa.dot.gov - keyword "Medical Examination Report Form."

Medical History and Physical Examination

Because the form does not provide a complete CVD history, the examiner may supplement the questions on the form when CVD is identified or suspected. The medical form asks specific questions about prior *diagnosed* CVD. The examiner may wish to supplement the questions on the form by asking about CVD *symptoms*, including questions about the presence of chest pain, pressure, or ache—at rest or with exertion—, dyspnea—at rest or with exertion—, and recurrent and/or severe palpitations. Similarly, questions can be asked about the symptoms of claudication, such as buttock, leg, or calf pain with ambulation that resolves with rest. The examiner should distinguish between pre-syncope (dizziness, light-headedness) and true syncope (loss of consciousness).

The examiner may elect to expand the physical examination when cardiovascular disease is present or suspected. Findings of the examination can be recorded for future reference. Findings that require additional testing should be documented.

Required Testing

The only test required is a urinalysis for specific gravity, protein, blood, and sugar.

Additional Tests Available to the Medical Examiner

The examiner may require additional testing if there are concerns about the presence or extent of CVD. The examiner may order and interpret additional tests or may refer the applicant to a specialist or the driver's primary healthcare provider.

An electrocardiogram is not required and should be obtained only if clinically indicated. It is often insufficient in detecting cardiac disease (33).

Interpretation of Test Results

Using only test measurements to define acceptable standards for the driver may be misleading. Estimates of parameters such as aortic valve gradient, left ventricular ejection fraction, or the degree of coronary artery stenosis on angiography are subject to observer error. As a result, a driver could be certified or removed from driving based on a minimal variation in the measurement. Therefore, in addition to individual technical measurements, the medical determination should reflect the clinical judgment of a knowledgeable medical examiner (6).

Consultation

The guidelines recommend caution in driver certification when a cardiovascular disease's clinical course is uncertain or unknown. If the examiner is uncertain about a driver's condition or prognosis, the decision for certification needs to be postponed until the additional necessary information is obtained. When helpful, the Panel recommends that the medical examiner review the decision to certify/disqualify a commercial driver with the treating doctor. A specialist who is treating the driver's CVD should be consulted.

Review of Results with Applicant

Findings that are disqualifying need to be explained to the applicant. Cardiovascular conditions that may not be immediately disqualifying should also be discussed with the applicant. This is particularly important when, if neglected, the condition could eventually interfere with the person's health and ability to drive safely. The driver should be referred to his or her doctor.

Completing the Form

The form should be completely and accurately filled out. It is useful to elaborate on a "Yes" answer by adding information to the driver's responses. Applicable restrictions need to be identified. The final decision should reflect a combination of clinical judgment and test results (6).

Waiting Period

The driver can be temporarily disqualified. The "waiting period" is the time interval during which commercial driving is not allowed. If more than one waiting period applies, the longer one should be used, except where stated otherwise. For example, for a commercial driver treated with coronary angioplasty (waiting period one week) following an acute myocardial infarction (waiting period two months), the waiting period should be two months. Recurrence of the disqualifying condition resets the waiting period.

Frequency of Re-certification Examinations

Under the FMCSRs, the Medical Examiner's Certificate is not valid for more than two years. The driver can be certified for three months, six months, one year, or another length of time decided on by the examiner, not to exceed two years.

A commercial driver with a clinical diagnosis of CVD should be re-certified at least annually. A commercial driver who has multiple risk factors for CHD and is 45 years of age or older should be re-certified annually.

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Ischemic Heart Disease

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ISCHEMIC HEART DISEASE

Background

The effect of Coronary Heart Disease (CHD) on the commercial motor vehicle driver's health and safety is significant now and will increase in the future. There are two major reasons for the significance of CHD:

1. The prevalence of CHD in the population. The prevalence should increase as the population ages and as diagnosis and therapy continue to improve; and
2. The direct relationship of CHD to sudden death in drivers.

Prevalence of Coronary Heart Disease

The major clinical manifestations of coronary heart disease (CHD) are acute myocardial infarction, angina pectoris (either stable or unstable), sudden death, and congestive heart failure (1). The National Health and Nutrition Examination Survey (NHANES III, 1988 to 1994) reported that 11.8% of those over 40 years of age have CHD. The age-adjusted prevalence of angina pectoris was 5.8%; the self-reported prevalence of myocardial infarction was 6.7%, and 3% had electrocardiographically defined myocardial infarction (2). This year, an additional 1.1 million individuals will have a coronary event, including fatal and nonfatal myocardial infarctions (3).

CHD and Motor Vehicle Crashes

While arrhythmia is the most likely cause of sudden driver incapacity, CHD is the most likely underlying etiology, ranging from 21% to 100% of the cases in reported studies (4, 5, 6). Persons who experience sudden death frequently have cardiomegaly (83% prevalence based on one necropsy study) (7), previously healed MIs (prevalence between 56% and 67%) (7-10), and extensive luminal narrowing of at least one coronary artery (prevalence range between 66% and 96%) (7,11).

In several investigations into the cause of crashes, it was difficult to define the role of CHD. Of 2,000 road crashes reported by police to the United Kingdom Driver Vehicle Licensing Center, epilepsy was the most common cause of incapacity, heart disease accounted for 8% of the incapacity caused accidents, and unexplained blackouts accounted for another 23% (6). A review of published and unpublished information on 9,390 serious or minor injury crashes reported to police in one county of England in 1963 and 1964 identified 15 (1.5/1,000) crashes caused by acute illness at the wheel. Among the 15 crashes, three were caused by heart disease and six had loss of consciousness of unknown origin, which could have been from heart problems (12).

Sudden Death and the Incidence of Crashes

Some studies reported the “surprising finding" that a significant number of drivers who died suddenly did not cause serious injury to other members of the community (9). Roberts reported that most drivers stopped their vehicles without injury to themselves or others. In their review of the literature, 26% to 67% of cases of sudden death involved collisions (7). Other studies have reported similar findings (10,13-16).

In Trapnell and Groff’s study of 50 cases of proven MI, 32 men drove tanker fuel trucks, 7 drove 2.5-ton delivery trucks, and 11 drove panel or pickup trucks incidental to their jobs. Although 12 of the initial attacks occurred during the scheduled work period, no vehicle crashes resulted. Thirty-five of the 50 returned to driving. Five of the 35 probably had second infarctions. Two of the five were found at the side of the road in their trucks, two died at service stations, and one, who had stopped driving three years before, died while working as a guard (17).

Another study analyzed 1,348 cases of sudden death due to CHD between 1956 and 1962 in Dade County, Florida. One hundred twenty two people had occupations potentially hazardous to the public, with 28 involved in their work at the time of death, including 15 who were driving trucks. None of the 15 cases had a serious crash (18).

Reasons for the relatively low incidence of crashes resulting from sudden death of the drivers include:

1. Drivers with significant CVD are disqualified from driving at their commercial driver medical examination;
2. Drivers at highest risk of cardiovascular collapse, for example in the first month or so following myocardial infarction, are not driving; and
3. Many persons do not feel well in the six to 24 hours preceding their infarction and cancel their plans to drive.

However, the major reason for the low incidence of crashes appears to be the length of time between the onset of the cardiovascular event and the driver's incapacitation.

Sudden Death and Instantaneous Death

The Framingham Study defined “sudden death” as going from a usual state of health to death within one hour (1). Among those dying within one hour, CHD is most always the cause. As the time between the onset of illness and death increases, the percentage dying from CHD decreases and the causes are more heterogeneous (5,19).

Sudden death as defined in the epidemiology literature is not instantaneous, where death is defined as occurring within seconds to several minutes. Instantaneous cardiac death is caused chiefly by ventricular fibrillation and is not often due to an orderly progression of

CHD, but occurs because of an electrical accident. It has been estimated that less than 20% of sudden cardiac deaths are due to recent infarction (20).

The electrical abnormalities leading to sudden cardiac dysfunction usually take time to develop. During that time the cerebral circulation is partially maintained and the person may become aware that something is wrong and have time to react.

Norman has contended that the typical road crash occurs in five seconds or less. The time involved for a driver to slow down or stop the vehicle comprises not only stopping the vehicle, but also recognition of illness, which may occupy five to 10 seconds (21).

Therefore, in most instances, drivers will have time to pull to the side of the road (22,23). However, severe injury or death may result if no warning occurs or a warning symptom is ignored or misinterpreted. In Parsons' study, drivers who fell asleep often did so without warning; they represented 27% of the entire sample, but 83% of the deaths attributable to trauma (24).

Need for Early Identification of Coronary Heart Disease

The estimated frequencies of the initial presentations of CHD are approximately 50% MI, 30% angina, and 20% sudden death (25). Due to the catastrophic nature of over two-thirds of these initial cases, there is great appeal in detecting subclinical atherosclerosis and in identifying individuals at high-risk for CHD.

Sudden cardiac dysfunction is particularly relevant to professions (e.g., aviation pilots, merchant marines, and commercial drivers) with safety-sensitive positions. In these jobs, policies are expected to protect against on-the-job sudden incapacitation and harm to the public.

Sudden Death as the First Sign of Coronary Disease

Unfortunately, in a significant number of sudden deaths, the underlying heart disease had not been previously recognized. In the Framingham study, 131 men and 49 women died suddenly and unexpectedly. Sixty percent had no prior indication of CHD (5). In Wikland's study of medically unattended sudden death, a history of previous myocardial infarction was present in 20% of males and 12% of females. Angina pectoris had been diagnosed in 32% of males and 30% of females. An additional 11% of males and 13% of females had symptoms of ischemic heart disease, but their physician had not yet confirmed the diagnosis. Importantly, 24% of males and 20% of the females had no history, signs or symptoms of ischemic heart disease, or coronary risk factors (26).

Herner's study concluded that of the 41 drivers who probably caused crashes from sudden incapacitation, only 19 could have been detected by pre-crash medical examination (27). Similarly, Ostrom and Eriksson found that only about 20% of drivers

with sudden death at the wheel had prior symptoms of heart disease (10). In Christian's 10-year prospective study on the incidence and implications of sudden death in road users, 56 died from CVD, only half of whom were known to have CVD. None of seven who died from a ruptured abdominal aortic aneurysm was aware of its presence (28). Out of a total of 2,000 road accidents from all causes reported by police to the United Kingdom Driver Vehicle Licensing Center, 8% had heart disease. In this group, 76% of accidents occurred in those who were already diagnosed with heart disease and 24% occurred in drivers with previously unrecognized heart disease (6).

Strategies to Detect Coronary Heart Disease

Over the last few years, several strategies have emerged in an attempt to identify those with early or significant CHD. The two strategies to decrease the mortality and morbidity of CHD are:

1. Risk factor identification and treatment; and
2. Early identification and treatment of CHD (29).

Strategy 1: Risk Factor Identification and Treatment

Extensive epidemiologic and observational research over the last several decades has led to the establishment of factors that play an important role in the pathogenesis and/or risk of developing CHD. The presence of these risk factors does not mean that the person will develop CHD. Similarly, the absence of risk factors does not mean that a person will not have a future CHD event. Sex-specific validated risk scores, such as those from Framingham, offer added value for predicting a driver's near term CHD event risk and can be used for risk stratification and as an indication for aggressive risk factor management or additional testing (30-32)

Risk Factors - Modifiable
Hypertension -- Systolic > 140 mmHg or diastolic > 90 mmHg)
Tobacco smoking -- Current or recent past (< 1 year)
Hypercholesterolemia
Low HDL
Diabetes mellitus
Overweight or obesity
Physical inactivity
Nutritional habits (contributing but not definite CHD risk factor)

Risk Factors - Non-modifiable
Family history of premature heart disease
Increasing age
Sex -- men and postmenopausal women

The risk factor set points vary among organizations. The presence of the major, non-modifiable risk factors emphasizes the need for more aggressive management of modifiable risk factors.

Tobacco Smoking

Cigarette smoking contributes to an estimated one-third of all CHD-related deaths in the United States on a per annum basis, with its impact being strongly dose-dependent (33,34). Active cigarette smoking is the greatest risk factor for the development of sudden cardiac death and portends at least a two-fold elevated risk for both CHD and thrombotic stroke. Among its many adverse effects, cigarette smoking damages vascular endothelium, heightens platelet aggregation and reactivity, and accelerates coronary plaque development and subsequent rupture.

There is a great need to encourage long-term smoking cessation, since individuals reduce their CVD risk to that of a nonsmoker within three years after quitting (35-38). Despite vigorous public campaigns to curtail its prevalence, an estimated 25% to 30% of all Americans continue to smoke, with the higher rates among blue-collar workers.

Hypercholesterolemia

There is sufficient evidence to show that elevated cholesterol plays a vital role in the pathogenesis of CVD through the promotion of endothelial cell dysfunction, atherosclerotic plaque formation, and plaque rupture. The National Cholesterol Education Program classifies a total cholesterol (TC) value below 200 mg/dL as desirable and a value between 200-239 mg/dL as borderline-high. Approximately 40 million people in the United States satisfy the criteria of high cholesterol as defined by a TC level $\geq$ 240 mg/dL (3). A high-density lipoprotein cholesterol (HDL-C) level < 40 mg/dL is unfavorably low while a level > 60 mg/dL is cardioprotective and regarded as a negative risk factor. A low-density lipoprotein cholesterol (LDL-C) < 130 mg/dL is near optimal and a triglyceride (TG) < 150 mg/dL is normal (39).

The Multiple Risk Factor Intervention Trial demonstrated that CHD rates progressively declined with lower TC levels, down to a level of 150 mg/dL (3.9 mmol/L) (corresponding to an LDL-C level of about 100 mg/dL), where subsequent CHD events become rare (40). The Framingham Study established that every 4 mg/dL (0.1 mmol/L) decrease in HDL-C level is associated with an approximate 10% increase in CHD risk (41). An individual's lipids are most effectively interpreted within the context of the person's age and other risk factors.

Diabetes Mellitus

Diabetes mellitus is defined as a fasting plasma glucose of 126 mg/dL or higher, measured on two separate occasions. Using this definition, diabetes mellitus now afflicts over 16 million Americans, and its incidence is increasing (3). Of these 16 million, at least 5 million are believed to be unaware of their diagnosis. Type 2 diabetes closely associates with other cardiovascular risk factors including dyslipidemia (usually high triglycerides and low HDL-C levels), hypertension, and obesity. Impaired glucose tolerance (defined by a fasting plasma glucose between 110 mg/dL and 126 mg/dL) also carries a heightened risk for future development of CVD as well as type 2 diabetes (42). Diabetes is regarded as a CHD-risk equivalent (e.g., has the same cardiovascular prognosis as an individual with established coronary disease) (39).

There is a need for early disease recognition, glycemic control, and comprehensive risk factor management because the diabetic patient has a 2 to 4-fold increased risk of cardiovascular events. The increased risk of "silent angina" in individuals with diabetes also increases the need for early disease recognition and ongoing follow-up.

Overweight/Obesity and Physical Inactivity

Either body mass index (weight in kg divided by height in meters squared) or waist (truncal) circumference can be used to define excess weight that carries an elevated risk of CVD. A body mass index of 25-29.9 kg/m^2 defines overweight; greater than 30 kg/m^2 defines obesity. A waistline circumference exceeding 35 inches (88 cm) for women and 40 inches (102 cm) for men is considered high risk and one of the defining features of the metabolic syndrome (39). Approximately one in two Americans are either overweight or obese.

There is some controversy as to whether excess body fat is a direct, independent predictor for CVD, since it strongly associates with other known risk factors such as hyperlipidemia, hypertension, and impaired glucose metabolism. The benefits of obesity treatment, however, are incontrovertible. Reductions in body weight by only 5% to 10% have been shown to improve lipid profiles, blood pressure, glycemic control, and CVD risk (43).

Lack of physical activity among overweight individuals compounds cardiovascular risk (44). In a nationally conducted telephone survey of 184,450 American adults, the Behavioral Risk Factor Surveillance System reported that more than 55% of respondents were either completely physically inactive or insufficiently regularly active (45). Physical activity is important for both primary and secondary prevention of CHD.

Age and Coronary Heart Disease

Age is an important non-modifiable risk factor. In a 10-year study, Bruce and Fisher reported on 8,389 men divided into four groups: Group 1 Healthy men; Group 2 Atypical chest pain, not angina; Group 3 Hypertensive; and Group 4 CHD. In Group 1, with advancing age, the prevalence of healthy people progressively declined to 12.1% for men over 64 years, the oldest age group. The prevalence of hypertension in Group 3 increased with age from 11% among those less than 38 years of age to approximately 25%. The prevalence of CHD increased nearly tenfold from 6.2% in the youngest men to 61.4% in the oldest group. The annual incidence of sudden cardiac incapacitation taken together among all groups was 0.084% per year in men less than 38 years old and progressively increased to 2.72% per year for men older than 64 years. The effect of age alone was significant for men older than 46 years (46).

In a study of sudden death conducted by Myerburg and Davis, CHD was the predominant cause of incapacitation, with the incidence increasing with advancing age.
In their study, 7.5% of sudden deaths were potentially hazardous to others. Two-thirds of the workers were 50 to 65 years old. The majority were employed in public ground transportation and could have created a public hazard if sudden death had occurred while they were working (18). Age is a risk factor after percutaneous coronary intervention (PCI)(47). The risk incurred by a post-coronary patient was less than the risk associated with 10 to 15 years of aging in a normal adult (21).

The aging of the population is also reflected in commercial drivers (48). While it is appropriate to recognize that age is a significant risk factor for the development of CVD and for the outcome of vascularization procedures, reliance only on chronological age to decide if a person is qualified to drive is arbitrary and unfair to low-risk older workers.

Hypertension (See Hypertension Section)

Commercial Drivers and Cardiovascular Disease

In a 1994 review of the literature, 28 of 32 reports demonstrated that CMV drivers had an increased risk of hypertension, ventricular arrhythmias, myocardial infarction, and other ischemic heart diseases when compared to their referents (49). Many of these studies noted the close relation between years worked as a driver and the young age at which these events occurred. This risk not only begins at an unusually young age, but also persists despite a significant "healthy worker effect" that selects against drivers with cardiovascular disorders at the time of hire or medical follow-up (50).

Not all studies have found a relationship between CVD and commercial driving. Two recent studies of tanker drivers in the petroleum industry demonstrated that drivers in that industry had low mortality rates from heart disease (51).

Cardiac Risk Factors Among Commercial Drivers

A potential link to the increased prevalence of CVD among commercial drivers is their increased prevalence of cardiovascular risk factors relative to other occupations (52). Commercial drivers have an especially high smoking prevalence (53-56), frequent hyperlipidemia (53-58), body mass indices of overweight or obesity (53,56-58), diabetes (56), and hypertension (59-62).

Commercial Driver Specific Risk Factors

Factors predisposing to the increased risk of CVD among commercial motor vehicle drivers are likely to include more causes than the standard cardiac risk factors.

Commercial driving demands full attention to the environment for prolonged periods of time. Momentary lapses in attention or wrong decisions can have serious repercussions to the driver, the driver's passengers, other drivers sharing the road at the time, and nearby property. Performance of vigilant tasks in order to avoid adverse consequences has been associated with sudden cardiac death in experimental animals (52,63), and hypertension and CHD in humans (52,64-66).

Commercial drivers face additional demands, including the need to adhere to tight schedules (54,67), deal with traffic congestion (68), an increased self-perception of responsibility for others (64), low decision latitude, and poor social support (57), and the need to maintain courtesy despite occasionally belligerent passengers (67,69). The increase in neurosympathetic and adrenocortical catecholamine and cortisol release that has been observed in response to one or more of these provocative stressors provide a mechanism whereby arterial tone, myocardial excitability and contractility, and thrombogenic propensity are increased (70,71).

Finally, environmental exposures such as excessive noise, temperature extremes, air pollution, whole body vibration, carbon monoxide, polyaromatic hydrocarbons, lead, and oncoming glare routinely encountered on the job by commercial drivers may be detrimental to the cardiovascular system (52,65,72-74). Prolonged sedentary habits, infrequent structured physical activity and erratic shift-work hours, particularly during the nighttime, portend additional cardiac risk.

Examiner Access to Risk Factor Data

The Panel is aware that the new medical form does not provide a complete review of risk factors.

Risk Factors - Identified	*Risk Factors - Not – Identified*
Increasing age (>60 years old)	Tobacco smoking
Hypertension	Hypercholesterolemia
Sex (men)	Sex (postmenopausal women)
Diabetes mellitus	Premature CHD in first-degree relative
(Male <55; Female <65)	
Obesity or overweight	Physical inactivity
	Low HDL

The examiner can obtain more information about the presence of risk factors by additional questioning of the driver-applicant, drawing additional blood tests, or contacting the driver's physician.

Driving and Electrocardiogram Changes

In 1968, Bellet reported a study of 66 previously diagnosed CHD patients and 65 normal subjects. With driving, 17% of the CHD patients had ECG changes suggesting myocardial ischemia (75). Lauers et al. studied 13 patients with a prior history of a myocardial infarction and normal electrocardiograms. With automobile driving, four subjects had ST-T changes of at least 1 mm and/or extrasystoles. These changes were also detected with cycloergometric (bike) exercise, except for one person with arrhythmias that were not reproduced. However, more subjects had ST-T changes on the bike test. This study linked physical exertion as well as mental stress from driving an automobile to potential myocardial ischemia (76).

Simonson et al. reviewed the literature on electrocardiographic changes during driving. Significant ST-T depression and T wave changes were reported in healthy drivers, but were more marked in drivers with a history of CHD. He concluded that the changes were not due to myocardial ischemia (although the oxygen consumption was never systematically measured) but were more likely caused by emotional stress mediated through the autonomic nervous system and the adrenomedullary hormonal system (77).

The Risk of Physical Exertion in the Presence of Coronary Heart Disease

The least physically demanding part of the job for a CMV driver may be the actual driving (23). Drivers may have to exert strenuously when changing a tire on the vehicle, securing the load, or loading and unloading cargo. Van Der Beek noted that lorry drivers

spent the majority of their work time driving, but the highest heart rates occurred during loading and unloading activities (78).

Sudden cardiac arrest may occur during or after vigorous physical exertion (79). In the Seattle Heart Watch Program, 11% of sudden deaths occurred in people during or immediately after exercise (80). In Friedman's study of 59 cases of instantaneous death, defined as occurring less than 30 seconds after onset of illness, the individuals rarely experienced acute symptoms or exhibited acute signs before death. More than 50% died immediately after severe exercise to which they had been accustomed. Death appeared to result from an arrhythmia and the hearts rarely showed acute lesions, but frequently exhibited one or two old occluded arteries or an occluded left anterior descending artery (81).

Under resting conditions, cardiac risk is a function of age; with exercise, the risk is enhanced, especially for those 40 to 49 years of age. Indicators of increased risk from exercise/exertion include (82):

1. The intensity and duration of exertion are more than the person has recently experienced;
2. The person persists in exerting himself/herself in spite of warning symptoms;
3. The person is under time, business or social pressure;
4. The activity involves heavy lifting or other isometric work; and
5. The person has an infection or does not feel well.

The Relationship between Risk Factors and Medical Certification of the Driver

The decision to medically disqualify a commercial driver should *not* depend solely on the detection of multiple risk factors. Disqualification requires that the commercial driver has a higher than acceptable likelihood of acute incapacitation from a cardiac event and is therefore an increased risk to himself/herself and others while driving.

Drivers 40 years of age and older with a Framingham CHD risk for a nonfatal myocardial infarction or CHD death of 20% over 10 years, diabetes mellitus, or peripheral vascular disease (CHD-risk equivalents) (39) could be subject to the same medical qualifying regulations as drivers with known CHD (see below).

Strategy 2: Early Identification and Treatment of CHD

An exercise treadmill stress test (ETT) provides quantitative information on a person's aerobic capacity, identifies ischemic changes on the electrocardiogram, elicits symptoms during exercise, and monitors blood pressure and heart rhythm changes. The ETT's most important prognostic variables for determining the driver's ability to work at a specified level of exertion with acceptable cardiac risk are maximal exercise capacity, increase in

systolic blood pressure, and ST segment displacement. Although uncommon, exertional hypotension and ventricular tachycardia identify a high-risk subgroup of patients (83).

There is some utility for using exercise testing to screen for CHD among a select, high-risk population of asymptomatic individuals. In one study, 22% of 170 healthy, obese middle-aged men had an ischemic response on treadmill testing. The seven-year follow-up demonstrated cardiac endpoints (sudden death, acute myocardial infarction, need for coronary angioplasty or coronary artery bypass grafting, and angina) in 46% of those with initial ischemic exercise tests compared with 11% with normal results (84). In the Seattle Heart Watch Study, men with $\geq$ CHD risk factor (positive family history of premature CHD, hypertension, elevated cholesterol, or smoking) and $\geq$ 2 abnormalities on treadmill testing (exercise duration < 6 minutes using the Bruce protocol, ST depression > 1 mm, chest pain, or achieving < 90% maximal predicted heart rate) had a nearly 30-fold increase in 5-year event risk. The annual CHD case fatality rate increased from 0.1% to 9.6%. Six-year survival rates fell from 97.8% $\pm$ 0.4 to 75.7% $\pm$ 7.1 (85).

Limitations of the Exercise Tolerance Test

Because of its less than desired specificity and sensitivity, the exercise test is not effective at distinguishing between those with CHD and those without CHD (82). The ability of the ETT to predict clinical CHD is highly dependent on the prevalence of the disease in the population tested and the criteria used to define a positive test. Among healthy, asymptomatic populations with a low prevalence of CHD, the positive predictive value of ETT is poor and the false-positive rate is high (86).

When Froelicher included angina as an endpoint for 5,526 individuals followed-up of for 3 to 13 years, the ETT's average sensitivity was 50%; the positive predictive value 26%; and the risk ratio was 9. Because angina is regarded as a "soft" endpoint, many studies have used only sudden death and acute myocardial infarction as endpoints. When angina was omitted as an endpoint, the sensitivity decreased to 27% and the positive predictive value decreased to 6% over the 6 to 8-year follow-up of 12,212 people (86,87).

The Exercise Tolerance Test in Asymptomatic Persons with No Risk Factors

After reviewing available information, the United States Preventive Services Task Force concluded that there was insufficient evidence to recommend for or against screening middle-aged and older men and women for asymptomatic CHD. Performing an ETT on asymptomatic persons with multiple risk factors was recommended only when the results would alter treatment decisions (29).

Exercise Tolerance Test in Commercial Drivers With Risk Factors and No Known CHD

Using the ETT to assess commercial drivers with risk factors but no symptoms or signs of CHD is controversial. Performing an ETT on commercial drivers could be justified by the potential risk if the commercial driver were to become suddenly incapacitated while driving. The United States Preventive Services Task Force concluded that screening asymptomatic individuals in certain occupations, such as pilots or commercial drivers, could be recommended on other grounds, including possible benefit to public safety (29).

The incidence of sudden death is directly related to age, antecedent high blood pressure, left ventricular enlargement on the electrocardiogram, heavy cigarette use, and obesity. However, these same risk factors were found in persons whose coronary deaths were not sudden (88). There is not adequate documentation that the ETT can specifically identify individuals at increased risk for sudden incapacitation (89).

In 1997, The American College of Cardiology and American Heart Association Guidelines for Exercise Testing noted that ETT is periodically done in groups in whom a sudden cardiac event could endanger public safety. However, the guidelines state that there "are insufficient data to justify this approach" and that the "usefulness/efficacy is less well established by evidence/opinion" (89). The medical examiner is asked to decide if the driver is medically safe to drive, not to determine if the driver is free from CHD or has risk factors for heart disease. Widespread screening for CHD among asymptomatic drivers seems impractical at this time (90).

Additional issues would need to be clarified before an ETT for commercial drivers with risk factors is required. The Panel would need to define the criteria for ETT pass/fail, decide who would require additional testing, and quantify the value of the program in promoting safety and health. It would also be necessary to determine how the results would affect job, insurability, and the direct and indirect costs to the individual and company. There would need to be a determination of the sensitivity and specificity of the ETT in this specific population. The Panel is not aware of any studies that have addressed either the efficacy or cost-effectiveness of performing an ETT on commercial motor vehicle drivers with coronary risk factors and no clinical symptoms or signs of CHD. A cost effectiveness model would need to be developed.

Another approach that the Panel discussed was testing drivers over 60 years of age and with multiple risk factors. This approach is not based solely on age, decreases the number of CMV drivers tested, and focuses on a high-risk population.

Currently, there are approximately 9 million commercial drivers and thousands of medical examiners. The Panel concluded that it is not realistic to expect the medical examiner to provide risk factor identification, risk stratification, testing and follow-up for asymptomatic drivers.

With these uncertainties, the Panel is not able to recommend for or against an ETT for asymptomatic drivers with risk factors for CHD. The Panel does recommend that the issue be intensely reviewed by the FMCSA in collaboration with interested medical organizations. However, an examiner can still require a commercial driver to have an ETT.

Additional Tests to Detect CHD

Radionuclide or echocardiographic myocardial imaging studies and/or angiography should be reserved for specific clinical indications. Stress imaging studies have superior sensitivity and specificity compared to the standard ETT and can be used in the presence of an abnormal resting electrocardiogram or non-diagnostic standard ETT. The cost of such testing is substantial and might not always be covered by insurance.

The use of other screening modalities is presently limited by inconsistency in their ability to predict CVD events (e.g., death or acute MI). Still, they may be useful for global risk assessment, since many individuals may have a high burden of atherosclerotic plaque in their vascular tree and display no hemodynamically significant obstructive disease (91,92). There has been recent support for using a high coronary calcification score (e.g., one exceeding the seventy-fifth percentile standardized for age and gender) or a low resting ABPI to replace age as a surrogate for atherosclerotic plaque burden and a risk factor in global risk assessment models (93). The data indicates that a zero calcium score by EBCT is associated with an excellent prognosis in patients with equivocal exercise stress tests (94) or atypical chest pain (95).

Certification of Drivers with Clinical Coronary Heart Disease

In 1963, Trapnell and Groff wrote that "...prohibiting the operation of commercial or passenger transport vehicles by any driver who has had a myocardial infarction is certainly a sound policy in general" (17). In the same year, Levy and his associates wrote in the Journal of the American Medical Association that, in their practice, London Transporters removes any bus driver in passenger service who has had episodes of angina or coronary insufficiency or frank infarction (15).

Advances in the treatment of occlusive coronary disease fostered increased optimism about the ability of an individual with CHD to return to the workforce, including returning to work as a commercial vehicle driver. By 1984, Kerwin wrote in the Canadian Medical Association Journal that a "history of heart disease should not necessarily prevent people from holding a commercial drivers license" (22).

Although the medical and surgical treatment of ischemic heart disease may lead to alleviation of symptoms and improve life expectancy, coronary arteriosclerosis tends to be progressive and the risk of heart attack and sudden death is greater than in healthy populations (96). Men with CHD have a short-term risk of sudden cardiac death that is

increased 5.3 fold, and a long-term risk increased 3.3 fold (97). Whether it is appropriate for asymptomatic individuals to return to occupations where sudden incapacitation or death can pose a risk to the public is often difficult to determine and usually requires additional evaluation (83).

Risk Factors in Established Coronary Heart Disease

With established CHD, predominant predictors of health include left ventricular function or damage, the severity of the coronary artery disease, coronary plaque event, electrical stability, and general health. Other prognostic indicators in patients with established CHD include increasing age, gender, symptoms of angina pectoris, and evidence of associated vascular disease (89).

The Exercise Tolerance Test and Work Capacity in Drivers with Coronary Heart Disease

Using the ETT to determine a person's ability to work at a specified level of exertion has proven useful. While driving requires isometric exercise, studies on the energy demands of a commercial driver would be difficult to do (98). There is, however, an extensive literature on the energy requirements of many physical tasks. Sedentary activity requires less than 2 METS. These activities include sitting, slow walking, and lifting light objects of no more than 10 pounds. Light work requires 2 to 4 METS, and includes carrying lightweight objects of 20 pounds; medium work requires 4 to 6 METS and includes carrying moderate weight objects up to 50 pounds; and heavy and very heavy work requires greater than 6 METS and includes carrying heavy objects and climbing stairs rapidly (99).

Because the commercial license does not provide the opportunity for the examiner to restrict work activity, the commercial driver must be able to perform heavy and very heavy work in order to be certified. Completion of Stage II (>6 METS) of the standard Bruce protocol is sufficient to demonstrate a driver's capacity to perform job-related tasks. The Panel recognizes that workload capacity is an important element of the certification process. In both persons with a history of myocardial infarction and healthy persons, the ability to increase exercise capacity (measured by increased METS on the exercise test) decreases CVD and all cause mortality risk (100).

The European Task Force on Driving and Heart Disease favored the exercise time as the major determinant of adequate cardiac function. The Task Force recommended that ST segment depression alone should not be relied upon because of the incidence of false positive results and the difficulties in interpretation of ST-T changes in certain clinical situations (6). Bruce and Fisher studied 2,373 men with clinical CHD who had undergone exercise evaluation and follow-up for an average of 61 months. The lowest risk group completed Stage III of the Bruce protocol, attained at least 85% of age-predicted maximal heart rate, and had less than 1 mm ST depression (46).

The Coronary Artery Surgery Study found that both the ST segment response and duration of exercise are important indicators of cardiac fitness. The highest risk group with an annual mortality of 5% or more could only exercise to stage 1 of the Bruce protocol and had at least 1mm ST segment depression. The lowest risk group could exercise into Stage III of the Bruce protocol and had a 1% or less annual mortality (101). The Canadian Cardiovascular Society defined Class 1 as a person with clinical or objective diagnostic evidence of heart disease without functional limitation and a working capacity of at least 7 METS (102).

Commercial Driver Certification After Myocardial Infarction

Current opinion among clinicians caring for post-myocardial infarction (MI) patients is that they can safely return to any occupational task, provided that there is no exercise-induced myocardial ischemia or left ventricular dysfunction (103). However, return to the workforce after an acute MI depends on a number of complex factors, including the individual's pre-MI working status, age, psychosocial stressors and mood, extent and location of myocardial necrosis, functional (exercise) limitation, occupation, and degree of manual labor required on the job (104). Optimizing the commercial driver's ability to return to work requires effective peri-MI management, aggressive secondary prevention therapy, and rehabilitation to reduce the risk of future major cardiac events.

The first six to 18 months is the period of highest risk for cardiac death following an initial MI (97). Mortality rates of 10% to 12% have been reported within the first three weeks and of 8% to 10% over the remainder of the year (105-107). Mortality rates level off at approximately 4% after the first year (108). Based on observations of the Framingham Heart Study, approximately one-half of these deaths occur suddenly. Individuals with left ventricular dysfunction (ejection fraction <40%), diabetes, excessive age, and/or female may have a substantially higher risk. Individuals with MIs that are uncomplicated or limited in severity can have substantially lower risks.

In general the period of time off work required for a person following a major cardiovascular event has become shorter due to the advent of more effective therapies and rehabilitation. Careful post-MI risk stratification provides a measure of the driver's short-term and long-term prognosis that can be useful in determining re-certification. Given that the first few post-infarction months pose the greatest risk of dying and that the majority of these deaths are sudden, there is sufficient reason for disqualifying a commercial driver from operating a commercial vehicle for at least the first two months after his/her MI.

Commercial drivers may be (re) certified two months after their MI. Certification requires that the major predictors of risk for future coronary events are evaluated before return to work. The driver should be able to achieve a workload consonant with the intensity of work required (> 6 METS), have no ischemic changes on the exercise electrocardiogram or no ischemic segments during myocardial imaging (if performed) (109).

A positive ETT after infarction implies that the risk of recurrence is increased by a factor of 2.29 relative to those with a negative test. Nevertheless, a positive test result is more often wrong than right. On the other hand, a negative stress test is correct in almost 90% of patients. The risk with a negative test is only 75% of the risk for an average member of the post-coronary population (21).

The Panel recommends the following when certifying a driver at least two months after an MI:

1. Examination and approval by a cardiologist for fitness to drive;
2. Annual qualifying examination;
3. Asymptomatic at examination;
4. Echocardiographic assessment obtained prior to work resumption (an in-hospital post- MI echocardiogram is sufficient) demonstrating an ejection fraction $\geq$40%;
5. Exercise tolerance test four to six weeks post MI and repeated as clinically indicated, but at least every two years. The driver should exercise to a workload capacity >6 METS (through Bruce Stage II or equivalent), attain a heart rate $\geq$85% of predicted maximum (unless on beta blockers), a rise in SBP $\geq$20 mmHg without angina, and have no significant ST segment depression; and
6. Tolerance to anti-angina and antihypertensive medications and no orthostatic symptoms.

Certification of Commercial Drivers with Stable Angina Pectoris

Angina Pectoris is caused by an imbalance between oxygen supply and demand to functional myocardium. This imbalance is typically due to a significant luminal narrowing in one or more coronary arteries. Significant narrowing is usually defined angiographically by a $\geq$70% diameter stenosis of at least one of the major epicardial coronary arteries or a 50% stenosis of the left main coronary artery. Coronary lesions with a lesser degree of stenosis have the capacity to cause angina, especially if there is disease in the microcirculation or impaired coronary flow reserve; however, their prognostic significance is markedly diminished (110). The severity of stenosis generally dictates the threshold for the development of anginal symptoms. Clinical history, stress testing with or without myocardial imaging, and catheterization are the best tools for assessing the importance of such stenoses.

The overall mortality of patients with documented CHD is approximately 2% to 4% per year. The annual mortality rate falls to 1 to 2% or less among individuals with stable angina pectoris, no history of MI, no history of unstable angina, no left ventricular dysfunction, and no arrhythmias. In the medically treated group of the Coronary Artery

Surgery Study (CASS), overall mortality was 1.4% a year and only 1% a year in patients with ejection fractions exceeding 50% (111). The annual rate for an acute MI is estimated to be 3% to 3.5% among patients with stable angina (112,113).

Stable angina brought on by exertion can be treated with rest, pharmacologic management, or interventional or surgical-based revascularization. Aggressive risk factor modification targeting dyslipidemia, hypertension, smoking, and hyperglycemia may lessen angina symptoms through improvement in either plaque composition or endothelial dysfunction. Beta-blockers reduce myocardial wall tension, lower oxygen consumption, and reduce resting and exercise-induced heart rate increases. Nitrates treat acute angina through reductions in preload and improvements in coronary flow. Infrequently, they may precipitate rapid drops in blood pressure, especially in the setting of dehydration.

Revascularization therapy is best reserved for individuals with debilitating and frequent symptoms, a strongly positive ETT, angina that is increasing in severity and frequency during exercise or at rest, and for those with left ventricular dysfunction.

The medical qualification of a commercial driver with stable angina is predicated on the fulfillment of several requirements:

1. Annual medical qualification examination;
2. Examination and approval of fitness to drive by the driver's physician, generally a cardiologist;
3. No rest angina or change in angina pattern within three months of examination;
4. An ETT at least every two years. The driver should exercise to a workload capacity of >6 METS (through Bruce Stage II or equivalent), attain a heart rate $\geq$85% of predicted maximum (unless on beta blockers), a rise in SBP $\geq$20 mmHg without angina, and have no significant ST segment depression or elevation;
5. Asymptomatic (light-headedness); no resting blood pressure on physical examination <95 mmHg systolic; and no systolic blood pressure decline >20 mmHg upon standing; and
6. General tolerance to cardiovascular related medications. Many patients may be taking anti-angina medications. Hypotension or orthostatic hypotension is an occasional side effect of such medications.

Certification of Commercial Drivers After Percutaneous Coronary Intervention

Percutaneous coronary intervention (PCI) procedures encompass a number of catheter-based techniques aimed at relieving coronary obstructions. These procedures are used for the emergent treatment of acute coronary syndromes (e.g., acute MI or unstable angina) or the relief of coronary artery narrowing sufficient to cause chronic angina. It is estimated that over 500,000 PCI procedures are performed yearly in the United States. Among these procedures, the intracoronary stent, with or without balloon angioplasty,

has emerged as the dominant interventional procedure to relieve obstruction, with both superior short- and long-term outcomes compared to percutaneous angioplasty alone.

The success of PCI is defined by a number of criteria including angiographic achievement of a minimal stenosis diameter of less than 20%, no in-hospital major cardiac complications (e.g., death, MI, emergent coronary artery bypass surgery) or procedurally-related complications, and relief of at least short-term signs and/or symptoms of myocardial ischemia (47).

The near-term success of PCI is between 96% and 99% with the use of new interventional techniques and devices and antithrombotic/antiplatelet agents such as glycoprotein IIb/IIIa, receptor antagonists and clopidogrel. A small threat in the early post-PCI period is caused by acute complications at the vascular access site (usually the groin) including bleeding, occlusion, dissection, pseudo-anuerysm, and arteriovenous fistula.

PCI patients have less immediate morbidity and less need for participation in a formal cardiac rehabilitation program compared to either coronary artery bypass graft (CABG) patients or post-MI patients. Drivers undergoing PCI in the setting of an acute MI or unstable angina warrant restriction from driving duties for the period of time required when these conditions occur without PCI.

The driver's long-term mortality and major event risk after PCI depend on both clinical and angiographic factors. These include CHD severity, complexity, and manner of presentation, presence or absence of risk factors, left ventricular and valvular function, age, and gender (47). Restenosis remains the major limitation of PCI. Long-term angiographic in-stent restenosis rates range between 15% and 32% (114). These re-stenosis rates justify careful post-PCI observation and aggressive risk factor modification for secondary prevention.

Although symptom status is relatively unreliable, typical angina symptoms should prompt evaluation with a stress imaging study or repeat angiography. It has been suggested that 25% of asymptomatic patients display signs of ischemia during exercise testing. The inability to detect ischemic location, however, is a major limitation of ETT. Myocardial imaging using either radionuclide or echocardiogram improves stress test sensitivity and is advocated for testing symptomatic individuals (47).

In the setting of an uncomplicated, elective PCI to treat stable angina, a commercial driver may return to work as soon as one week after the procedure. Criteria for return to work after PCI include:

1. Examination and approval by the treating cardiologist;
2. Asymptomatic;
3. No injury to the vascular access site;
4. ETT three to six months post PCI. In the commercial driver this requires exercising to a workload capacity of at least six METS (through Bruce Stage II or equivalent),

attaining a heart rate ≥85% of predicted maximum (unless on beta blockers), a rise in SBP ≥20 mmHg without angina, and having no significant ST segment depression or elevation. Stress radionuclide or echocardiographic imaging should be performed for symptomatic individuals, individuals with an abnormal resting electrocardiogram, or those drivers who fail to obtain the minimal standards required from the standard ETT;

5. Annual medical qualification examination;
6. Negative ETT at least every other year (criteria above); and
7. Tolerance of all cardiovascular medications. The driver should not experience orthostatic symptoms, including symptomatic light-headedness; a resting SBP <95 mmHg systolic; or a systolic blood pressure decline >20 mmHg upon standing.

Certification of Commercial Drivers After Coronary Artery Bypass Grafting

A number of well-conducted, large randomized trials and registries have identified populations with CHD who benefit from coronary artery bypass graft surgery (CABG) rather than medical therapy or PCI (115). While many of these studies are dated and did not use the highly effective newer pharmaceuticals to treat CHD, CABG has remained the preferred choice of therapy for individuals with multivessel CHD, severe narrowing of the proximal left anterior descending or left main coronary artery, and extensive atherosclerosis in the presence of left ventricular dysfunction or debilitating angina.

Compared to medical therapy, CABG improves quality of life by reducing angina severity and slightly extending length of life. CABG is associated with a higher near-term (peri-operative) MI risk that closely equals the higher long-term MI risk associated with medical therapy. Compared to PCI, CABG achieves improved revascularization rates and fewer repeat procedures for recurrent angina. Coronary bypass patients are at less risk of sudden death than those treated medically. Hammermeister reported that 29% of deaths in bypass patients occurred within 1 hour after the onset of symptoms, compared to 56% of medically treated patients (as cited by Kerwin, 1984) (20).

Compared to PCI patients, CABG patients have longer initial hospitalizations, notable delays in recovery, and a less rapid return to work. Another significant problem associated with CABG is the high long-term re-occlusion rate of the bypass graft. Fitzgibbon et al. reported that vein graft patency was 88% early on, 81% at 1 year, 75% at 5 years, and 50% at ≥15 years (116). Grondin et al. reported an occlusion rate of 12-20% in the first year post-CABG, an annual rate of 2-4% over the next 4-5 years, and approximately a 50% occlusion rate at 10 years (117). Other analyses have reported re-occlusion rates of saphenous vein grafts ranging between 41-50% at 10 years post-CABG (118,119). These re-occlusions not only cause the development of angina, but also threaten survival.

Survival curves demonstrate a steep decline seven years post-CABG (114), the time around which grafts undergo their most rapid progression of atherosclerotic narrowing. The follow-up of more than 22,000 bypass surgery patients reported an annual death rate

of 1.9% to 3.9%. Five and 10-year survival rates were 90% and 66% respectively, among which two-thirds were deemed cardiac (120).

The most reliable predictors of decreased long-term survival after CABG include advanced age, poor left ventricular function, diabetes, multiple diseased vessels, and female gender. Several analyses have suggested that the presence of angina, prior MI, hypertension, renal dysfunction, and clinical congestive heart failure also play a predictive role in survival. In addition to these predictors of survival, bypass graft type (e.g., internal mammary versus saphenous vein) and obesity influence survival and predict future angina, suggesting graft failure and occlusion.

Despite long-term graft patency concerns, most individuals who undergo CABG are able to return to work within months after their operation. Commercial drivers should abstain from work for at least three months after their operation to minimize the risk of improper sternal wound healing from upper body manual labor.

The following criteria apply to re-qualification of the commercial driver who has recently undergone CABG:

1. Qualifying examination at least three months after CABG;
2. Examination and approval by a cardiologist before resuming commercial driving;
3. Asymptomatic;
4. Annual medical qualification examination;
5. The appropriate frequency of ETT after CABG is uncertain (89). However, after five years, yearly ETT because of accelerated graft closure. An acceptable exercise capacity is one in which the maximal heart rate achieved is $\geq$85% of the age-predicted maximum (unless the patient is on beta blockers), no ischemic signs or symptoms, a workload of at least six METS, and appropriate systolic blood pressure and heart rate responses and no ventricular dysrhythmias;
6. Radionuclide stress testing or echocardiographic myocardial imaging is indicated if the driver is not able to achieve a satisfactory ETT result, has a dysrhythmia, or has an abnormal resting electrocardiogram. The development or recurrent chest pain is not useful for detecting graft occlusion, with a reported sensitivity of 60% and specificity of 20% (121). The examiner should have a low threshold for requiring stress imaging studies instead of a standard ETT;
7. Resting echocardiogram at the time of the first qualifying examination after CABG (a documented report of an echocardiogram performed in-hospital after CABG is equally sufficient). Disqualification occurs in the presence of left ventricular dysfunction (ejection fraction < 40%); and
8. Tolerance to all cardiovascular medications with no orthostatic symptoms.

Target Organ Damage

The detection of target organ damage should trigger additional evaluation. The presence of atherosclerosis in one vascular bed (stroke or TIA, peripheral vascular disease, CHD) requires evaluation to determine if there is also significant atherosclerosis in other vascular beds. Nephropathy or retinopathy also reflect significant target organ damage and should trigger an evaluation for other CVD.

Target Organ Damage

Heart Disease
- Left Ventricular Hypertrophy
- Angina/Prior MI
- Prior Coronary Revascularization
- Heart Failure

Stroke or TIA
Nephropathy
Peripheral Vascular Disease
Retinopathy

RECOMMENDATION TABLES
COMMERCIAL DRIVERS WITHOUT KNOWN CHD

DIAGNOSIS	PHYSIOLOGIC/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Asymptomatic, healthy	Low CHD event risk. Assess for clinically apparent risk factors. Use, when possible, Framingham risk score model to predict 10-year CHD event risk; Increasing age is a surrogate marker for increasing atherosclerotic plaque burden.	Yes, if asymptomatic. Rarely disqualifying alone.	Biennial
Asymptomatic, high risk person (as designated by CHD risk-equivalent condition)* Asymptomatic, high risk person > 45 years with multiple risk factors for CHD	Sub-clinical coronary atherosclerosis is a concern; High-risk status requires close physician follow-up and aggressive comprehensive risk factor management.	Yes, if asymptomatic. No if: Abnormal ETT** Ischemic changes on ECG † Functional incapacitation by one of conditions.	Annual

*CHD risk equivalent is defined as presence of diabetes mellitus, peripheral vascular disease, or Framingham risk score predicting a 20% CHD event risk over the next 10 years. (Please see text for definition of abbreviations)

** Abnormal ETT is defined by an inability to exceed 6 METS (beyond completion of Stage II, or 6 minutes) on a standard Bruce protocol or the presence of ischemic symptoms and/or signs (e.g., characteristic angina pain or 1 mm ST depression or elevation in 2 or more leads), inappropriate SBP and/or heart rate responses (e.g., inability in the maximal heart rate to meet or exceed 85% of age-predicted maximal heart rate), or ventricular dysrhthymia.

† Ischemic ECG changes are defined by the presence of new 1 mm ST-segment elevation or depression and/or marked T wave abnormality.

RECOMMENDATION TABLES
COMMERCIAL DRIVERS WITH KNOWN CHD

DIAGNOSIS	PHYSIOLOGIC/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Post MI	Risk of recurrent major cardiac event highest within the first months post-MI; Drivers in a rehabilitation program can receive comprehensive secondary prevention therapy.	No if: Recurrent angina symptoms; Post-MI ejection fraction <40% (by echocardiogram or ventriculogram); Abnormal ETT demonstrated prior to planned work return; Ischemic changes on rest ECG; Poor tolerance to current cardiovascular medications. Yes if: At least 2 months post-MI; Cleared by cardiologist; No angina; Post-MI ejection fraction ≥40% (by echocardiogram or ventriculogram); Tolerance to current cardiovascular medications.	Annual Biennial ETT at minimum (If test positive or inconclusive, imaging stress test may be indicated); Cardiologist examination recommended.
Angina Pectoris	Lower end of spectrum among CHD patients for risk of adverse clinical outcomes. Condition usually implies at least one coronary artery has hemodynamically significant narrowing.	Yes, if asymptomatic. No if: Rest angina or change in angina pattern within 3 months of examination; Abnormal ETT; Ischemic changes on rest ECG; Intolerance to cardiovascular therapy.	Annual Biennial ETT at minimum (If test positive or inconclusive, imaging stress test may be indicated); Cardiologist examination recommended.

RECOMMENDATION TABLES
COMMERCIAL DRIVERS WITH KNOWN CHD
(Continued)

DIAGNOSIS	PHYSIOLOGIC/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Post PCI	Rapid recovery for elective PCIs for stable angina; Delayed re-stenosis is the major PCI limitation and requires intensive secondary prevention.	Yes if: At least 1 week after procedure; Cardiologist's approval; and Tolerance to medications. ETT 3 to 6 months after PCI. No if: Incomplete healing or complication at vascular access site; Rest angina; Ischemic ECG changes.	Annual Recommend Cardiologist examination. Biennial ETT at minimum (If test positive or inconclusive, imaging stress test may be indicated).
Post Coronary Artery Bypass Surgery (CABG)	Delay in return to work to allow sternal incision healing. Because of increasing risk of graft closure over time, ETT is obtained.	Yes if: At least 3 months after CABG; LVEF $\geq$ 40% post CABG; Approval by cardiologist; Asymptomatic; and tolerance to medications.	Annual After 5 years: Annual ETT. Imaging stress test may be indicated.

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Hypertension

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HYPERTENSION

Epidemiology and Impact on Public Health

An estimated 50 million Americans have hypertension, making this disease the most prevalent cardiovascular condition among adults in the United States (1). The definition of hypertension is somewhat arbitrary and not directly tied to a threshold blood pressure (BP) that must be crossed before it leads to pathology. Hypertension is typically defined as the level of pressure associated with a doubling of long-term risk (2). Blood pressures greater than 140/90 mmHg are deemed high for most individuals without other significant cardiovascular risk factors.

There are a number of factors in the clinical setting that should be considered before diagnosing hypertension. Blood pressure levels may follow a circadian cycle and may also fluctuate according to a person's emotional and physical state. Transient elevation of BP commonly occurs in the physician's office or hospital setting and defines the "white coat" hypertensive individual who has normal BP levels outside of the traditional medical setting (3).

While automatic ambulatory recorders and at-home sphygmomanometers may be useful for confirming the diagnosis of hypertension or guiding therapy, there is insufficient follow-up data to support their use as a long-term risk predictor. Office readings are the standard on which to base medical certifying decisions. An appropriately sized BP cuff should be used to minimize measurement bias. An initial elevated BP determination should be confirmed by at least two readings taken on two different follow-up days.

Current disease recognition and treatment strategies for hypertension are far from optimal. Data from the Third National Health and Nutrition Examination Survey NHANES III) indicate that 32% of all persons with hypertension are unaware of their condition and are not receiving treatment, 15% are aware of it but are not receiving treatment, and 26% have treated but uncontrolled hypertension. This leaves only 27% of all U.S. hypertensive individuals with adequate control of their blood pressure (4). Reasons for these poor care patterns are likely to be due to a number of complex factors that include poor adherence to antihypertensive medical regimens due to the chronic duration of their administration, their cost, or side effects, patients' or providers' biases, attitudes, and priorities regarding the importance of timely BP control, and inadequate health care access for, or attention to, the management of chronic illness (5).

Hypertension is closely associated with the aging process. The rising prevalence of age-associated hypertension is related to the increased stiffness in the walls of aging major vasculature. The replacement of elastin by collagen in these arteries leads to a process of progressive dilatation and lengthening of the aorta and its immediate branches through fibrosis and hypertrophy of the arterial muscularis (6). The loss of arterial compliance leads to a progressive rise in systolic blood pressure (SBP) and widening of pulse pressure (PP). Both SBP and PP are suitable markers of arterial stiffness, are easily and

reliably measured in the clinical setting, and provide the greatest predictive power for assessing risk.

The relationship between hypertension and adverse cardiovascular outcomes is graded, independent and continuous. The Multiple Risk Factor Intervention Trial convincingly demonstrated that in 316,099 white men the 12-year CHD death rate was continuously and linearly related to the SBP level (7). Twenty-year follow-up data from the Framingham Heart Study corroborated these data. However, in this cohort of middle- and older-aged men, PP proved superior both to SBP and diastolic blood pressure (DBP) in predicting CHD risk. Diastolic pressure, when placed in the Framingham multivariate risk model, is actually inversely related to CHD risk. This supports the concept that a low diastolic pressure in the setting of a normal or high systolic pressure implies stiff vasculature and carries with it a heightened risk of cardiovascular morbidity and mortality (8).

While elevations in SBP remain more closely associated with increased CHD events than do elevations in DBP, these data do not diminish the value of recognizing elevated DBP levels in adult populations. Both elevated SBP and DBP were recently observed to predict 25-year CHD, CVD, and all-cause mortality in a cohort of men (18-39 years) from the Chicago Heart Association Detection Project in Industry (9). Long-term data similarly show increased rates of cerebral, cardiac, and renal complications in patients with poorly treated, elevated levels of DBP (10). Among hypertensive patients, the number of deaths from CHD exceeds deaths due to stroke by a ratio of 3-4:1 (11). Hypertension is a potent risk factor for the development of peripheral vascular disease and chronic renal insufficiency.

Hypertension directly promotes CHD by impairing endothelial cell function, increasing the risk of plaque rupture, and promoting left ventricular hypertrophy (12,13). Left ventricular hypertrophy lowers the threshold for development of subendocardial ischemia and independently predicts long-term risk of sudden cardiac death (14).

The diagnosis of hypertension should trigger an evaluation for other cardiovascular risk factors and target-organ damage, since these add prognostic information and may impact the goal of hypertension therapy. Smoking, family history, diabetes, cholesterol status, and degree of alcohol consumption should be obtained from the clinical history.

Causes of Hypertension Among Commercial Drivers

The majority of younger commercial drivers have a low prevalence of traditional coronary risk factors, including hypertension. This may be due largely to a rigorous up-front licensing process that adversely selects against “unfit” individuals or the natural selection of those healthy enough to meet the job requirements -- the "healthy worker effect."

Once within the profession, however, commercial drivers have a higher propensity to develop hypertension than their peers in other professions (15-20). Ragland et al. demonstrated in a cohort study of 2,052 transit vehicle operators in San Francisco that the prevalence of hypertension (systolic blood pressure $\geq$ 140 mmHg or diastolic blood pressure $\geq$ 90 mmHg) increased in a stepwise fashion from 29% in the group of drivers without any driving experience, to 32% in drivers with 10-20 years experience, and to 39% in drivers with > 20 years driving experience (15). As the years of experience increase, part of the increase in hypertension may relate to the accompanying aging, increase in body mass, or decline in physical activity.

Other mediators of hypertension may be directly related to commercial driving. Urine catecholamine excretion rates have been found to be markedly elevated among lorry drivers engaged in either mentally stressful activities (e.g., driving during poor weather or in heavy traffic) or physically stressful activities (e.g., heavy physical workload) (21, 22). These elevations persist after adjustment for alcohol consumption and body mass index. The fact that adrenaline and noradrenaline excretion rates remain elevated through working days and incompletely recover after the end of work periods implies a sustained level of sympathoadrenal medullary activation in affected drivers. Such a sustained response that recurs for years on end could lead to changes in arterial tone, vascular endothelial function, and/or myocardial energetics that are unfavorable to cardiovascular health.

Further supporting an activated sympathomimetic response among healthy commercial drivers at high risk for hypertension and CVD is their heightened BP responsiveness to either threat avoidant aspects of driving presented in the laboratory setting or during exercise stress testing. Belkic et al. (19) compared electroencephalographic (EEG) and polygraphically recorded cardiovascular reactivity to the glare pressor test in 19 healthy, young male professional drivers and 8 non-driver controls. While no significant EEG or BP changes occurred in the control group, substantial alterations occurred in the large majority of truck drivers when exposed to sequential headlight impulses (e.g., the glare). Diastolic BP increased an average of 7.3 +/- 9.5 mmHg, hyperactive EEG changes occurred, heart rates slowed, and blood flow measured in the finger diminished (implying vasoconstriction). This vigilance response to an oncoming headlight that both truck and bus drivers frequently encounter demonstrates the demands that the operation of a commercial vehicle has on a driver's psyche and his/her neuro-cardiovascular system.

Drivers also have a significantly lower maximum exercise level when compared with their non-driving peers. The fact that they generate a significantly higher double product than non-drivers to attain this maximum level indicates a lower level of physical fitness (23). A significantly higher DBP at the end of exercise and more diastolic hypertensive reactions (DBP > 115 mmHg) identify a cardiovascular system at higher risk for developing hypertension and/or atherosclerotic vascular disease.

The Effect of Hypertension on Driver Safety

The standard (49 C.F.R. section 391.41(b)(6)) permits qualification of individuals to drive if the driver "has no current clinical diagnosis of high blood pressure likely to interfere with his/her ability to operate a motor vehicle safely." Controversy surrounds the impact of hypertension on commercial driving safety. A study of truck drivers (24) and a second study of bus drivers (25) failed to find a significant relationship between hypertension and the frequency of crashes. A more recent study by the same group of investigators demonstrated that crashes involving bus drivers with hypertension (DBP between 110 and 130 mmHg which were controlled by medication to < 110 mmHg) were more severe than those of healthy drivers (26). The study was limited by its use of only one data set from the province of Québec, Canada, and by its inability to adjust for crash responsibility. Other studies have correlated elevated DBP and decreased cognitive function (27-29). A decline in cognitive function has been associated with an increased risk for motor vehicle crashes (30).

Defining Medically Acceptable Blood Pressure in the Commercial Driver

The Sixth Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 6) (31) established three stages of hypertension. These levels define hypertension and guide therapy and are useful in determining medical certification to a driver. Although recommending specific therapy is beyond the scope of the physical examination, the medical examiner is concerned with the blood pressure response to treatment. Failure to achieve the blood pressure goals of JNC 6 can pose long-term health risks.

Stage 1 Hypertension

Stage 1 hypertension corresponds to a SBP of 140-159 mmHg and/or a DBP of 90-99 mmHg. The driver with a BP in this range is at low risk for hypertension-related acute incapacitation and may be medically certified to drive for a one-year period. DOT certification examinations should be done annually thereafter. The BP should be $\leq$140/90 at these annual examinations. All hypertensive drivers should be strongly encouraged to pursue consultation with their personal physicians to ensure appropriate therapy targeting an optimal BP below 130/85 mmHg with education and management related to lowering risk for CVD.

Stage 2 Hypertension

Stage 2 hypertension corresponds to a SBP of 160-179 mmHg and/or a DBP of 100-109 mmHg. A BP in this range is considered an absolute indication for anti-hypertensive drug therapy (31). A driver with Stage 2 hypertension at the time of the DOT examination may be medically certified to drive commercially for a single three month

period while he/she seeks initiation of therapy with his/her primary provider. Provided the driver has received adequate treatment that is also well-tolerated and demonstrates a BP value <140/90 mmHg, he/she may be medically qualified for 12 months from the date of the examination when the three month certification was given.

Subsequent certification should occur annually thereafter. Because the commercial driver is presumed to be on medication, the Panel recommends that the BP be ≤ 140/90 mmHg. Effective BP management includes routine primary physician follow-up, an optimal BP of <130/85 mmHg, and periodic screens for the presence of target organ damage (TOD) and clinical manifestations of CVD.

Stage 3 Hypertension

Stage 3 hypertension is defined as a SBP ≥ 180 mmHg and/or DBP ≥ 110 mmHg and carries a high risk for the development of acute hypertension-related symptoms that could impair judgment and driving ability. Stage 3 hypertension is immediately disqualifying and an indication for immediate drug therapy.

Provided the driver is receiving adequate treatment that is well-tolerated and demonstrates a BP value ≤140/90 mmHg, he/she may be medically certified for six months from the date of the examination when the driver was disqualified. Subsequent re-certification should occur every six months. Because the commercial driver is presumed to be on medication, the Panel recommends that the BP be maintained at ≤ 140/90 mmHg.

Risk of Acute Incapacitation from Hypertension

In general, isolated hypertension is unlikely to cause sudden incapacitation, although the presence of target organ damage, particularly when the cerebrovascular system is involved, increases the likelihood. Acute incapacitation is more likely to be caused by a sudden ischemic coronary event.

Acute manifestations of an elevated BP can include sudden stroke, acute pulmonary edema, subarachnoid hemorrhage, aortic dissection, or aortic aneurysm rupture. Meningismus, acute neurological deficits, abrupt onset of shortness of breath, or severe, ripping back or chest pain could signal an impending hypertensive catastrophe that requires immediate cessation of driving and emergent medical care. Symptoms of hypertensive urgency such as headache and nausea are likely to be more subtle, subacute in onset, and more amenable to treatment than a hypertensive emergency.

Treatment

There is also strong prospective, randomized trial evidence that effective hypertension management reduces cardiovascular morbidity and mortality in the primary and secondary settings. Healthy lifestyle modification and pharmacotherapy are the mainstays of anti-hypertensive treatment regimens. Contemporary medical therapies are effective in lowering BP, reducing complications, and are generally regarded as safe. The driver should have ongoing hypertensive management from his/her primary provider.

To meet qualification standards, commercial drivers on antihypertensive medications must be free of any side effects that could impair their job performance. Medications that predispose to precipitous declines in BP, syncope, fatigue, or excessive electrolyte shifts should be avoided. Commercial drivers should also be made aware of their drugs' interactions with other prescription and non-prescription drugs and alcohol.

The Need for Blood Pressure Control to Prevent Target Organ Damage

Medical examiners should search for target organ damage using their clinical and physical examination skills and, when indicated, request additional ancillary diagnostic tests such as serum creatinine or urine (micro) albumin tests, an electrocardiogram, or an echocardiogram. A driver with evidence of target organ damage should receive close follow-up from his/her personal physician and aggressive BP management with a goal BP < 130/80 mmHg.

The commercial driver with multiple risk factors for heart disease or target organ damage may be required to meet more intensive blood pressure control by his or her primary care physician. Severe target organ damage that has occurred due to longstanding, refractory hypertension may be grounds for disqualification if the damage impairs the driver's ability to carry out safely his/her daily job operations.

Secondary Hypertension

The prevalence of secondary hypertension in the general population is estimated to be between 5% and 20%. Examples of secondary causes of hypertension include pheochromocytoma, primary aldosteronism, renovascular disease, or unilateral renal parenchymal disease.

The primary care physician may evaluate patients with refractory hypertension despite being on near maximal doses of two to three pharmacologic agents for secondary hypertension. Some causes of secondary hypertension may be amenable to surgical intervention or specific pharmacologic treatment.

RECOMMENDATION TABLES
HYPERTENSION

DIAGNOSIS	PHYSIOLOGIC/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Essential Hypertension	Evaluate for other clinical CVD including TOD†; Presence of TOD, CVD or diabetes may affect therapy selected.		
Stage 1 (140-159/90-99 mmHg)	Usually asymptomatic; Low risk for near-term incapacitating event.	Yes Rarely disqualifying alone.	Annual BP <140/90 at annual exam; If not, but <160/100, certification extended 1 time for 3 months.
Stage 2 (160–179/100–109 mmHg)	Low risk for incapacitating event; risk increased in presence of TOD; Indication for pharmacologic therapy.	Yes One time certification for 3 months. Yes, at recheck if: BP ≤140/90mmHg Certify for 1 year from date of initial exam.	 Annual BP ≤140/90.
Stage 3 (≥180/110 mmHg?	High risk for acute hypertension-related event.	No Immediately disqualifying; Yes, at recheck if: BP ≤140/90 mmHg., and treatment is well tolerated. Certify for 6 months from date of initial exam.	 Every 6 months; BP ≤140/90.
Secondary Hypertension	Evaluation warranted if persistently hypertensive on maximal or near-maximal doses of 2-3 pharmacologic agents; May be amenable to surgical/specific therapy.	Based on above stages. Yes if: Stage 1 or nonhypertensive. At least 3 months after surgical correction.	 Annual BP ≤140/90

† TOD – Target Organ Damage – Heart Failure, Stroke or Transient Ischemic Attack, Peripheral Artery Disease, Retinopathy, Left Ventricular Hypertrophy, Nephropathy. Examiner may disqualify a driver if TOD significantly impairs driver's work capacity. Driver should have no excess sedation or orthostatic change in BP.

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Valvular Heart Disease

Myocardial Disease

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VALVULAR HEART DISEASE

General Recommendations

This report draws heavily upon the ACC/AHA Guidelines for the Management of Patients with Valvular Heart Disease (1).

Throughout this section on valvular heart disease, congestive heart failure, and the cardiomyopathies, it is recommended that a cardiologist perform the necessary evaluation. Echocardiography plays a key role in the evaluation of the severity of valvular heart disease and it is recommended that the echocardiography laboratory performing the evaluation have expertise and experience in the assessment of valvular heart disease and left ventricular function.

The standard initial evaluation includes a history, physical examination, electrocardiogram, and in many patients, a chest x-ray. Recommendations regarding echocardiography and other investigations, including stress testing, will be addressed in the text under the specific condition.

It is not likely that the medical examiner will conduct the testing outlined in this section. However, the review of the testing requirements provides the examiner the information to assess whether the driver has been adequately evaluated, and to classify the severity of the disease as it relates to the driver's general health and ability to be medically certified.

MITRAL STENOSIS (TABLE 1)

Natural History

The natural history of untreated mitral stenosis is based on studies in the 1950s and 1960s (2-7). In both North America and Europe, the decline in the incidence of rheumatic fever has probably resulted in a milder, delayed course of progression and the mean age of presentation is now in the fifth and sixth decades (3,4,8).

Prior studies suggest that there is a 20 to 40-year latent period from the onset of rheumatic fever to the development of symptoms. Overall 10-year survival of asymptomatic or minimally symptomatic patients is greater than 80%, and symptoms remain stable in 60% (6,7,9). With the development of significant limiting symptoms, New York Heart Association Class II or higher and/or severe pulmonary hypertension, subsequent survival is poor. Management is based primarily on the development of symptoms and pulmonary hypertension rather than the severity of the stenosis, per se.

Evaluation and Management

Initial:

1. Standard evaluation;
2. 2-Dimensional Doppler Echocardiography; and
3. Stress testing may be helpful in some situations to assess objective evidence of effort tolerance and the role of mitral stenosis upon functional capacity.

The severity of mitral stenosis is assessed by 2-dimensional and Doppler echocardiography incorporating parameters such as the planimetry of the orifice area, the morphological appearance of the mitral valve apparatus, including the leaflets and subvalvular structures and the presence of calcification, in addition to measurement of the hemodynamic severity of the obstruction (10-13). The hemodynamic severity is determined by the mean transmitral gradient measured from the continuous wave Doppler signal across the mitral valve. The mitral valve area can be derived non-invasively from Doppler echocardiography using the diastolic half-time method and the continuity equation (11,13-16). It is suggested that both these methods be used in the individual patient.

The normal mitral valve area is 4-6 cm^2. Mild mitral stenosis is defined by a valve area of 1.6-2.0 cm^2 and a mean gradient of < 5 mmHg; moderate mitral stenosis is a valve area of 1.0 to 1.6 cm^2; and severe mitral stenosis by a valve area of $\leq$ 1.0 cm^2, or a resting mean pressure gradient of $\geq$ 10 mmHg or a pressure half-time (PHT) of $\geq$ 220 msec (13).

Follow-Up Evaluation

Although the frequency of follow-up examinations depends on the development and severity of symptoms, it should be performed by a cardiologist at least annually and should include a history, physical examination, chest x-ray, ECG, and usually 2-dimensional echocardiography. In certain situations, cardiac catheterization and transesophageal echocardiography may be required, particularly if a surgical or percutaneous procedure is contemplated.

Current Recommendations

The 10-year natural history of asymptomatic or minimally symptomatic mitral stenosis is favorable (greater than 80% survival at ten years). In addition, mortality is usually due to progressive congestive heart failure, systemic embolism, pulmonary embolism or infection. Sudden cardiac death in the absence of severe symptoms is rare (1).

Moreover, the development of symptoms will limit the ability of the individual to perform specific tasks associated with commercial driving; for example, changing a tire or lifting loads. For these reasons it is reasonable not to base restrictions upon valve area alone, but to rely on symptoms, atrial fibrillation or systemic embolism to determine disqualification (Table 1).

Disqualify:

1. Symptoms - New York Heart Association ≥Class II;
2. Paroxysmal or established atrial fibrillation;
3. History of systemic embolism;
4. Pulmonary hypertension (Pulmonary artery systolic pressure of ≥ 50% of systemic systolic blood). The pressure is determined by echocardiography or cardiac catheterization;
5. Stress test with inability to exercise to a workload greater than 6 METS on the Bruce protocol (Stage II); or
6. Effort intolerance attributable to a cardiac cause.

MITRAL REGURGITATION (TABLE 2)

The etiologies of mitral regurgitation are diverse and, to some extent, determine its natural history. Mitral regurgitation can present as an acute or chronic condition. The latter is the subject of this discussion.

Despite the volume overload resulting from moderate to severe mitral regurgitation, both the left ventricle and the left atrium may accommodate to an extent and patients may be asymptomatic even during vigorous exercise (17,18). The duration of the compensated phase of mitral regurgitation is variable and may last for years, but the development of left ventricular dysfunction (19), atrial fibrillation, infective endocarditis, or progressive mitral regurgitation due to chordal rupture or pulmonary hypertension, ultimately leads to decompensation. The development of symptoms, especially dyspnea, fatigue, orthopnea, and/or paroxysmal nocturnal dyspnea is a marker of a poor prognosis including an inability to perform tasks associated with commercial driving and sudden cardiac death.

Evaluation and Management

Initial:

1. Standard initial evaluation;
2. Chest X-ray; and
3. 2-Dimensional and Doppler Echocardiography (20).

The precise assessment of the severity of mitral and other regurgitant lesions is difficult using invasive or noninvasive techniques (20). It is essential that the echocardiography laboratory be experienced in these techniques. Methods of quantitating the severity of mitral regurgitation include an assessment of the regurgitant jet characteristics (length, height, area, and width at the vena contracta), the effective regurgitant orifice area and regurgitant flow volume, and the proximal isovelocity surface area method (PISA) for the regurgitant fraction.

Other estimates of regurgitant severity include the rate of decline in regurgitant gradient as measured by the slope of the diastolic flow and by a reduction or reversal of the systolic

components of venous inflow (13, 21-24). Assessment of left ventricular and left atrial size, in addition to left ventricular systolic function, may provide added information about the severity of chronic mitral regurgitation.

Severe chronic mitral regurgitation is defined by a mitral regurgitant volume of $\geq$ 50 cc, an effective regurgitant orifice area of $\geq$ 40 cm^2, a regurgitant fraction of $\geq$ 55%, and 2-dimensional echocardiographic evidence of disruption of the mitral valve apparatus (flail mitral leaflet), and ruptured chordae tendineae (13).

Stress testing is indicated to obtain objective measurements of effort tolerance and the severity of mitral regurgitation during exercise in patients in whom the symptomatic status is unclear (8). Although transesophageal echocardiography is not routinely indicated, it may be helpful to assess structural abnormalities, such as ruptured chordae and flail leaflet.

Follow-up Evaluation

1. Mild Mitral Regurgitation: Annual evaluation in patients with no evidence of left ventricular dysfunction, left ventricular enlargement, or pulmonary hypertension. Annual echocardiography is not necessary in the absence of progression in the severity of regurgitation;
2. Moderate Mitral Regurgitation: Annual evaluation. Echocardiography should not be performed more than once per year;
3. Severe Mitral Regurgitation: Six to 12 month evaluation and echocardiography to assess left ventricular function. Exercise testing may be helpful to assess symptoms or changes in effort tolerance.

Current Recommendations

Disqualify:
Severe mitral regurgitation.

1. Symptoms or reduced effort tolerance ($\leq$ 6 METS or $\leq$ 6 minutes on a Bruce protocol);
2. Ruptured chordae or flail leaflet;
3. Atrial fibrillation;
4. Left ventricular dysfunction. (ejection fraction <60% or left ventricular end systolic dimension >45 mm or left ventricular end diastolic dimension >70 mm);
5. Thromboembolism; or
6. Pulmonary hypertension (pulmonary artery pressure >50% of systemic arterial pressure as determined by echocardiography or cardiac catheterization.

MITRAL VALVE PROLAPSE

The natural history of mitral valve prolapse is extremely variable and depends on the extent of myxomatous degeneration, the degree of mitral regurgitation, and association with other conditions (e.g., Marfan syndrome and connective tissue diseases). In a substantial proportion of patients, mitral valve prolapse may be associated with tricuspid valve prolapse and occasionally the pulmonic and aortic valves may be involved (25,26).

For the vast majority of patients, the natural history of the mitral valve prolapse syndrome is benign, with survival equivalent to that of age and sex-matched controls (27,28). In a minority of patients (particularly among patients with thickened, redundant valve leaflets and significant mitral regurgitation), the regurgitation may be progressive, resulting in left ventricular and left atrial enlargement, atrial fibrillation, and congestive heart failure. Sudden death is a rare complication (27,29-32), with an annual mortality of less than 1% per year, but may be higher in patients with the familial form of the syndrome (33) and severe myxomatous degeneration of both leaflets (34). Infective endocarditis is a potentially serious complication (35) and an association between mitral valve prolapse and cerebrovascular accident in young patients has been suggested (36).

Diagnosis and Evaluation

Initial:

1. Standard Initial Evaluation; and
2. 2-Dimensional and Doppler Echocardiography.

All patients should have an initial echocardiogram to confirm the diagnosis, assess the severity of mitral regurgitation and the likelihood of complications based upon the extent of leaflet thickening and redundancy. In some patients, prior cardiac catheterization may suffice for diagnostic purposes.

Follow-up Evaluation

Regular echocardiographic follow-up in an asymptomatic patient with minimal mitral regurgitation and without severely thickened or redundant leaflets is not necessary. Among patients who have definite mitral regurgitation (even if mild) or markedly thickened leaflets, follow-up should follow the guidelines stated under the "Mitral Regurgitation" section, and requires annual re-evaluation.

Disqualify:

1. Symptoms or reduced effort tolerance due to mitral valve prolapse or mitral regurgitation;
2. Ruptured chordae or flail leaflet;
3. Systemic emboli;
4. Atrial fibrillation;

5. Syncope or documented ventricular tachycardia; or
6. Severe mitral regurgitation or left ventricular dysfunction.

AORTIC STENOSIS (TABLE 3)

Natural History

The most common cause of aortic stenosis in adults is a degenerative process associated with many of the risk factors underlying atherosclerosis. Aortic stenosis in younger patients due to a congenital bicuspid valve is also susceptible over time to progressive fibrosis and calcification. Calcification is a common feature of aortic stenosis in older adults, regardless of the primary cause (9,37,38).

The natural history is characterized by a prolonged latent period during which the development of symptoms and morbidity are very low (39-41). This is primarily due to the development of compensatory left ventricular hypertrophy, which provides an adaptation to increases in wall stress and increased intracavity pressures (42-44). Although sudden cardiac death without preceding cardiac symptoms is reported, it is a rare event occurring at a rate of less than 1% per year (45).

Following the onset of symptoms, namely angina, syncope, or congestive heart failure, average survival is less than two to three years (45-50), and sudden death is well documented. In a recent series of patients who were asymptomatic in daily life, 6% experienced sudden death over a 4-to 5-year follow-up, and all of these had a positive exercise test and severe aortic stenosis with a valve area of ≤ 0.6 cm^2 (51). In three recent prospective echocardiographic series, sudden cardiac death was uncommon, but was preceded by symptoms in all patients (52).

Management and Evaluation
Asymptomatic Commercial Driver

Initial:

1. Standard initial evaluation;
2. 2-Dimensional and Doppler Echocardiography to assess gradient, valve area, and severity of left ventricular hypertrophy. Cardiac Catheterization and Coronary Angiography is occasionally required; and
3. Exercise Testing is occasionally required to assess symptoms, effort tolerance and prognosis (51). Close monitoring of the blood pressure and electrocardiogram is essential.

Follow-up Evaluation

The frequency of evaluation depends upon symptoms and the severity of the aortic stenosis. The classification used in the ACC/AHA Guidelines is that mild aortic stenosis is defined by a valve area of >1.5 cm^2, moderate by a valve area ≥1.0 to 1.5 cm^2, and severe by a valve area of <1 cm^2 (1,13,41,53). Among patients with normal left ventricular systolic function, aortic stenosis is usually considered as severe when the peak aortic valve velocity is ≥ 50 mmHg (54).

Follow-up Evaluation

1. Mild Aortic Stenosis (valve area > 1.5 cm^2): Annual clinical evaluation; Echocardiography every five years unless clinical findings change;
2. Moderate Aortic Stenosis (valve area ≥ 1.0-1.5 cm^2): Annual clinical evaluation; Echocardiography every one or two years; and
3. Severe Aortic Stenosis (valve area < 1 cm^2).

Current Recommendations

Disqualify:

1. Any symptoms due to aortic stenosis (e.g., syncope, congestive heart failure, reduced effort tolerance, or angina) in patients with moderate or severe aortic stenosis;
2. If symptoms are consistent with aortic stenosis but the clinical and echocardiographic presentation suggests mild aortic stenosis, the severity of the valve lesion and alternative explanations for symptoms needs to be reassessed. This may require cardiac catheterization in some patients or documentation of reduced effort tolerance by stress testing;
3. Asymptomatic, severe aortic stenosis (aortic valve area <1.0 cm^2);
4. Moderate aortic stenosis plus left ventricular dysfunction (ejection fraction less than 40%);
5. Atrial fibrillation and moderate to severe aortic stenosis;
6. Thromboembolism and moderate to severe aortic stenosis; or
7. Positive stress test.

AORTIC REGURGITATION (TABLE 4)

There are several common causes of aortic regurgitation and multiple other unusual etiologies (1). Aortic regurgitation is usually a chronic condition characterized by a prolonged, asymptomatic phase and gradual left ventricular dilatation, whereas other conditions such as infective endocarditis and aortic dissection can result in acute severe aortic regurgitation. This discussion is confined to chronic aortic regurgitation.

Compensatory mechanisms for chronic aortic regurgitation include left ventricular dilatation with both eccentric and concentric hypertrophy, which usually results in an asymptomatic, compensated phase of many years' duration (55-57). Eventually, decompensation begins with a

decline in left ventricular ejection fraction, followed or accompanied by the development of the symptoms of dyspnea or, in some patients, angina (58-60).

Seven studies of the natural history of aortic regurgitation involving 490 patients have been performed and are summarized in the ACC/AHA Guidelines (1). From the standpoint of commercial driving, it is relevant to note that among asymptomatic patients with normal systolic function, the rate of sudden cardiac death is extremely low, and the rate of progression to symptoms and/or left ventricular dysfunction is approximately 4.3% per year. In contrast among patients with asymptomatic left ventricular dysfunction, the development of cardiac symptoms occurs at a rate of >25% per year. In symptomatic patients, the mortality rate is >10% per year with angina, and greater than 20% per year with congestive heart failure (41,61,62). Recent studies on symptomatic patients indicate a poor outcome on medical therapy, even in the presence of preserved left ventricular function (63,64).

Evaluation and Management

1. Standard Initial Evaluation;
2. Chest X-ray;
3. 2-Dimensional and Doppler Echocardiography;
4. Exercise testing may be helpful in patients who are sedentary or who have equivocal symptoms. Some studies have suggested that the radionuclide ejection fraction response to exercise may be of prognostic value; and
5. Echocardiography. The severity of aortic regurgitation is determined semiquantitatively by echocardiography in addition to its assessment of left ventricular mass, left ventricular dimensions, systolic function, and aortic root size (13). Direct semiquantitative measures of the severity of aortic regurgitation comprise an assessment of color-flow jet area and width by Doppler echocardiography. Additional indirect information is provided by the rate of decline in regurgitant gradient as measured by the slope of diastolic flow velocity, the degree of reversal of the pulse wave velocity in the descending aorta, and the magnitude of the left ventricular outflow tract velocity (1).

The ACC/AHA Guidelines state that comparison of stroke volumes at the aortic valve compared with other uninvolved valves may provide a quantitative measurement of regurgitant volume. The echocardiography evaluation requires an expert and experienced laboratory and echocardiographer (1,13,65). On the basis of the data from 2-dimensional Doppler and color flow imaging, the severity of aortic regurgitation is determined at Mayo Clinic as follows (13):

Severe Aortic Regurgitation:

1. Regurgitant jet width / left ventricular outflow tract (LVOT) diameter ratio $\geq$60%;
2. Regurgitant jet area / LVOT area ratio $\geq$60%.
3. Aortic regurgitation pressure half time (PHT) $\leq$250 msec;
4. Holodiastolic flow reversal in the descending aorta;
5. Restrictive mitral flow pattern (usually in an acute setting);
6. Dense, continuous-wave Doppler signal;

7. Regurgitant fraction ≥55%, regurgitant volume ≥60 mL; and
8. Effective regurgitant orifice ≥0.30 cm^2

Mild Aortic Regurgitation:

1. Regurgitant jet width/LVOT diameter ratio ≥30%;
2. Regurgitant jet area/LVOT diameter ≥30%;
3. Aortic regurgitation PHT ≥300 Msec;
4. Mild early diastolic flow reversal in the descending aorta;
5. Faint, continuous-wave Doppler signal;
6. Regurgitant fraction <30%;
7. LV diastolic dimension (chronic) <6.0 cm; and
8. Effective regurgitant orifice ≤1.0 cm^2 (13).

Follow-up Evaluation

The severity, stability, and chronicity of the lesion and the response of the left ventricle to volume overload determine testing frequency. The ACC/AHA Guidelines recommend a second examination two to three months after the initial examination to ensure that the severity of the aortic regurgitation is not progressive (1).

Asymptomatic Patients with Mild or Moderate Aortic Regurgitation
In the presence of normal left ventricular systolic function and little or no left ventricular enlargement, patients should be seen annually with an echocardiogram performed every two to three years.

Asymptomatic Patients with Severe Aortic Regurgitation
With a normal left ventricular systolic function but significant left ventricular dilatation, the patient should be clinically evaluated every six months and an echocardiogram performed every six to twelve months. If the patient with more advanced left ventricular dilatation (end-diastolic dimension >60 mm or end-systolic dimension >50 mm), is not referred immediately for surgery, it is reasonable to perform echocardiography every four to six months.

Current Recommendations

Disqualify driver with Severe Aortic Regurgitation if:

1. Symptomatic or unable to achieve workload > 6 METS on Bruce protocol;
2. Reduced ejection fraction (≤ 50%);
3. Normal ejection fraction but end-systolic dimension >55 mm or end-diastolic dimension >70 mm; or
4. Atrial fibrillation.

TRICUSPID VALVE REGURGITATION

Discussed in the congenital heart disease section.

TRICUSPID VALVE STENOSIS

This is rare as an isolated entity. Drivers with symptoms from tricuspid stenosis should be disqualified.

PULMONARY VALVE STENOSIS AND REGURGITATION

Discussed in the congenital heart disease section.

PERCUTANEOUS BALLOON VALVOTOMY OR SURGICAL COMMISSUROTOMY FOR MITRAL STENOSIS

Symptomatic improvement occurs almost immediately, but after nine years, recurrent symptoms are present in approximately 60% of patients (66-68). In the minority of patients, these may be due to restenosis, but other valvular problems or coronary artery disease is frequently implicated (68). The recurrence of symptoms due to restenosis or mitral regurgitation is a function, in part, of the initial valve morphology and the anatomical result of the procedure.

Annual Evaluation

The driver should have an annual cardiology evaluation which should include:

1. History;
2. Physical examination;
3. Electrocardiogram;
4. Chest X-Ray; and
5. 2-Dimensional Echocardiography with Doppler is performed after the procedure and prior to discharge. The frequency of repeat echo-Doppler examinations is variable and depends upon the initial periprocedural outcome and the occurrence of symptoms.

Recommendations (Post Procedure Certification)

1. Waiting period of at least 4 weeks after percutaneous balloon mitral valvotomy and 3 months after surgical commissurotomy;
2. Cleared by cardiologist;
3. Use criteria similar to that for isolated mitral stenosis or regurgitation;

4. No thromboembolic complications following procedure; and
5. No pulmonary hypertension. (Pulmonary artery pressure >50% of systemic blood pressure.

MITRAL VALVE REPAIR FOR MITRAL REGURGITATION

The majority of inadequate valvular repair procedures can be detected in the early perioperative period. Careful evaluation at this time include a 2-Dimensional Doppler echocardiogram and, if necessary, transesophageal echocardiography (1).

Recommendations

Clearance to return to work after Mitral valve repair for commercial drivers with asymptomatic mitral regurgitation is based on whether the condition is mild, moderate, or severe:

1. Three-month waiting period;
2. No thromboembolic complications;
3. Atrial fibrillation (refer to section on arrhythmias); and
4. No Pulmonary hypertension (Pulmonary pressure >50% of systemic blood pressure).

AORTIC VALVE REPAIR

Early postoperative evaluation is required to assess adequacy of repair and extent of residual aortic regurgitation. Two-Dimensional with Doppler echocardiography should be performed prior to discharge.

Recommendations

1. 3-month waiting period;
2. Cleared by cardiologist;
3. No thromboembolic complications post procedure; and
4. Use similar criteria as for asymptomatic aortic regurgitation.

PROSTHETIC VALVES (TABLE 5)

It is understood that these recommendations may not apply to all prosthetic valves, since certain models with an increased incidence of dysfunction will require disqualification.

Aortic and Mitral Mechanical Prostheses
Natural History

There are a wide range of reported complications depending upon the variable methods of reporting, the make and model of the prosthesis, the site of implantation, comorbidities, and underlying left ventricular function, among other factors. The clinical course is heavily influenced by factors other than valve-related complications, for example, left ventricular dysfunction, congestive heart failure, progression of disease in other valves, coronary disease, or pulmonary hypertension (69).

Recommendation-Initial Postoperative Evaluation (TABLE 5)

The evaluation before re-certification should be performed at least 3 months after surgery.

Disqualify:

1. Persistent symptoms;
2. Left ventricular dysfunction (ejection fraction < 40%);
3. Thromboembolic complications post procedure;
4. Atrial fibrillation (refer to "Arrhythmia" section);
5. Pulmonary hypertension (> 50% systemic pressure); or
6. Commercial motor vehicle driver is unable to maintain adequate anticoagulation based on INR checks at least monthly.

Biologic Prosthesis

Recommendations are as above, with the exception that a requirement for anticoagulant therapy is not necessary for patients in sinus rhythm (after the initial 3 months), in the absence of prior emboli or a hypercoagulable state.

Recertification Evaluation for Asymptomatic Commercial Motor Vehicle Drivers
(Annual clinical evaluation)

The need for an annual echocardiographic evaluation in clinically stable patients remains a matter of some debate. Clearly, echocardiography is indicated in the event that there are concerns about prosthetic valve dysfunction, perivalvular leaks, new murmurs, or left ventricular function. Exercise testing may be required to assess effort tolerance.

MYOCARDIAL DISEASE

HYPERTROPHIC CARDIOMYOPATHY (TABLE 6)

Hypertrophic cardiomyopathy is a complex disease characterized by marked heterogeneity--morphologically, genetically, and prognostically. In the outpatient community setting, overall mortality is in the range of 1.3% per year (70, 71). Many patients experience a benign and stable clinical course, while in others the disease is characterized by sudden death or progressive symptoms.

In some patients, sudden death may be the first definitive manifestation of cardiac disease. In a recent series of patients undergoing implantation of an implantable cardioverter defibrillator for the primary or secondary prevention of sudden cardiac death, 65% were New York Heart Association Class I. In an international population of 744 patients, hypertrophic cardiomyopathy-related deaths occurred in 86 patients (12%) over a follow up period of 8 +/- 7 years. 51% of the deaths were sudden, 36% were due to progressive congestive heart failure, and hypertrophic cardiomyopathy-related stroke (associated with atrial fibrillation) occurred in 13%. The respective age groups were age 45+/- 20 years, 56 +/- 19 years, and 73 +/- 14 years (72).

The five most powerful risk factors for sudden cardiac death are:

1. Nonsustained ventricular tachycardia on Holter monitoring;
2. Abnormal blood pressure fall on exercise testing;
3. Family history of sudden cardiac death;
4. Recurrent, unexplained syncope; and
5. Massive left ventricular hypertrophy (73,74).
   (For the individual patient, however, the sensitivity and specificity is quite markedly limited.)

Initial Evaluation:

1. History;
2. Physical;
3. Electrocardiogram;
4. Chest X-Ray; and
5. 2-Dimensional with Doppler Echocardiography is essential for diagnosis.

The above investigations are adequate for diagnostic purposes in most patients. Occasionally, however, the diagnosis may require additional testing including cardiac catheterization, magnetic resonance imaging, and genotyping. Exercise testing, ambulatory monitoring, and thallium scintigraphy are frequently utilized for the assessment of risk factors for sudden cardiac death and functional capacity. It is essential to differentiate hypertrophic cardiomyopathy from the "athlete's heart." An echocardiogram performed in a laboratory experienced with this disease may be helpful to evaluate diastolic function.

Current Recommendations

Disqualify:

Irrespective of symptoms, a person should not be certified as a CMV driver if a firm diagnosis of hypertrophic cardiomyopathy is made on echocardiography. Patients with a diagnosis of "sigmoid septum" or "borderline hypertrophic cardiomyopathy" or "hypertensive hypertrophic cardiomyopathy" should not be disqualified, but should be re-evaluated after one year.

CONGESTIVE HEART FAILURE AND IDIOPATHIC DILATED CARDIOMYOPATHY (TABLE 6)

Natural History of Congestive Heart Failure

Senni et al. described the natural history of congestive heart failure in a population-based setting and in a cohort of patients in Olmsted County, Minnesota, between 1981 and 1991. The survival rate was poor in both cohorts, with one year mortality 28% and 23% respectively and 66% and 67% at 5 years. Among patients with symptoms of congestive heart failure but preserved systolic function in whom the presumed etiology was diastolic dysfunction, adjusted survival was not significantly different from patients with left ventricular systolic dysfunction (75).

Similarly, in the Studies Of Left Ventricular Dysfunction (SOLVD) Treatment Trial of patients with symptomatic heart failure randomized to enalapril and placebo (among whom the entry ejection fraction was 35% or less), one-year mortality ranged from approximately 10% to 20%, with the lowest mortality in patients with nonischemic cardiomyopathy and the highest in diabetics with ischemic heart disease (76). In the SOLVD Prevention Trial of patients with asymptomatic left ventricular dysfunction, all-cause mortality at one year was approximately 5% and at two years 10%, with approximately one third of the deaths attributed to an arrhythmia without worsening congestive heart failure.

Natural History of Idiopathic Dilated Cardiomyopathy

The majority of patients with systolic dysfunction entering into randomized control trials have coronary artery disease (77). Among patients with a non-ischemic etiology, hypertension is a major cause or associated factor in the development of heart failure. In the majority of the remaining patients who present with systolic dysfunction and no evidence of significant underlying coronary artery disease, the most frequent diagnosis is that of idiopathic dilated cardiomyopathy.

Although the survival of patients with idiopathic dilated cardiomyopathy and congestive heart failure has improved during the last decade (78,79), the natural history of idiopathic dilated cardiomyopathy remains unfavorable, with approximately a 40% five-year mortality in the referral population seen at the Mayo Clinic between 1982 and 1987. Even among a population-based cohort who were seen between 1976 and 1981, the five-year mortality was still in the

range of 15% (78, 80). Among patients with a diagnosis of *asymptomatic* left ventricular dysfunction consistent with idiopathic dilated cardiomyopathy, short-term survival was excellent (100% two-year survival), but long-term survival was poor (78% five-year survival and 53% seven-year survival), which was not very different from a group of patients with *mildly* symptomatic congestive heart failure (81). Among asymptomatic patients with ejection fractions of <0.35 in the SOLVD Prevention Trial, one-year mortality was approximately 4%, whereas it was approximately 15% at five years (76).

In summary, idiopathic dilated cardiomyopathy, despite improvements during the last decade, remains a serious disease with a significant mortality both in the first year after diagnosis and the ensuing five to ten years. Data are lacking, however, in regard to the prognosis of "mild" idiopathic dilated cardiomyopathy characterized by a left ventricular ejection fraction of 40% to 50%.

In commercial motor vehicle drivers, the crucial question relates to the incidence of sudden cardiac death in patients with congestive heart failure and idiopathic dilated cardiomyopathy. Ventricular arrhythmias, and in particular nonsustained ventricular tachycardia, increased from a prevalence of <10% in patients with New York Heart Association functional Class I to 70% among Class IV patients (82). Sudden cardiac death accounts for the majority of deaths in mildly symptomatic patients. Even though the majority of deaths in patients with severe symptoms are due to progressive heart failure, sudden death is responsible for 10 to 30% of all deaths, irrespective of functional class (82).

In mildly symptomatic patients with congestive heart failure or among asymptomatic patients with idiopathic dilated cardiomyopathy, overall mortality over one to five years is quite substantial, and sudden cardiac death is the cause in the majority. The decision to disqualify individuals with symptomatic congestive heart failure or asymptomatic individuals with an ejection fraction of <40% is fairly clear-cut.

Nonetheless, mild abnormalities in left ventricular function are of less certain significance, given the limitations of echocardiography in the measurement of left ventricular ejection fraction and the fact that spontaneous improvement in ejection fraction has been noted over time (18,83). Moreover, there is a lack of natural history data in patients with asymptomatic, mildly diminished left ventricular dysfunction in the absence of coronary artery disease.

Initial Evaluation:

1. History;
2. Physical;
3. ECG;
4. Chest X-Ray;
5. 2-Dimensional and Doppler Echocardiography to assess ejection fraction, left ventricular size, valvular heart disease in the presence of regional wall motion abnormalities. Resting Radionuclide Ventriculography may be indicated for more precise measurements of ejection fraction; and
6. Stress Testing and, in some patients cardiac catheterization, may be necessary to exclude coronary artery disease.

Current Recommendations

These recommendations do not apply to asymptomatic patients with coronary artery disease or hypertension and an ejection fraction of less than 40%. (See sections on Coronary Artery Disease and Hypertension.)

Disqualify:

1. All patients with symptomatic congestive heart failure irrespective of systolic function;
2. Asymptomatic patients with ejection fractions of ≤50% but with ventricular arrhythmias (sustained or nonsustained ventricular tachycardia or symptomatic palpitations); or
3. Asymptomatic patients with an ejection fraction of < 40% (such patients can be re-certified if an annual evaluation demonstrates an improvement in left ventricular ejection fraction to ≥40%).

Recertify:

1. Symptomatic patients without ventricular arrhythmias, with resolution of symptoms, and improvement in ejection fraction as defined by an ejection fraction of ≥40%;
2. Asymptomatic patients without ventricular arrhythmias, with an initial EF of less than 40% in whom the ejection fraction increases to 40% or greater; and
3. Asymptomatic patients without ventricular arrhythmias and an ejection fraction of 40% to 50%.

Annual evaluation is required to assess for improvement or deterioration.

RESTRICTIVE CARDIOMYOPATHY

Very little data is available, but the large study done at the Mayo Clinic indicated a five-year survival of only 64%, compared with an expected survival of 85% (84).

Recommendation
Disqualify all commercial motor vehicle drivers with restrictive cardiomyopathy.

RECOMMENDATION TABLES
MITRAL STENOSIS

TABLE 1

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
Mild Mitral Stenosis MVA $\geq$1.6 cm^2	In the presence of symptoms consistent with moderate to severe mitral stenosis but a calculated valve area suggesting mild mitral stenosis, the severity of the stenosis should be reassessed and an alternative explanation for symptoms should be considered.	Yes, if asymptomatic.	Annual
Moderate Mitral Stenosis MVA 1.0 to 1.6 cm^2		Yes, if asymptomatic.	Annual
Severe Mitral Stenosis MVA $\leq$1.0 cm^2		No if: NYHA Class II or higher; Atrial fibrillation; Pulmonary artery pressure $\geq$50% of systemic pressure; Inability to exercise for >6 Mets on Bruce protocol (Stage II). Yes if: At least 4 weeks post percutaneous balloon mitral valvotomy; or At least 3 months post surgical commissurotomy; Clearance by cardiologist.	 Annual Annual evaluation by a cardiologist.

MVA= mitral valve area

RECOMMENDATION TABLES
MITRAL REGURGITATION

TABLE 2

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
Mild Mitral Regurgitation		Yes if: Asymptomatic; Normal LV size and function;* Normal PAP.	Annual Annual echo not necessary.
Moderate Mitral Regurgitation		Yes if: Asymptomatic; Normal LV size and function; * Normal PAP.	Annual Annual Echocardiogram.
Severe Mitral Regurgitation		Yes, if asymptomatic. Yes if: At least 3 months post-surgery. Asymptomatic; cleared by cardiologist. No if: Symptomatic; Inability to achieve > 6 METS on Bruce protocol; Ruptured chordae or flail leaflet; Atrial fibrillation; LV dysfunction;* Thromboembolism; Pulmonary artery pressure 50% of systolic arterial pressure;	Annual Echocardiogram every 6-12 months. Exercise testing may be helpful to assess symptoms. Annual

EF = Ejection fraction; LVESD = Left ventricular end-systolic dimension
LVEDD = Left ventricular end-diastolic dimension; PAP = Pulmonary artery pressure
*Measures of left ventricular function
LVEF < 60%; LVESD $\geq$ 45mm; LVEDD $\geq$ 70mm

RECOMMENDATION TABLES
AORTIC STENOSIS

TABLE 3

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
Mild Aortic Stenosis (AVA $\geq$ 1.5 cm^2)	If symptoms are consistent with aortic stenosis but calculated valve area suggests mild aortic stenosis, the severity of the stenosis and an alternative explanation for symptoms needs to be reassessed.	Yes, if asymptomatic.	Annual Echocardiogram every 5 years.
Moderate Aortic Stenosis (AVA $\geq$1.0-1.5 cm^2)		Yes, if asymptomatic. Yes if: At least 3 months after surgery. No if: Angina, Heart failure, Syncope; Atrial fibrillation; LV dysfunction with EF <50%; Thromboembolism.	Annual Echocardiogram every 1 to 2 years. Annual
Severe Aortic Stenosis (AVA <1.0 cm^2)		No, irrespective of symptoms or LV function. Yes, if at least 3 months after surgery.	 Annual

AVA = aortic valve area

RECOMMENDATION TABLES
AORTIC REGURGITATION

TABLE 4

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
Mild Aortic Regurgitation		Yes, if asymptomatic.	Annual Echocardiogram every 2 to 3 years.
Moderate Aortic Regurgitation		Yes if: Normal LV function; No or mild LV enlargement.	Annual Echocardiogram every 2 to 3 years.
Severe Aortic Regurgitation		Yes if: Asymptomatic; Normal LV function (EF = 50%); LV dilatation (LVEDD < 60mm, LVESD < 50mm). If LVEDD = 60mm or LVESD = 50mm. No if: Symptoms, Unable to complete Bruce protocol Stage II, Reduced EF < 50%, LV dilatation LVEDD > 70mm or LVESD > 55mm. Yes if: Valve surgery and at least 3 months post surgery. Asymptomatic; cleared by cardiologist.	Every 6 months. Echocardiogram every 6 to 12 months. Every 4 – 6 months. Echocardiogram every 4 – 6 months if no surgery performed. Annual

EF=Ejection fraction
LVESD=Left ventricular end-systolic dimension
LVEDD=Left ventricular end-diastolic dimension

RECOMMENDATION TABLES
VALVE REPLACEMENT

TABLE 5

DIAGNOSIS	PHYSIOLOGY /FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
Mechanical Valves		Yes if: At least 3 months post-op; Asymptomatic; Cleared by cardiologist. No if: Symptomatic; LV dysfunction-EF <40%; Thromboembolic complication post procedure; Pulmonary hypertension; Unable to maintain adequate anticoagulation (based on monthly INR checks).	Annual Recommend evaluation by cardiologist.*
	Prosthetic valve dysfunction.	No Yes if: Surgically corrected; At least 3 months post-op; Asymptomatic; Cleared by cardiologist.	Annual Recommend evaluation by cardiologist.*

RECOMMENDATION TABLES
VALVE REPLACEMENT
(Continued)

TABLE 5

DIAGNOSIS	PHYSIOLOGY /FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
	Atrial fibrillation.	Yes if: Anticoagulated adequately for at least 1 month and monitored by at least monthly INR, rate/rhythm control adequate; Cleared by cardiologist.	Annual
Biologic Prostheses	Antiocoagulant therapy not necessary in patients in sinus rhythm (after initial 3 months), in absence of prior emboli or hypercoagulable state.	Yes if: At least 3 months post-op; Asymptomatic; None of above disqualifying criteria for mechanical valves; Cleared by cardiologist.	Annual Recommend evaluation by cardiologist.*

***** Role of annual echocardiography in stable patients is controversial.

RECOMMENDATION TABLES
CARDIOMYOPATHIES AND CONGESTIVE HEART FAILURE

Table 6

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL STATUS	CERTIFICATION	RE-CERTIFICATION
Hypertrophic Cardiomyopathy		No	
Idiopathic Dilated Cardiomyopathy and Congestive Heart Failure		No, if symptomatic CHF.	
		No if: Asymptomatic; Ventricular arrhythmias present; and LVEF ≤ 50%.	
		No if: Asymptomatic; No ventricular arrhythmias but LVEF < 40%.	
		Yes if: Asymptomatic; No ventricular arrhythmias; and LVEF 40% to 50%.	Annual Requires annual cardiology evaluation including Echocardiography and Holter monitoring.
Restrictive cardiomyopathy		No	

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Cardiac Arrhythmias, Pacemakers, Implantable Defibrillators

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ARRHYTHMIA

Background

There are two major considerations for the medical examiner when certifying a CMV driver who has a history of an arrhythmia. First, CMV drivers with arrhythmias and those treated with antiarrhythmia devices should not be certified if they are at risk for cerebral hypoperfusion and impaired consciousness. In the worst circumstance, loss of consciousness is due to a fatal arrhythmia, such as ventricular fibrillation or hypotensive ventricular tachycardia. Secondly, the examiner should search for any underlying heart disease that could be disqualifying. For example, an arrhythmia is the most likely cause of sudden death (1), but coronary heart disease (CHD) is the most likely underlying etiology (2). (For additional information, please refer to the Ischemic Heart Disease section).

Risk of Arrhythmia

The medical examiner must determine the risk of a driver developing an arrhythmia. Risk determination is made all the more difficult since the number of variables that must be considered makes it almost impossible to estimate the probability of a driver with CHD experiencing sudden, unexpected arrhythmic death or incapacitation and losing control of their vehicle. For example, patients with ventricular arrhythmias may have concomitant CHD and left ventricular dysfunction that compound the risk for an adverse outcome. Second, instantaneous cardiac death caused chiefly by ventricular fibrillation is often not due to a progression of CHD, but rather is an electrical accident. Although defibrillation may restore a normal rhythm, there is a high risk for recurrence (3).

When the term "symptomatic" is used, it refers to significant complaints suggesting cerebral hypoperfusion (e.g., profound dizziness with near loss of consciousness, complete loss of consciousness), and not more nonspecific complaints such as "flip flops" without hemodynamic compromise. The decision to regard symptoms as major or trivial rests on a carefully recorded history and interpretation by an expert in the evaluation of cardiovascular disease and arrhythmias.

Driving and Electrocardiographic Changes

Driving an automobile can be a source of significant mental stress (4,5,6). Consequently, there can be an adverse effect on cardiac function among drivers with CHD (7). In 1967, Taggart and Gibbons described the effects on nine healthy subjects driving in dense, fast moving city traffic. Heart rates ranged from 70-85 beats per minute (bpm) at rest to 100-140 bpm while driving (8). In a follow-up paper two years later, amateur drivers, 32 normal and 24 with CHD, were monitored during a 20-minute drive in heavy London traffic. There were significant ST-T changes in three of the normal subjects, not secondary to a more rapid heart rate. In the 24 drivers with CHD, 13 had increased ST-T abnormalities, with T wave changes occurring in six; five developed multiple premature ventricular contractions (PVCs), of which some were multifocal; one driver had a short run of ventricular tachycardia. Two other drivers with CHD experienced angina and two

developed left ventricular failure. The authors concluded that persons with easily provoked angina or borderline left ventricular failure should be advised not to drive (9).

Twenty-two drivers with stable heart disease who were wearing Holter monitors drove into a police radar trap. All patients reported cardiac symptoms and their Holter monitors recorded increased heart rates, ventricular arrhythmias, and, in some cases, myocardial ischemia. The authors believed that the data confirmed the effects of stress on adrenergic tone (10).

Supraventricular Arrhythmias

Supraventricular tachycardia is the generic term for paroxysmal, regular and rapid supraventricular arrhythmias (11). There are a variety of supraventricular tachycardias that are not usually considered a great risk for sudden arrhythmic death. They rarely cause loss of consciousness or impairment of cerebral function, but on occasion can do so, thereby increasing the risk for a crash. The usual mechanism for paroxysmal supraventricular tachycardia is AV nodal reentry (due to dual AV nodal pathways) or atrioventricular re-entry (due to an accessory pathway directly connecting the atria to the ventricles). When there is electrocardiographic evidence for an accessory pathway on the surface electrocardiogram, the term Wolff-Parkinson-White Syndrome (WPW) is used.

The most common supraventricular arrhythmia seen in older individuals is atrial fibrillation, characterized by rapid, disorganized asynchronous contraction of the atrial muscle with an irregular ventricular response (12-14). The major risk associated with this arrhythmia is peripheral embolization, most commonly stroke (12-15). Atrial flutter is a regular supraventricular tachycardia that uses the tricuspid valve annulus as a circuit for reentry. Like atrial fibrillation, it is usually seen in older individuals but can occur at any age (12,13).

Regular supraventricular tachycardias, including atrial flutter, are often managed with curative therapy in the form of catheter ablation (16-18). In these procedures, catheters are positioned in the heart, and the origin or vulnerable portion of the arrhythmia in question is identified and destroyed with radiofrequency energy, commonly called cautery. These procedures are usually curative and allow drug therapy to be withdrawn.

Atrial fibrillation is oftentimes more problematic than regular supraventricular tachycardias since the sequelae of disorganized atrial contraction is the development of atrial thrombi that can become dislodged and cause strokes and peripheral emboli. Thus, patients with risk factors for stroke (age >65 years; prior stroke, systemic embolism, or transient ischemic attack; diabetes; hypertension; left ventricular ejection fraction <0.40; congestive heart failure; or left atrial size ≥50 mm) are anti-coagulated, optimally with warfarin (12-14). This strategy has been shown to decrease the risk of peripheral embolization by 60% to 70% (19,20). Other than in the setting of stroke, atrial fibrillation rarely impairs consciousness and the ability to drive.

However, when atrial fibrillation occurs in the presence of the Wolff-Parkinson-White Syndrome, conduction to the ventricles can be rapid and lead to hemodynamic collapse. For this reason, when atrial fibrillation occurs in patients with an accessory pathway, curative therapy with catheter ablation of the pathway is generally recommended.

Furthermore, ablation of accessory pathways often leads to resolution of atrial fibrillation.

Ventricular Arrhythmias

The majority of sudden cardiac deaths are thought to be secondary to ventricular tachycardia or fibrillation. Furthermore, they occur most often in persons with no prior knowledge or diagnosis of heart disease (21). Ventricular fibrillation is the rapid disorganized and asynchronous contraction of the ventricles. On the surface electrocardiogram, it is characterized by the absence of clearly defined electrical activity and usually represents the final common pathway for death in patients who experience cardiac arrest. Ventricular tachycardia is an arrhythmia originating in the ventricles, most often due to scar resulting from myocardial infarction. However, both ventricular tachycardia and fibrillation can occur in patients with structurally normal hearts.

The American Heart Association estimates that each year in the United States approximately 220,000 deaths due to CHD occur outside the hospital, most of which are sudden and presumed arrhythmic in origin (22). The prevalence is variable and depends on the presence or absence of structural heart disease. Prognosis is generally determined by the underlying heart disease: patients with structurally normal hearts have better prognoses than those with structurally abnormal hearts (23,24).

Nevertheless, patients with ventricular fibrillation and normal mechanical function, for example from genetic disorders such as the Brugada and Long QT syndromes, have guarded prognoses (25-29). Furthermore, patients with ventricular tachycardia and fibrillation in the setting of non-ischemic cardiomyopathies (i.e., hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, and congenital heart disease) also have unpredictable long-term outcomes (30,31). In contrast, patients with ventricular tachycardia originating in the right ventricular outflow tract or from the left ventricle in the absence of structural heart disease have excellent prognoses (32,33). Despite these general statements, it must be recognized that irrespective of the underlying heart disease, syncope as a consequence of an arrhythmia while driving, places the driver and others around the driver at the time in serious jeopardy.

In addition to arrhythmia management, treatment of the underlying heart disease, if present, is of paramount importance. Patients with CHD and left ventricular dysfunction, ischemia and heart failure should be aggressively managed. For patients with ventricular tachycardia and structurally normal hearts, curative therapy can often be offered in the form of catheter ablation (34). If these procedures are successful, drugs often can be withdrawn and patients should have a normal life expectancy. On the other hand, when ventricular tachycardia or fibrillation occurs in the setting of structural heart disease, especially CHD, implantation of a defibrillator is usually recommended because of its proven survival benefit (35,36). Finally, drugs are available that are effective in managing ventricular arrhythmias. Although they are designed to prevent occurrences, they are not "fail-safe" and if arrhythmia recurs, syncope may follow.

Bundle Branch Blocks and Hemiblocks

The significance of bundle branch block is that progression of disease in the conduction system can lead to third degree heart block with total loss of electrical connection between the atria and ventricles. Syncope of sudden death can occur when the heart block prevents adequate ventricular contraction. The hemiblocks are not associated with a marked increase in progression to complete heart block or with sudden arrhythmic death. The risk is much higher when a hemiblock is combined with RBBB.

No single clinical or electrocardiographic finding can determine the risk for future syncope or death from a bradyarrhythmia due to conduction system disease. Death in persons with conduction system disease is often neither sudden nor due to AV block, but rather from underlying heart disease and non-arrhythmic cardiac causes. However, because of underlying heart disease, the risk for syncope and sudden and non-sudden death remains.

Certification of CMV drivers with asymptomatic bundle branch block or an axis deviation depends on the risk from underlying disease. Conduction system disease that caused syncope must be treated before a driver is certified. Bradycardia can be treated with a pacemaker. However, certification of drivers who have been symptomatic from conduction system disease also depends on the risk from their underlying heart disease.

Pacemakers

A pacemaker is an implantable device designed to treat bradycardia. The indications for implantation are detailed in the American College of Cardiology/American Heart Association guidelines (presently being updated) (36). Over one million people in the United States have pacemakers; more than 435 new implantations per 1 million people occur each year. Currently, pacemakers and their lead systems are reliable and durable over the long-term.

When assessing the risk for sudden, unexpected incapacitation in a patient with a pacemaker, the underlying disease responsible for the pacemaker indication must be considered. If patients have had cerebral symptoms or syncope from bradycardia, symptoms generally resolve after pacemaker implantation. Even when patients have neurocardiogenic syncope with both vasodepressor and negative chronotropic components, maintaining the heart rate can attenuate the vasodepressor aspect (37, 38).

Implantable Cardioverter-Defibrillators

The Implantable Cardioverter-Defibrillators (ICDs) are electronic devices that treat cardiac arrest (ventricular fibrillation) and ventricular tachycardia by the delivery of rapid pacing stimuli or shock therapy. The particular therapy delivered depends on programming and characteristics of the arrhythmia identified by the device. These devices do not prevent arrhythmias but only treat them when they occur. Thus, patients remain at risk for syncope.

ICDs can be implanted for both primary as well as secondary prevention (35,36). Since these devices do not prevent arrhythmias, patients who have had an arrest or hemodynamically important ventricular tachycardia, and who have received ICD implantation should not be certified for commercial driving. Similarly, when patients receive a defibrillator for primary prevention (e.g., they never have had a sustained arrhythmia but are deemed at risk for having one due to electrophysiologic testing or other evaluation), the risk of loss of consciousness is considerable. For this reason, patients with defibrillators implanted for primary prevention are similarly unfit for certification to drive commercially.

Arrhythmias and Syncope

Syncope in a commercial driver can cause loss of control of the vehicle. It is necessary to differentiate cardiac-based syncope from other causes of syncope. Syncope from cardiac causes may herald a markedly increased future risk of sudden death. Fitzpatrick reported that out of 332 patients with recurrent syncope evaluated over 15 years, A-V block was found in 34%, sinus node disease in 21%, carotid sinus syndrome in 10%, and inducible sustained tachyarrhythmias in 6%. A large percentage of the remaining 29% had a diagnosis of malignant vasovagal syndrome (39).

RECOMMENDATION TABLES
SUPRAVENTRICULAR TACHYCARDIAS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Atrial Fibrillation			
Lone Atrial Fibrillation	Good prognosis and low risk for stroke.	Yes	Annual
Atrial Fibrillation as cause of or a risk for stroke	Risk for stroke decreased by anticoagulation.	Yes if: Anticoagulated adequately for at least 1 month; Anticoagulation monitored by at least monthly INR; Rate/rhythm control deemed adequate (Recommend assessment by cardiologist).	Annual
Atrial fibrillation following thoracic surgery	Good prognosis and duration usually limited.	In atrial fibrillation at time of return to work; Yes if: Anticoagulated adequately for at least 1 month; Anticoagulation monitored by at least monthly INR; Rate/rhythm control deemed adequate (Recommend assessment by cardiologist).	Annual

RECOMMENDATION TABLES
SUPRAVENTRICULAR TACHYCARDIAS
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Atrial flutter	Same as for atrial fibrillation.	Same as for atrial fibrillation. Yes if: Isthmus ablation performed and at least 1 month after procedure; Arrhythmia successfully treated; Cleared by electrophysiologist.	Same as for atrial fibrillation. Annual
Multifocal Atrial Tachycardia	Often associated with comorbidities, such as lung disease, that may impair prognosis.	Yes if: Asymptomatic; Unless associated condition is disqualifying. No, if symptomatic. Yes if: Symptoms controlled and secondary cause is not exclusionary.	Annual Annual.
Atrioventricular Nodal Reentrant Tachycardia (AVNRT) Atrioventricular Reentrant Tachycardia (AVRT) and Wolff-Parkinson-White (WPW) Syndrome Atrial Tachycardia Junctional Tachycardia	Prognosis generally excellent, but may rarely have syncope or symptoms of cerebral hypoperfusion. For those with WPW, preexcitation presents risk for death or syncope if atrial fibrillation develops.	No if: Symptomatic; or WPW with atrial fibrillation. Yes if: Asymptomatic; Treated and asymptomatic for at least 1 month and assessed and cleared by expert in cardiac arrhythmias.	Annual Recommend consultation with cardiologist.

RECOMMENDATION TABLES
VENTRICULAR ARRHYTHMIAS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Coronary Heart Disease (CHD)	Sustained VT: Poor prognosis and high risk.	No	
	NSVT, LVEF <0.40: Unfavorable prognosis.	No	
	NSVT, LVEF ≥0.40: Generally considered to have good prognosis.	No, if symptomatic.	
		Yes if: Asymptomatic. At least 1 month after drug or other therapy is successful; Cleared by cardiologist.	Annual Cardiology examination required. .
Dilated Cardiomyopathy	NSVT (LVEF ≤0.40).	No	
	Sustained VT, any LVEF.	No	
	Syncope/near syncope, any LVEF: High risk.	No	
Hypertrophic Cardiomyopathy	Variable but uncertain prognosis.	No	

RECOMMENDATION TABLES
VENTRICULAR ARRHYTHMIAS
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Right Ventricular Outflow VT	Favorable prognosis and low risk for syncope.	No, if symptomatic. Yes, if asymptomatic. Yes if: At least 1 month after drug or other therapy successful; Asymptomatic; Cleared by electrophysiologist.	 Annual Recommend evaluation by cardiologist. Annual Evaluation by cardiologist required.
Idiopathic Left Ventricular VT	Favorable prognosis and low risk for syncope.	No, if symptomatic Yes, if asymptomatic. Yes if: At least 1 month after successful drug therapy or ablation; Cleared by electrophysiologist.	 Annual Recommend evaluation by cardiologist. Annual Evaluation by cardiologist required.
Long QT Interval Syndrome	High risk for ventricular arrhythmic death.	No	
Brugada Syndrome	High risk for ventricular arrhythmic death.	No	

EF = ejection fraction
LV = left ventricular
NSVT = nonsustained ventricular tachycardia
VT=ventricular tachycardia

RECOMMENDATION TABLES
BUNDLE BRANCH BLOCKS AND HEMIBLOCKS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Bundle Branch Block Axis Deviation	Progression of disease in the conduction system can lead to third degree heart block with total loss of electrical connection between the atria and ventricles causing syncope or sudden death.	Yes if: Asymptomatic. (Depends on risk from underlying heart disease.) Yes, if treated for symptomatic disease (see pacemaker); no disqualifying heart disease; and cleared by cardiologist. No, if symptomatic.	Every 2 years. Annual

RECOMMENDATION TABLES
PACEMAKERS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Sinus node dysfunction	Variable long term prognosis depending on underlying disease, but cerebral hypoperfusion corrected by support of heart rate by pacemaker.	No Yes if: 1 month after pacemaker implantation; and documented correct function by pacemaker center. Underlying disease is not disqualifying.	Annual Documented pacemaker checks.
Atrioventricular (AV) block	Variable long term prognosis depending on underlying disease, but cerebral hypoperfusion corrected by support of heart rate by pacemaker.	No Yes if: 1 month after pacemaker implantation and documented correct function by pacemaker center, and underlying disease is not disqualifying.	Annual Documented pacemaker checks.

RECOMMENDATION TABLES
PACEMAKERS
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Neurocardiogenic syncope	Excellent long-term survival prognosis but there is risk for syncope that may be due to cardioinhibitory (slowing heart rate) or vasodepressor (drop in blood pressure) components, or both. Pacemaker will affect only cardioinhibitory component, but will lessen effect of vasodepressor component.	No, with symptoms. Yes if: 3 months* after pacemaker implantation; and documented correct function by pacemaker center; Absence of symptom recurrence.	Annual Documented pacemaker checks; Absence of symptom recurrence.

* Three months recommended due to possible vasodepressor component of syndrome not necessarily treated by pacing.

RECOMMENDATION TABLES
PACEMAKERS
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Hypersensitive carotid sinus with syncope	Excellent long-term survival prognosis but there is risk for syncope that may be due to cardioinhibitory (slowing heart rate) or vasodepressor (drop in blood pressure) components, or both. Pacemaker will affect only cardioinhibitory component, but will lessen effect of vasodepressor component.	No, with symptoms. Yes if: 3 months* after pacemaker implantation; and documented correct function by pacemaker center; Absence of symptom recurrence.	 Annual Documented regular pacemaker checks; and Absence of symptom recurrence.

*Three months recommended due to possible vasodepressor component of syndrome not necessarily treated by pacing.

RECOMMENDATION TABLES
IMPLANTABLE DEFIBRILLATORS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Primary prevention	Patient has high risk for death and sudden incapacitation.	No	
Secondary prevention	Patient demonstrated to have high risk for death and sudden incapacitation.	No	

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Congenital Heart Disease

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CONGENITAL HEART DISEASE

Introduction

Due to advances in surgical and medical management, over 85% of infants born with congenital heart diseases are expected to survive to adult life. This has led to a dramatic increase in the population of adults with congenital heart disease in the United States (1). The number of individuals with congenital heart disease requesting commercial driver certification is expected to rise proportional to the increasing patient population. Recommendations for medical certification of commercial motor vehicle drivers are based on the natural history of individuals with congenital heart disease, the early and late results of surgery, and the risk of sudden death. Progressive heart failure and sudden death account for the majority of deaths among patients with congenital heart disease. Certification of drivers with operated congenital heart disease will depend on serial evaluation of existing cardiac residua and sequelae.

Diagnostic Evaluation

It is not likely that the medical examiner will conduct the testing outlined in this section. However, the review of the testing requirements provides the examiner the information to assess whether the driver has been adequately evaluated, and to classify the severity of the disease as it relates to the driver's general health and ability to be medically certified.

Congenital heart disease should not automatically disqualify applicants from commercial driver medical certification. This decision should be based on the anatomic diagnosis, evaluation of the severity of the congenital defect, and the results of treatment. The decision for certification should consider the applicant's present status and the possibility of functional impairment. When the history or examination suggests the presence of a congenital cardiac anomaly, the applicant should be referred for further cardiac evaluation. Due to the complexity of these problems, the guidelines recommend that a cardiologist knowledgeable in adult congenital heart disease define the diagnosis, severity, and prognosis (2,3). The diagnostic evaluation should use appropriate noninvasive studies and invasive techniques when necessary.

A non-invasive evaluation should include a complete history and physical examination, 12-lead electrocardiogram, chest roentgenogram and comprehensive transthoracic two-dimensional and Doppler echocardiogram. By providing anatomic detail and hemodynamic information, echocardiography can often replace the need for cardiac catheterization (4). Among individuals considered at risk for the development of a dysrhythmia, ambulatory electrocardiographic monitoring at rest and during exercise should be carried out (5,6). Exercise tolerance testing is useful in certain cases of significant congenital heart disease in an effort to determine functional status and the presence or absence of exercise related arrhythmias (7,8).

Individuals who have had surgery to correct a congenital cardiac defect should have testing to evaluate cardiac function and to elicit cardiac rhythm disturbances. Intracardiac electrophysiologic studies may be necessary to evaluate those patients with symptoms or findings suggestive of a potentially life-threatening arrhythmia. In selected cases, hemodynamic cardiac catheterization should be performed to clarify discrepant or incomplete diagnostic information obtained by non-invasive measures.

Overview of Certification Guidelines

Applicants for certification are likely to be those with milder forms of congenital heart disease for whom surgery is not indicated or in whom the condition has spontaneously resolved, for example spontaneous closure of a ventricular septal defect. Persons who have had surgical repair of a malformation may apply for commercial driver certification.

Except for certain specific instances, the presence of a prosthetic device, such as a mechanical or biological heart valve, does not by itself disqualify a person from commercial driver certification. The result of surgery, not the presence of prosthetic material, should determine eligibility for driving status. These individuals also require regular, informed cardiovascular follow-up in order to maintain certification. Repeat cardiac surgical intervention is often required for cardiac residua and sequelae.

While the following congenital defects are the most common, drivers with other congenital defects may need to be evaluated for fitness to drive. Criteria for evaluating applicants with the reviewed congenital cardiovascular anomalies should serve as guidelines for assessing individuals with other forms of congenital heart disease. For postoperative applicants, a minimal waiting period of three months following cardiac surgery is recommended for clinical and hemodynamic evaluation of results.

BICUSPID AORTIC VALVE

A bicuspid aortic valve occurs in 1% to 2% of the population and is more common in men than women. A bicuspid aortic valve may result in aortic stenosis or regurgitation. These disorders are outlined in the section on Valvular Diseases.

Individuals with bicuspid aortic valves are predisposed to aortic root enlargement and aortic aneurysm formation. Until satisfactory surgical intervention has been performed, the commercial driver should be disqualified if the aortic transverse diameter is over 5.5 cm, because of the risk of rupture.

MARFAN SYNDROME

Marfan syndrome is an autosomal dominant connective tissue disorder. Cardiovascular complications are the most common causes of death in individuals with Marfan syndrome. The primary concern is that of aortic root enlargement with the potential

complications of aortic dissection or rupture. Aortic valve regurgitation may occur as a result of aortic root enlargement. Mitral valve prolapse and mitral valve regurgitation are important cardiovascular manifestations that may also occur (9).

Disqualify:

1. Any aortic root enlargement;
2. Moderate or more severe aortic valve regurgitation;
3. More than mild mitral valve regurgitation related to mitral valve prolapse; or
4. Left ventricular dysfunction (with no associated valvular lesion) with an ejection fraction (EF) less than 40%.

SUBVALVULAR AORTIC STENOSIS

Individuals with discrete or diffuse mild subaortic left ventricular outflow tract obstruction without associated valvular abnormalities or hypertrophic cardiomyopathy may be certified for commercial driving. In most individuals, the severity of subvalvular aortic stenosis can be defined by Doppler echocardiographic measurement of a peak and mean pressure gradient, as discussed in the ACC/AHA Guidelines for the Clinical Application of Echocardiography (10).

Disqualify:

1. Symptoms of dyspnea, chest pain or syncope;
2. More than moderate aortic valve regurgitation; or
3. A mean pressure gradient across the left ventricular outflow tract greater than 30 mmHg in the setting of normal cardiac output (11).

Commercial driving is not recommended when the resting obstruction is moderate or severe. Surgical intervention may be required. Individuals may be certified three months after successful surgery provided they meet the criteria outlined above. Because of postoperative complications and the risk of recurrence, an annual cardiovascular evaluation, including comprehensive echocardiography, is required after resection of subaortic stenosis (12,13)

DISCRETE SUPRAVALVULAR AORTIC STENOSIS

Persons with uncorrected supravalvular aortic stenosis do not qualify for certification as a commercial motor vehicle driver due to the risk of aortic and coronary complications (14,15). After surgical correction, drivers may be certified if they meet the criteria described for persons following correction of valvular and subvalvular aortic stenosis (16,17).

ATRIAL SEPTAL DEFECT

A patent foramen ovale is present in nearly 30% of normal adults (18). The presence of a patent foramen ovale does not preclude CMV certification in the absence of complications, such as a history of paradoxical embolus.

The size and hemodynamic consequence of the atrial septal defect can often be determined by transthoracic echocardiography. Rarely, transesophageal echocardiography is required. Risk of a sudden incapacitating event due to the presence of such an isolated atrial septal defect is very rare in the absence of an arrhythmia, hypercoagulable state (increasing the risk of paradoxical embolism) or pulmonary hypertension.

ATRIAL SEPTAL DEFECT: OSTIUM SECUNDUM

Disqualify:

1. Symptoms of dyspnea, palpitations or a paradoxical embolus;
2. Echo-Doppler examination demonstrating pulmonary artery pressure greater than 50% systemic;
3. Echo-Doppler examination demonstrating a right-to-left shunt; or
4. Pulmonary to systemic flow ratio greater than 1.5 to 1.

These individuals have an increased risk of sudden death or incapacitation. If clinical and echocardiographic findings are discordant, hemodynamic cardiac catheterization may be required. After certification, annual cardiovascular and echocardiographic evaluation by a cardiologist with expertise in adult congenital heart disease is necessary for renewal of the commercial driving certificate in the presence of an unrepaired secundum atrial septal defect.

Persons who have had surgical or device closure of a secundum atrial septal defect may be certified at least 3 months after surgery or at least 4 weeks after device closure if they do not have any of the disqualifying criteria outlined above. In addition, these individuals should have no symptomatic arrhythmia and no significant residual shunt. After atrial septal defect closure, patients have an excellent prognosis with little evidence to suggest subsequent cardiac deterioration (19). Evaluation by a cardiologist should be performed every two years, due to the risk of developing cardiovascular complications such as atrial fibrillation, which may require intervention.

ATRIAL SEPTAL DEFECT: OSTIUM PRIMUM

Disqualify:

1. Symptoms of dyspnea, palpitations or a paradoxical embolus;

2. Echo-Doppler examination demonstrating pulmonary artery pressure greater than 50% systemic;
3. Echo-Doppler examination demonstrating a right-to-left shunt;
4. A pulmonary to systemic flow ratio greater than 1.5 to 1;
5. Heart block on an electrocardiogram;
6. More than mild mitral valve regurgitation; or
7. Left ventricular outflow tract obstruction with a gradient greater than 30 mmHg.

The mitral valve is often cleft in individuals with an ostium primum atrial septal defect; and mitral valve regurgitation eventually occurs in these individuals. Left ventricular outflow tract obstruction and conduction system disease may also occur over time in individuals with this type of atrial septal defect. Annual cardiovascular and echocardiographic evaluation by a cardiologist knowledgeable in adult congenital heart disease is necessary for commercial driving re-certification.

Following surgical correction, commercial drivers may be (re)-certified after three months if they do not have any of the disqualifying factors noted above. In addition, these individuals should have no symptomatic arrhythmia and no significant residual shunt. For all such persons, annual evaluation by a cardiologist knowledgeable in adult congenital heart disease is necessary, due to the risk of cardiovascular complications that may require intervention (20).

ATRIAL SEPTAL DEFECT: SINUS VENOSUS DEFECT

Sinus venosus atrial septal defect is an uncommon type of atrial septal defect. Most individuals have associated anomalous pulmonary venous connection, which increases the degree of left-to-right shunt. The diagnosis is often suggested by transthoracic echocardiography, and confirmed by transesophageal echocardiography.

Disqualify:

1. Symptoms of dyspnea, palpitations or a paradoxical embolus;
2. Echo-Doppler examination demonstrating pulmonary artery pressure greater than 50% systemic;
3. Echo-Doppler examination demonstrating a right-to-left shunt;
4. A pulmonary to systemic flow ratio greater than 1.5 to 1; or
5. Heart block or sinus node dysfunction on an electrocardiogram.

In the setting of appropriate hemodynamic and functional status, commercial driving certification is feasible three months after repair of sinus venosus atrial septal defect, assuming the absence of the disqualifying factors noted above. Cardiovascular evaluation by a cardiologist with special interest in adult congenital heart disease should be performed annually and include ambulatory heart rate monitoring because of the risk of sinus node dysfunction. Sinus node dysfunction is common after surgical repair of this type of atrial septal defect.

VENTRICULAR SEPTAL DEFECT

Ventricular septal defects may be located anywhere on the ventricular septum. For purposes of commercial driver certification, the size and hemodynamic consequence of the ventricular septal defect is important. The size of the VSD can be measured by echocardiography or angiography. In this review, the size is based on comparison to the aortic valve annulus. Those that are about the same size as the annulus are considered large, those half the diameter of the aortic root are considered moderate, and those less than half the aortic root size are considered small (21).

In general, small ventricular septal defects have little clinical impact, except for the need for endocarditis prophylaxis. Moderate or large size ventricular septal defects may have a significant effect on pulmonary pressure and also on ventricular size and function.

Disqualify:

1. Symptoms of dyspnea, palpitations or syncope;
2. Echo-Doppler examination demonstrating pulmonary artery pressure greater than 50% systemic;
3. Echo-Doppler examination demonstrating a right-to-left shunt, left ventricular enlargement or reduced function; or
4. A pulmonary to systemic flow ratio greater than 1.5 to 1.

Cardiac catheterization is only required in individuals with discrepant clinical and echocardiographic data, or in individuals with indeterminate pulmonary pressure or findings of pulmonary hypertension. Persons who undergo spontaneous closure or a clinically significant reduction in the size of a ventricular septal defect may qualify for certification if they do not have any of the disqualifying criteria listed above (22,23).

Individuals with a moderate sized ventricular septal defect should not be certified prior to closure due to the risk of sudden incapacitation from a paradoxical embolism or progressive pulmonary hypertension. In addition, these individuals are prone to progressive left ventricular dilation and dysfunction.

Certification is possible three months after successful surgical closure with:

1. Absence of the disqualifying criteria outlined above;
2. QRS complex duration less than 120 milliseconds on the electrocardiogram; or
3. No serious dysrhythmia on 24-hour ambulatory electrocardiographic monitoring.

Cardiovascular evaluation and ambulatory monitoring should be repeated annually. If the right ventricular conduction delay is greater than 120 milliseconds on the electrocardiogram, the driver may still be certified if invasive HIS bundle studies show no infra-HIS block or other serious electrophysiologic disorders indicating a high-risk for incapacitation, as determined by a cardiologist with expertise in cardiac electrophysiology (23,24).

PATENT DUCTUS ARTERIOSUS

A patent ductus arteriosus results in left to right shunting at the pulmonary artery level. A small communication has no clinical or hemodynamic implication, except for requiring endocarditis prophylaxis. A moderate or large patent ductus arteriosus may have important impact on pulmonary artery pressure as well as ventricular size and function.

Disqualify:

1. Symptoms of dyspnea or palpitations;
2. Echo-Doppler examination demonstrating pulmonary artery pressure greater than 50% systemic; or
3. Echo-Doppler examination or clinical evaluation demonstrating a right-to-left shunt, left ventricular enlargement or reduced systolic function.

These individuals do not have an increased risk of sudden incapacitation. A driver may be certified three months after their patent ductus has been surgically closed or one month after their patent ductus has been device closed, if the results of the electrocardiogram and echo-Doppler examination demonstrate none of the disqualifying features noted above at the time of re-evaluation (25,26).

COARCTATION OF THE AORTA

Persons with uncorrected coarctation of the aorta have a shortened life expectancy, with an average age of 35 years at death (27). An individual with a history of repaired or unrepaired coarctation is at risk for developing serious cardiovascular complications, including intracranial hemorrhage, aortic dissection, aortic rupture and congestive heart failure. Systemic hypertension occurs in over 70% of individuals after repair of coarctation. In 25% the hypertension is severe.

Magnetic resonance imaging is an excellent noninvasive diagnostic tool to evaluate the location and severity of coarctation, to determine the presence of collateral vessels, and to detect the presence of potential complicating features such as aneurysm formation. Bicuspid aortic valve occurs in over 50% of individuals with coarctation of the aorta. Because each of these lesions can be progressive, annual cardiovascular examination is necessary for re-certification.

Disqualify:

1. Symptoms of dyspnea or angina;
2. Coarctation gradient greater than 20 mmHg noted by echo-Doppler examination or cardiac catheterization;
3. Coarctation diameter narrowing $\leq$ 50% of the aortic diameter at the level of the diaphragm;
4. Echo-Doppler examination or clinical evaluation demonstrating

more than mild left ventricular hypertrophy or mild left ventricular dysfunction;
5. More than mild aortic valve regurgitation by echocardiography;
6. More than mild aortic valve stenosis with a mean gradient by echocardiography > 30 mm Hg with a normal cardiac output;
7. Aortic diameter greater than 5.5 cm at any location;
8. Findings consistent with an intracranial aneurysm; or
9. Uncontrolled hypertension (see Hypertension section).

Individuals whose aortic coarctation has been corrected surgically may be certified only if they meet all criteria outlined above, have less than a 20 mmHg systolic pressure gradient across the coarctation repair, and no evidence of aortic aneurysm or pseudoaneurysm formation. Due to the documented reduction in survival in individuals with a history of coarctation repair compared to the general population and the high risk of sudden cardiovascular events, certification after coarctation repair is uncommon and individuals in this group should be evaluated carefully and certified with great caution pending further data (28).

PULMONARY VALVE STENOSIS

Pulmonary valve stenosis is usually a well-tolerated cardiac lesion and gradual progression is the rule. Sudden incapacitation related to pulmonary valve stenosis is uncommon but may occur in certain circumstances.

Disqualify:

1. Symptoms of dyspnea, palpitations or syncope;
2. Pulmonary valve peak gradient greater than 50 mmHg in the presence of a normal cardiac output;
3. Right ventricular pressure > 50% systemic pressure;
4. More than mild right ventricular hypertrophy noted by echocardiography;
5. More than mild right ventricular dysfunction noted by echocardiography;
6. More than moderate pulmonary valve regurgitation noted by echocardiography; or
7. Main pulmonary artery diameter over 5 cm by echocardiography or other imaging modality.

Such individuals are at risk for sudden incapacitation (29). Intervention is indicated when the peak pulmonary valve gradient is over 50 mmHg or the patient is symptomatic. Pulmonary balloon valvuloplasty is the treatment of choice and compares favorably with surgical valvotomy (30). CMV drivers may be certified if they meet the criteria outlined above at least three months following surgical valvotomy or one month post-balloon valvuloplasty (31).

Other causes of right ventricular outflow obstruction may be encountered in persons with congenital heart disease, such as double chambered right ventricle, infundibular

pulmonary stenosis, supravalvar pulmonary stenosis and pulmonary artery stenosis. The certification of individuals with these disorders is possible. Hemodynamic data and criteria should be similar to individuals with isolated pulmonary valve stenosis who are eligible for certification.

EBSTEIN ANOMALY

The natural history of patients with Ebstein anomaly depends on its severity (32). The diagnosis is made by echocardiography. Adults with a mild form of Ebstein anomaly can remain asymptomatic throughout their lives. With moderate tricuspid valve deformity and dysfunction, individuals usually develop symptoms during late adolescence or early adult life. Individual assessment is imperative. Over 50% of individuals with Ebstein anomaly have an intra-atrial shunt. Twenty percent of individuals with Ebstein anomaly have one or more accessory conduction pathways.

Disqualify:

1. Symptoms of dyspnea, palpitations or paradoxical embolism;
2. More than mild cardiac enlargement;
3. Associated intracardiac lesions such as atrial septal defect, ventricular septal defect or pulmonary valve stenosis;
4. More than mild right ventricular dysfunction noted by echocardiography;
5. More than moderate tricuspid valve regurgitation noted by echocardiography;
6. History of symptomatic arrhythmia or accessory conduction pathway noted on electrocardiogram;
7. Right to left shunt; or
8. Left to right shunt with a pulmonary to systemic flow ratio >1.5 to 1.

Certification is possible after surgical intervention for Ebstein anomaly, assuming the individual has none of the disqualifying features outlined above. Annual cardiovascular re-evaluation is required for certification of commercial drivers with Ebstein anomaly, which should include echocardiography and evaluation by a cardiologist knowledgeable in adult congenital heart disease.

TETRALOGY OF FALLOT

Without surgical intervention, few individuals with tetralogy of Fallot survive beyond the second decade of life (33). Persons with uncorrected tetralogy of Fallot do not qualify for commercial driving certification.

Most adults with tetralogy of Fallot have undergone one or more palliative procedures and subsequent intracardiac repair. Complications may arise due to previous palliative or reparative procedures. Common complications include pulmonary artery stenosis and distortion, ventricular volume overload and dysfunction, and occasional severe pulmonary hypertension as a result of a large palliative systemic to pulmonary shunt.

Intracardiac repair dramatically alters the prognosis of patients with tetralogy of Fallot. While the long-term survival of patients with repaired tetralogy of Fallot is excellent, it is not normal (34). Cardiac residua and sequelae are common and re-operation is often necessary (35).

Pulmonary valve regurgitation is expected following the transannular type surgical repair of tetralogy of Fallot. Long-standing pulmonary valve regurgitation may cause progressive right ventricular dilatation and systolic dysfunction, which can lead to inability to augment cardiac output with exercise and right heart failure in some cases. This group also has a significant incidence of ventricular arrhythmias associated with late sudden death.

Pulmonary valve replacement is required in many adult patients with severe pulmonary valve regurgitation following repair of tetralogy of Fallot. The most appropriate timing of pulmonary valve replacement is controversial but should be considered when patients develop symptoms, progressive right heart enlargement or dysfunction, worsening tricuspid valve regurgitation or arrhythmias.

It is difficult to meet certification requirements for a commercial motor vehicle driver following repair of tetralogy of Fallot.

Disqualify:

1. Symptoms of dyspnea or palpitations;
2. More than mild cardiac enlargement;
3. Persistent intracardiac lesions such as ventricular septal defect or more than mild pulmonary valve stenosis;
4. More than mild right or left ventricular enlargement or dysfunction identifiable by echocardiography;
5. Moderate or greater tricuspid valve regurgitation by echocardiography;
6. Severe pulmonary valve regurgitation by echocardiography;
7. History of atrial or ventricular arrhythmia (7);
8. Electrocardiogram demonstrating heart block or marked prolongation of the QRS interval $\geq$ 180 ms (36);
9. Residual right to left shunt or significant residual left to right shunt; or
10. Right ventricular systolic pressure greater than 50 mmHg.

The continuing risk of postoperative complications in patients with repaired tetralogy of Fallot warrants annual cardiovascular examination by a cardiologist knowledgeable in the management of adults with congenital heart disease. Annual evaluation required for re-certification should include electrocardiogram, 24-hour ambulatory electrocardiographic monitoring, exercise testing and Doppler echocardiogram (36-38).

TRANSPOSITION OF THE GREAT VESSELS

Due to the presence of substantial cyanosis and obligatory right to left shunt, individuals with uncorrected transposition of the great arteries do not qualify for commercial driver certification. Persons who have undergone surgery with atrial switch correction of transposition of the great arteries (Mustard or Senning procedures) do not qualify for commercial driver certification due to the risk of developing an unexpected arrhythmia and due to the propensity to develop systemic ventricular dysfunction and atrioventricular valve regurgitation (39,40).

The Rastelli operation involves placement of an intracardiac baffle to direct left ventricular blood to the aorta and an extracardiac valved conduit to establish continuity between right ventricle and pulmonary artery. The prognosis is favorable and certification is feasible (41).

Disqualify with Rastelli operation:

1. Symptoms of dyspnea or palpitations;
2. More than mild cardiac enlargement;
3. Persistent intracardiac lesions such as ventricular septal defect;
4. More than moderate pulmonary conduit stenosis with a right ventricular pressure over 50% systemic;
5. Left ventricular outflow tract obstruction with the gradient > 30 mmHg;
6. More than mild right or left ventricular enlargement or dysfunction;
7. Moderate or greater tricuspid valve regurgitation;
8. History of atrial or ventricular arrhythmia;
9. Electrocardiogram demonstrating heart block or marked prolongation of the QRS interval; or
10. Residual right-to-left shunt or significant residual left-to-right shunt.

The arterial switch procedure has been performed since the late 1970s. Some individuals who have had the arterial switch operation are now adults entering the work force. The advantages of the arterial switch procedure compared with atrial-level repairs include a lower incidence of arrhythmias and the likelihood of normal systemic ventricular function over the long term.

Although complications may occur after the arterial switch procedure, the prognosis appears excellent and certification may be potentially feasible (42). However, data on the clinical status of adults who have undergone the arterial switch procedure are currently lacking because most patients have not yet reached adulthood. The issue of certification should be reconsidered in the future as more data on their long-term outcome becomes available.

CONGENITALLY CORRECTED TRANSPOSITION OF THE GREAT ARTERIES

In congenitally corrected transposition of the great arteries, the connections of both the atria to ventricles and of the ventricles to the great arteries are discordant. The circulation is thus "physiologically" corrected, but the morphological right ventricle supports the systemic circulation. Associated anomalies occur in up to 95% and consist of ventricular septal defect, pulmonary or subpulmonary stenosis, and left-sided (tricuspid) valve anomalies (43). The conduction system is inherently abnormal in congenitally corrected transposition of the great arteries. Five percent of individuals with this type of congenital heart disease are born with a complete heart block. Complete heart block continues to occur over time at a rate of 2% per year.

Disqualify:

1. Symptoms of dyspnea or palpitations, syncope or paradoxical embolus;
2. Intracardiac lesions such as ventricular septal defect;
3. More than moderate pulmonary stenosis with a pulmonary ventricular pressure over 50% systemic;
4. More than mild right or left ventricular enlargement or dysfunction;
5. Moderate or greater tricuspid valve (systemic atrioventricular valve) regurgitation;
6. History of atrial or ventricular arrhythmia;
7. Electrocardiogram demonstrating heart block; or
8. Right-to-left shunt or significant residual left-to-right shunt.

Prolonged survival has been reported in these individuals (44). Progressive systemic (tricuspid) atrioventricular valve regurgitation and systemic (right) ventricular dysfunction tend to occur from the fourth decade onward, whereas atrial tachyarrhythmias are more common after the fifth decade (45).

Annual cardiac re-evaluation with echocardiography and ambulatory electrocardiographic monitoring is recommended. Persons with uncorrected cyanotic congenital heart disease should not be certified due to their risk of sudden cardiac compromise or death. After surgical intervention, certification is possible if the criteria outlined above are met. If a prosthetic valve is present, the individual must meet the criteria outlined in the section on Valvular Heart Disease for commercial driver certification.

PULMONARY HYPERTENSION

Significant pulmonary hypertension (pulmonary artery pressure over 50% systemic) from any cause should disqualify an individual from commercial driving. Persons with primary pulmonary hypertension are continually at risk of sudden death. Similarly, individuals with secondary pulmonary hypertension, such as those with Eisenmenger’s syndrome, are also at risk of incapacitation and sudden death.

COMPLEX CONGENITAL HEART DISEASE WITH PRIOR FONTAN OPERATION

Individuals with palliated complex congenital heart disease are not candidates for certification. Survival depends on preoperative criteria and may be as low as 60% to 71% in "all comers" (46). Progressive deterioration of functional status with time is the rule. Atrial flutter or fibrillation is common (15% to 20% at 5 years). Progressive deterioration of systemic ventricular function, with or without progressive atrioventricular valve regurgitation, is common. Most deaths are from congestive heart failure and atrial arrhythmias.

RECOMMENDATION TABLES
AORTIC CONGENITAL HEART DISEASE

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Bicuspid Aortic Valve	May result in aortic stenosis or regurgitation (see section on Valvular Diseases), aortic root enlargement, aortic aneurysm formation and aortic rupture.	See section on Valvular Diseases. No if: Aortic transverse diameter > 5.5 cm. Yes if: Surgical intervention successfully performed.	See section on Valvular Diseases. Annual
Subvalvular Aortic Stenosis	Mild = favorable Has potential for progression. Moderate or severe = unfavorable.	Yes if: No valvular abnormality or hypertrophic cardiomyopathy. No if: Symptomatic and mean pressure gradient >30 mm Hg. Yes if: At least 3 months after successful surgical resection when cleared by cardiologist knowledgeable in congenital heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congential heart disease is required. Annual Evaluation by cardiologist knowledgeable in adult congential heart disease required, including echocardiogram.
Discrete supravalvular Aortic Stenosis	Unfavorable prognosis due to associated coronary and aortic disorder.	No, unless surgery. Yes if: At least 3 months post surgical intervention; and cleared by cardiologist knowledgeable in adult congenital heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease is recommended.

RECOMMENDATION TABLES
AORTIC CONGENITAL HEART DISEASE
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Marfan syndrome	Cardiovascular disorders are the major cause of morbidity and mortality including risk of sudden death.	Yes if: No cardiovascular involvement. No if: Any aortic root enlargement; moderate or more severe aortic regurgitation; > mild mitral regurgitation related to mitral valve prolapse; LV dysfunction with EF <40% and no associated valve disease.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease required including aortic root imaging and echocardiography.

RECOMMENDATION TABLES
ATRIAL SEPTAL DEFECTS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Atrial Septal Defect: Ostium Secundum	Small = favorable.	Yes if: Asymptomatic.	Annual Evaluation by cardiologist knowledgeable in congential heart disease including echocardiogram.
	Moderate to large = unfavorable.	No if: Symptoms of dyspnea, palpitations or a paradoxical embolus; Pulmonary hypertension; Right-to-left shunt; or Pulmonary to systemic flow ratio > 1.5 to 1.	
		Yes if: At least 3 months after surgery or at least 4 weeks after device closure; asymptomatic and clearance by cardiologist knowledgeable in adult congenital heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease every two years.

RECOMMENDATION TABLES
ATRIAL SEPTAL DEFECTS
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Atrial Septal Defect: Ostium Primum	Small ASD = favorable prognosis.	Yes, if asymptomatic.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease required including echocardiogram.
	Moderate to large ASD = unfavorable prognosis	No if: Symptoms of dyspnea, palpitations or a paradoxical embolus; or Echo-Doppler demonstrates pulmonary artery pressure > 50% systemic; or echo-Doppler demonstrates right-to-left shunt; or pulmonary to systemic flow ratio greater than 1.5 to 1; or heart block on an electrocardiogram; more than mild mitral valve regurgitation; or left ventricular outflow tract obstruction with a gradient >30 mmHg.	
	.	Yes if: At least 3 months after surgical intervention if none of the above disqualifying criteria; and no symptomatic arrhythmia and no significant residual shunt; cleared by cardiologist knowledgeable in adult congenital heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease.

RECOMMENDATION TABLES
ATRIAL SEPTAL DEFECTS
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Sinus Venosus Atrial Septal Defect	Usually associated with anomalous pulmonary venous connection. Prognosis depends on size of atrial septal defect. Commonly associated with sinus node dysfunction, particularly after surgery.	Yes if: Small shunt and hemodynamically insignificant. No if: Symptoms of dyspnea, palpitations or a paradoxical embolus; Echo-Doppler examination demonstrating pulmonary artery pressure greater than 50% systemic; Echo-Doppler examination demonstrating a right-to-left shunt; A pulmonary to systemic flow ratio greater than 1.5 to 1; or Heart block or sinus node dysfunction on an electrocardiogram. Yes if: At least 3 months after surgical intervention; and hemodynamics are favorable; and cleared by cardiologist knowledgeable in adult congenital heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease. Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease, including Holter Monitor.

RECOMMENDATION TABLES
VENTRICULAR SEPTAL DEFECTS

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Ventricular Septal Defect	Small = favorable.	Yes, if small shunt.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease recommended.
	Moderate to large VSD has effect on pulmonary pressure and ventricular size and function.	No if: Moderate to large VSD; Symptoms of dyspnea, palpitations or syncope; Pulmonary artery hypertension; Right-to-left shunt, left ventricular enlargement or reduced function; Pulmonary to systemic flow ratio greater than 1.5 to 1.	
		Yes if: At least 3 months after surgery; None of above disqualifying criteria; No serious dysrhythmia on 24 hour Holter Monitoring; QRS interval <120 ms; (If right ventricle conduction delay >120 ms on ECG, can be certified if invasive HIS bundle studies show no infra-His block or other serious electrophysiologic disorder); Cleared by cardiologist knowledgeable in adult congential heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congential heart disease, including 24 hour Holter Monitoring.

RECOMMENDATION TABLES
CONGENITAL HEART DISEASE

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Patent Ductus Arteriosus (PDA)	Small = favorable. Moderate to large = unfavorable.	Yes, if small shunt. No if: Symptoms of dyspnea or palpitations; Pulmonary hypertension; Right to left shunt; Progressive LV enlargement or decreased systolic function. Yes if: At least 3 months after surgery or 1 month after device closure; None of above disqualifying criteria; Cleared by cardiologist knowledgeable in adult congential heart disease.	Annual Annual Should have evaluation by cardiologist knowledgeable in adult congenital heart disease.
Coarctation of the Aorta	Mild = favorable. Moderate or severe = unfavorable prognosis.	Yes if: Mild and unoperated; BP controlled; and No associated disqualifying disease. No	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease recommended.
Coarctation of the Aorta after intervention	Unfavorable prognosis with persistent risk of cardiovascular events.	Yes, if perfect repair (see text).	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease required.

RECOMMENDATION TABLES
CONGENITAL HEART DISEASE
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Pulmonary Valve Stenosis (PS)	Mild and moderate = favorable. Severe PS may be unfavorable, associated with arrhythmias and rarely sudden death.	Yes, if mild or moderate. No if Symptoms of dyspnea, palpitations or syncope; Pulmonary valve peak gradient >50 mmHg with normal output; RV pressuve >50% systemic pressure; >mile RVH; >mild RV dysfunction; >moderate pulmonary valve regurgitation; or main pulmonary artery >5cm. Yes if: 3 months after surgical valvotomy or 1 month after balloon valvuloplasty; None of above disqualifying criteria; Cleared by cardiologist knowledgeable in adult congenital heart disease.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease. Annual Recommend evaluation by cardiologist knowledgeable in adult congenital heart disease.
Other causes of right ventricular outflow obstruction in persons with congential heart disease.	Double chambered right ventricle, Infundibular pulmonary stenosis, Supravalvar pulmonary stenosis, Pulmonary artery stenosis.	Yes if: Hemodynamic data and criteria similar to individuals with isolated pulmonary valve stenosis who are eligible for certification.	Annual Recommend evaluation by cardiologist knowledgeable in adult congenital heart disease.

RECOMMENDATION TABLES
CONGENITAL HEART DISEASE
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Ebstein anomaly	Mild = favorable.	Yes if: Mild; Asymtomatic; No intracardiac lesions; No shunt; No symptomatic arrhythmia or accessory conduction; Only mild cardiac enlargement; Only mild RV dysfunction.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease.
	Moderate and severe variants = unfavorable.	No Yes if: At least 3 months post-surgical intervention; None of above disqualifying features.	Annual Echocardiogram and evaluation by cardiologist knowledgeable in adult congenital heart disease required.
Tetralogy of Fallot	Unfavorable in the unrepaired state.	No, if uncorrected.	
	Repaired = variable prognosis.	Yes if: Excellent result obtained from surgery; Asymptomatic; No significant pulmonary or tricuspid valve regurgitation; No pulmonary stenosis; No history of arrhythmias; No residual shunt.	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease required, including EKG, 24 hour Holter Monitor, exercise testing, Doppler Echocardiogram.

RECOMMENDATION TABLES
CONGENITAL HEART DISEASE
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Transposition of the Great Vessels	Unfavorable if uncorrectable.	No	
	Atrial switch repair (Mustard or Senning procedures). Unfavorable long-term prognosis.	No	
	After Rastelli repair.	Yes if: Asymptomatic and excellent result obtained from surgery (see text).	Annual Evaluation by cardiologist knowledgeable in adult congenital heart disease.
	After arterial switch repair, prognosis appears favorable.	No (Data currently not sufficient to support qualification in this group).	

RECOMMENDATION TABLES
CONGENITAL HEART DISEASE
(Continued)

DIAGNOSIS	PHYSIOLOGY/ FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Congenitally corrected transposition	95% have associated intracardiac lesions. Conduction system is inherently abnormal.	Yes if: None of below disqualifying criteria.	Annual Required annual evaluation by cardiologist knowledgeable in adult congenital heart disease, includes echocardiography and 24 hour Holter Monitor.
	.	No if: Symptoms of dyspnea, palpitations, syncope or paradoxical embolus; Intracardiac lesion such as VSD; >moderate pulmonary stenosis with a pulmonary ventricular pressure >50% systemic; >mild RV or LV enlargement or dysfunction; Moderate or greater tricuspid valve (systemic atrioventricular valve) regurgitation; History of atrial or ventricular arrhythmia; ECG with heart block; or Right-to-left shunt or significant residual left-to-right shunt.	
		Yes if: At least 3 months after surgery; None of above disqualifying criteria; If prosthetic valve – must meet requirements for that valve; Cleared by cardiologist knowledgeable in adult congential heart disease.	Annual Recommend evaluation by cardiologist knowledgeable in adult congential heart disease.

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Aortic Aneurysms

Peripheral Vascular Disease

Venous Disease

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AORTIC ANEURYSMS

Epidemiology

In autopsy studies, the incidence of abdominal aortic aneurysms (AAA) ranges from 1.8% to 6.6 % and is increasing. Males are more likely to have an AAA than are females by a 3:1 to as much as an 8:1 margin (1,2,3). The majority of AAA occur in the sixth and seventh decades of life, because aging causes changes in the elastic tissue, collagen, smooth muscle and ground substance which make the aorta less distensible and less able to absorb the forces from left ventricular contraction (4,5).

AAA and Sudden Death or Driver Incapacitation

Rupture or dissection of the aorta accounts for 1.3% to 3.8% of sudden death in drivers (6,7,8). The 10-year prospective study (1/1/1978 to 12/31/1987) by Christian re-focused attention on AAA as a significant cause of sudden death in drivers. This study identified seven who died of a ruptured AAA out of a total of 56 drivers (12.5%) who died from cardiovascular disease (CVD) within two hours of arrival at an emergency room. All seven cases occurred in men, three while driving. None of the seven had prior knowledge of the condition (9).

Anatomy of Abdominal Aortic Aneurysms

Aortic aneurysms can be classified by morphology, etiology or location. In general, the location determines the cause, clinical presentation, and treatment. An aneurysm can be located in the thoracic, the thoracoabdominal or abdominal area. The abdominal aorta begins at the diaphragm and descends to the fourth lumbar vertebra, where it divides into the common iliac arteries. The abdominal aortic aneurysms are almost always located below the renal arteries (3).

Risk Factors and Associated Conditions

Approximately 90% of aneurysms are secondary to atherosclerosis (4,10). AAA are eight times as frequent among those smoking one or two packs of cigarettes per day than among non-smokers (11). Hypertension is present in 60% of patients with aortic aneurysms and 80% of persons with aortic dissection. Because the risk of rupture is proportional to the stress on the artery wall, hypertension probably plays a role in aneurysm formation; however, it is not clear whether hypertension accelerates the aneurysm's growth rate (12). More recently, there has been increasing investigation into the role genetics plays in the development of AAA (2).

Diagnosis

Most aneurysms are asymptomatic and discovered either on routine examination or on an abdominal x-ray done for another reason. On x-ray, the calcified wall of the aneurysm is seen in approximately 50% of cases (13). Auscultation of an abdominal bruit may indicate the presence of an aneurysm. Detection of an AAA on physical examination depends on the aneurysm's size and the person's obesity. Larger aneurysms are more easily detected. Approximately 90% of AAA greater than 6 cm are identified on clinical examination. Aneurysms larger than 4 cm may be palpable in non-obese persons. The overall detection rate on examination is 31% when compared to detection by ultrasound, which has an almost 100% sensitivity and specificity (2,3,13,14,15).

Complications

An AAA can be an acute life-threatening risk, a condition requiring ongoing follow-up, or a disease associated with other CVD. Rupture is the most feared complication of an aortic aneurysm (16), and is the tenth leading cause of death among men older than 55 years of age, with a mortality rate of 78% to 94%. (1,14).

While it is difficult to predict when an aneurysm is going to rupture, the risk increases geometrically as the aneurysm increases in size. AAA less than 4 cm rarely rupture. AAA smaller than 5 cm have a 1% to 3% per year rate of rupture, AAA of 5 to 6 cm have a 5% to 10% per year rate of rupture, and AAA greater than 7 cm have approximately a 20% per year risk of rupture (1,17).

The average growth rate has been reported to be 0.21cm to 0.40 cm per year; but greater increases in size occur in at least one-quarter of patients (3). Factors that correlate with the rate of growth of an aneurysm include the largest aneurismal cross-sectional area, tobacco use, and tortuosity (18).

Bickerstaff reported that the mean period from detection of an AAA to rupture was 48.7 months (5). In another study, when the criteria used for operative intervention was an aneurysm > 5 cm or an increase in size > 0.5 cm in six months, less than 50% of patients entering the study with an AAA less than 5 cm needed surgery within a mean of three years (19). A more aggressive approach for surgical repair is to operate when the surgical mortality is less than the per year risk of rupture. With improved surgical outcomes, and without contraindication for surgery, aneurysms greater than 4 cm are electively repaired to prevent dissection and rupture (1).

Less common complications include distal embolization, sudden and complete thrombosis, infection, chronic coagulopathy, aortic intestinal fistula, and development of AV fistula between the aorta and vena cava (4).

Commercial Driver Certification

Though the decision to operate on a person with an aneurysm is often controversial and is beyond the scope of the medical examination, certain guidelines for CMV driver certification are useful. An AAA larger than 5.0 cm should disqualify the driver because of the high risk of rupture. A vascular specialist should review an AAA larger than 4 cm before a commercial driver is certified to drive. The decision by the driver's health care provider not to surgically repair an aneurysm does not mean that the driver can be certified to drive safely. However, a recommendation to operate on an aneurysm disqualifies the driver until the aneurysm has been repaired and a satisfactory recovery period has passed.

Detection of an AAA should prompt a search for the presence of other CVD. AAA are found in approximately 5% of patients with coronary disease, 10% of those with cerebrovascular disease, and 20% of patients with peripheral vascular disease (14,15).

Thoracic Aortic Aneurysms

The thoracic aorta runs from the base of the left ventricle to the diaphragm. The thoracic aorta can be divided into the ascending, transverse (arch), and descending segments (3). While relatively rare, thoracic aneurysms are increasing in frequency (20). Aortic size has been considered the major factor in determining risk for dissection or rupture. The rate of rupture or dissection has ranged from 8.8% for a 3.5 cm aneurysm to 27.9% for an aneurysm greater than 6 cm. The growth rate of thoracic aneurysms has been reported to average 0.10 cm per year. Those that required surgery had more rapid growth rates (21). Following repair, at least a 3-month period before driving a commercial vehicle is recommended.

Aneurysms of Other Vessels

Much of the information on aortic aneurysms is applicable to aneurysms in other arteries. Although less common than aortic aneurysms, aneurysms can develop in the visceral arteries: splenic, subclavian, celiac, gastroduodenal, and renal; and can develop in peripheral arteries: including the femoral, iliac, and popliteal arteries; and venous vessels. Rupture of any of these aneurysms can lead to sudden incapacitation and death (4,22-26).

Certification of the driver should require review by a vascular specialist. Surgical repair requires an appropriate waiting period before driving again. The location of the aneurysm and surgical repair required affect the length of the waiting period before certification. In general, a 3-month waiting period is recommended.

PERIPHERAL VASCULAR DISEASE

Peripheral Vascular Disease and its Symptoms

Obstructive vascular disease of the lower extremities is a widely recognized peripheral vascular disease (PVD) in adults. In 90% of cases, atherosclerotic plaques cause the obstruction (27). PVD increases with age, with an estimated prevalence of 12% to 17% among those over age 50 (28).

PVD may be present for many years before symptoms and signs of obstruction develop. Approximately 7% to 9% of persons with PVD develop intermittent claudication, (IC) the primary symptom of obstructive vascular disease of the lower extremity. With severe arterial insufficiency, necrosis, neuropathy and atrophy may occur (29,30,31). The occurrence of pain at rest marks the onset of a critical degree of ischemia.

Diagnosis

At times, the clinical history of IC may be confused with other medical conditions, including venous claudication, chronic compartment syndromes, peripheral nerve pain from a herniated disc, spinal cord compression, and osteoarthritis of the hip. However, each of these other disease entities has specific symptoms and findings that aid in making the diagnosis.

The diagnosis of IC may be suspected from the characteristic history of slowly developing aching leg pain on exercise that is relieved by rest. Absence of a femoral artery, posterior tibial or dorsalis pedis pulse on physical examination may indicate PVD, although approximately 10% of normal adults lack at least one of these three pulses. In addition to palpation, auscultation over the arteries may detect a bruit, indicative of partial obstruction with resulting turbulence of the blood flow.

The skin of the legs may have changes in color and temperature. The skin may also reveal changes associated with chronic ischemia, including thin, dry skin, loss of hair, loss of subcutaneous fat, and thickened nails.

The level and the extent of the blockage determine the location of the pain and the amount of exercise required to cause the pain. Among those under 40, aortoiliac disease is more common, while femoral-popliteal disease causes two-thirds of the cases of claudication in people over age 40. Superficial femoral obstructive disease generally causes pain in the calf; aortoiliac or internal iliac disease usually causes aching pain in the thigh, hip or buttocks (31,32,33).

Associated Cardiovascular Disease

Muluk et al. reported that the yearly mortality rate was 12% among those with IC, much more than the age-adjusted mortality in the United States population (34). The atherosclerotic changes found in the peripheral arteries are often present in other vessels in the body (29). Among those with symptomatic PVD, the 10-year mortality rate from CVD is 10 to 15 fold greater compared to those without symptoms. A significant majority of patients with PVD die from cardiovascular diseases, with the majority of deaths from coronary heart disease (29,35,36,37).

Clinical Course

PVD is usually a slowly progressive disease with a benign course and essentially no risk for sudden incapacitation. Approximately 25% of those with claudication develop worsening symptoms, usually in the first year (30,31,38). Systolic hypertension, cigarette use, diabetes and hyperlipidemia can each increase the likelihood of developing increasingly severe PVD (33,39,40). In more advanced cases, ulceration and/or gangrene may be present.

For the commercial vehicle driver, rest pain represents a critical degree of ischemia and is disqualifying because of the likelihood of reduced dexterity of the affected limb. The reported rates for surgical revascularization, angioplasty or amputation have ranged from 3% to 22%. The amputation rate is less than 10% at 10 years (29,31,34).

Treatment

Risk factor modification, especially stopping smoking, is the cornerstone of treatment. Staying active carries little risk for the person with IC; an exercise program designed to increase the activity of a person with IC has been effective (38).

Although more promising pharmacotherapy is being researched, there is currently limited pharmacological treatment for IC. Anti-platelet agents are used to treat a variety of atherosclerotic conditions. The effectiveness of oral anticoagulants is more uncertain. Tangelder reported that use of an oral anticoagulant at a dose to maintain the INR between 3.0 and 4.0 is optimal therapy to prevent infrainguinal bypass occlusion. In this study, there were 0.9 ischemic and 2.9 hemorrhagic events per 100 patient years (41). The use of oral anticoagulant is not disqualifying, but does require more intensive monitoring, with at least monthly INR measurements to help assure appropriate anticoagulation. The role of aspirin after lower extremity grafting has been difficult to define. Current drug treatments do not appear to prevent the commercial vehicle driver from working (42,43**).**

Surgery and angioplasty are successful in treating persons with severe leg ischemia when other non-invasive treatments, including risk factor modification, have failed. Angioplasty is more effective in the treatment of common iliac disease and for short segment disease. Below the inguinal ligament, however, the patency rate is decreased (44**).** Chetter reported a cumulative patency rate with angioplasty of 75% at six months, at the same time noting that patency rates from 50% to 90% had been reported for iliac lesions and 43% to 75% for femoral-popliteal lesions (45). Lower extremity bypass graft patency rates of 70% at five years, with a limb salvage rate of 91% at five years reported (46).

The commercial driver who has had a vascular procedure needs time to recover from the surgery, to be regulated on medication(s), and observed for early graft failure. The majority who return to commercial driving after surgery have done so by six months.

VENOUS DISEASE

Deep Vein Thrombosis

Deep vein thrombosis (DVT) can cause life-threatening acute complications as well as long-term venous problems (47). Venous stasis had been considered the most important cause of lower extremity DVT. Research interest now centers on the balance between activator and inhibitor substances secreted by the vessel wall and circulating levels of pro- and anticoagulants (48,49).

DVT can be the source of pulmonary emboli that can cause sudden incapacitation and significant mortality and morbidity (50). Lopez-Beret reported that 41% of people with active DVT had occult pulmonary emboli (51). Acute DVT is disqualifying for the commercial driver until adequately treated. Chronic thrombotic venous disease of the legs predisposes to pulmonary emboli, but the level of risk has not been studied (52). The majority of individuals who have had an episode of untreated DVT develop venous insufficiency, with stasis changes and stasis ulcers (50). Although superficial phlebitis is a benign and self-limited disease, DVT is often a co-existing condition and needs to be excluded (52).

Adequate anticoagulation decreases the risk of recurrent thrombosis by approximately 80%. Treatment includes heparin for the first few days, followed by oral anticoagulant. In general, anticoagulant treatment is continued for three to six months (53). More recently, low molecular weight heparin has been advocated as a more effective treatment than oral anticoagulants for the long-term management of DVT (52,53).

Varicose Veins

Varicose veins with their associated symptoms and complications are the most common disorder of the lower extremities, affecting over 20 million people in the United States (50). Complications include chronic venous insufficiency, leg ulcerations and recurrent DVT. Varicose veins do not medically disqualify the commercial driver.

RECOMMENDATION TABLES
ANEURYSMS

DISEASE	PHYSIOLOGY / FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Abdominal Aortic Aneurysm	Evaluate for associated cardiovascular diseases.		
	Aneurysm < 4.0 cm.	Yes, if asymptomatic.	Annual
	Aneurysm 4.0 to <5.0 cm. Ultrasound to identify change in size.	Yes if: Asymptomatic; Cleared by vascular specialist.	Annual Ultrasound for change in size.
		No, if: Symptomatic; or surgery recommended by vascular specialist.	
		Yes if: At least 3 months after surgical repair. Cleared by cardiovascular specialist.	Annual
	Aneurysm $\geq$ 5.0 cm.	No.	
		Yes if: At least 3 months after surgical repair. Cleared by cardiovascular specialist.	Annual
Thoracic Aneurysm	Evaluate for associated cardiovascular diseases.	No, if >3.5cm.	
		Yes if: At least 3 months after surgical repair. Cleared by cardiovascular specialist.	Annual
Aneurysms of other vessels	Assess for risk of rupture and for associated cardiovascular diseases.	No	
		Yes if: At least 3 months after surgical repair. Cleared by cardiovascular specialist.	Annual

RECOMMENDATION TABLES
PERIPHERAL VASCULAR DISEASE

DIAGNOSIS	FUNCTIONAL/ PHYSIOLOGIC	CERTIFICATION	RE-CERTIFICATION
Peripheral Vascular Disease (PVD)	Evaluate for associated cardiovascular diseases.	Yes, if no other disqualifying cardiovascular condition.	Annual
Intermittent Claudication	Most common presenting manifestation of occlusive arterial disease.	Yes if: At least 3 months after surgery; Relief of symptoms; No other disqualifying cardiovascular disease.	Annual
	Rest pain.	No, if symptoms. Yes if: At least 3 months after surgery; Relief of symptoms and signs; No other disqualifying cardiovascular disease.	Annual

RECOMMENDATION TABLES
VENOUS DISEASE

DISEASE	PHYSIOLOGY / FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Acute Deep Vein Thrombosis (DVT)		No, if symptoms. Yes if: No residual acute deep venous thrombosis; If on Coumadin: Regulated for at least 1 month; INR monitored at least monthly.	Annual
Superficial phlebitis		Yes if: DVT ruled out; No other disqualifying cardiovascular disease.	Biennial
Pulmonary Embolus		No, if symptoms. Yes if: No pulmonary embolism for at least 3 months; On appropriate long-term treatment. If on Coumadin, regulated for at least 1 month; INR monitored at least monthly; and No other disqualifying cardiovascular disease.	Annual
Chronic Thrombotic Venous Disease		Yes, if no symptoms.	Biennial
Varicose veins		Yes, if no complications.	Biennial
Coumadin	Use of INR required.	Yes if: Stabilized for 1 month; INR monitored at least monthly.	Annual

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Heart Transplantation

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HEART TRANSPLANTATION

Background

Although the number of heart transplant recipients is relatively small, some recipients may wish to be (re)-certified as a commercial motor vehicle driver. These recipients are already closely monitored by experts in the fields of cardiology and transplant medicine. The major medical concerns for certification of a commercial driver heart recipient are transplant rejection and post-transplant atherosclerosis.

Criteria for Commercial Driving

1. At least 1 year post-transplant;
2. Asymptomatic;
3. Initial consent from cardiologist to drive commercially;
4. Stable on medications;
5. No rejection; and
6. Certification every 6 months after evaluation by a cardiologist, with a focus on accelerated atherosclerosis, transplant status, and general health.

RECOMMENDATION TABLE
HEART TRANSPLANTATION

DIAGNOSIS	PHYSIOLOGY / FUNCTIONAL	CERTIFICATION	RE-CERTIFICATION
Heart Transplantation	Special attention to: Accelerated atherosclerosis, transplant rejection, general health.	Yes if: At least 1 year post-transplant; asymptomatic; stable on medications; no rejection; Consent from cardiologist to drive commercially.	Biannual Clearance by cardiologist required.