



# **New Standards for Driving and Cardiovascular Diseases**

Report of the Expert Group on Driving and Cardiovascular Disease

Brussels, October 2013



1	Introduction.....	4
2	Psychosocial issues of driving restriction and adherence to recommendations.....	5
3	The evidence base for driving and heart disease – Estimates and acceptance of risk.....	6
3.1	The relationship between heart disease and accidents.....	6
3.2	Risk of sudden (cardiac) incapacitation (SCI).....	7
3.3	The likelihood that a disease state leads to third party personal injury – the “Risk of Harm Formula”.....	7
3.4	Relative risk of an accident as a function of yearly risk of SCI.....	8
3.5	Acceptable risk of SCI in group 1 and group 2.....	8
3.6	References.....	9
4	Definition of private drivers and professional drivers.....	9
5	Driving for specific cardiovascular diseases.....	10
5.1	Arrhythmias.....	10
5.1.1	Introduction.....	10
5.1.2	Bradycardias.....	10
5.1.3	Supraventricular arrhythmias.....	11
5.1.4	References.....	12
5.1.5	Recommendations.....	12
5.1.6	Ventricular arrhythmia.....	14
5.1.7	Implantable Cardioverter-Defibrillators.....	16
5.2	Syncope.....	26
5.2.1	Introduction and definitions.....	26
5.2.2	The risk of recurrence of syncope.....	27
5.2.3	The risk of a syncope during driving.....	28
5.2.4	From recurrence rates to guidelines for driving after syncope of reflex or unknown origin.....	29
5.2.5	Suggested recommendations.....	29
5.2.6	References.....	30
5.3	Coronary Artery Disease (CAD).....	30
5.3.1	Acute coronary syndrome, stable CAD/angina, Percutaneous Coronary Intervention, Coronary Artery Bypass Graft.....	30
5.4	Peripheral vascular disease.....	35
5.4.1	Carotid artery stenosis.....	35
5.4.2	Thoracic and abdominal aortic aneurysm.....	36
5.5	Heart failure.....	39
5.5.1	Heart failure.....	39
5.5.2	Heart transplantation.....	42
5.5.3	Cardiac assist devices.....	43
5.6	Valvular heart disease.....	43
5.6.1	Aortic regurgitation.....	43
5.6.2	Aortic stenosis.....	43
5.6.3	Mitral regurgitation.....	44
5.6.4	Mitral stenosis (MS).....	44
5.6.5	Valvular heart surgery.....	45
5.7	Arterial hypertension.....	46
5.7.1	Background.....	46
5.7.2	Recommendations.....	47
5.7.3	References.....	47
5.8	Congenital heart disease.....	48
5.8.1	Background.....	48
5.8.2	Arrhythmias and sudden cardiac death.....	48
5.8.3	Recommendations.....	48
5.8.4	References.....	48
5.9	Structural and electrical cardiomyopathies.....	49
5.9.1	Hypertrophic cardiomyopathy (HCM).....	49
5.9.2	Long QT syndrome.....	51
5.9.3	Brugada syndrome.....	53
5.9.4	Other cardiomyopathies.....	54
6	Final recommendations: Proposition of legal text.....	55

## 1 Introduction

This report has been prepared by the Working Group on Driving and Cardiovascular Diseases as the basis for suggested amendments to the Annex III to Directive 2006/126/EC on driving licences.

The provisions on cardiovascular diseases in the current Annex III, which lays down minimum standards of physical and mental fitness for driving, was last revised in 1991 and are generic in nature. A medical opinion delivered by the European Society of Cardiology and the European Heart Rhythm Association to the Commission emphasised the need to update the provisions to scientific progress and new forms of treatment, like the use of implantable cardioverter defibrillators. Against this background, the Driving Licence Committee agreed in February 2010 to establish a Working Group on Driving and Cardiovascular Diseases. The Working Group's objective was to assess the road safety risks associated with cardiovascular diseases from a current medical perspective and to formulate appropriate guidelines. All EU Member States were invited to nominate an expert from their respective country and the working group was formally established in June 2010. The Working Group comprised 12 experts from 10 Member States, including Norway and Switzerland. The experts were coming from the field of cardiovascular disorders (representing the European Society of Cardiology) and from the licensing regulatory authorities. In addition, the Working Group also consulted with external experts.

The first Working Group meeting was on 29 June 2010 and the group subsequently met on the following dates: 16 November 2010, 8 February 2011, 20 September 2011, 18 October 2011, 13 December 2011 and 12 June 2012. An editing group was created whose task was to summarize, revise and edit all contributions and to prepare the final report. The editing group also assisted the European Commission in the formulation of draft amendments to Annex III to Directive 2006/126, based on the recommendations of the final report. The Editing group met on 23 June 2013 and on 26 and 27 September 2013 and carried out much of the work also via extensive email and telephone correspondence.

The objective of this report is to give a *bona fide* overview of the current knowledge on the cardiovascular disease and its relevance to driving as a basis for the above mentioned draft amendments. This report reflects the consensus of the experts forming part of this Working Group. A consensus is generally defined as the majority opinion or general agreement of the Group. It should be noted that consensus does not imply that all experts unanimously agreed on all the findings and recommendations. The Working Group was aware that they had to balance public road safety with the right of individual mobility, especially if there is no significant risk to public road safety. Rules must be as liberal, simple and as clear as they can. Furthermore, they should be based on evidence and on calculated risk as far as possible. However, it has to be stressed that rigorous scientific proof is not always sufficiently available for the decisions that have to be taken with regard to cardiovascular diseases and driving. In such cases, the best available evidence, reasonable estimates and consensus expert opinion were used in the report.

Cardiovascular (heart) disease is a broad term for a group of disorders that affect the heart and the blood vessels. The recommendations in this report are mainly relevant to those cardiovascular disorders which either pose a current or a prospective risk of a significant, sudden and disabling event and/or impair an individual from safely controlling his/her vehicle. The main conditions discussed in this report are – coronary artery disease, cardiac rhythm disorders, syncope, cardiac failure, valvular heart disease, hypertension, peripheral vascular disease, congenital heart disease and cardiomyopathy.

A recent WHO report “Global Atlas on Cardiovascular Disease Prevention and Control” states that cardiovascular disease is the leading cause of death and disability in the world. Although a large proportion of cardiovascular disease is preventable, they continue to rise mainly because preventative measures are inadequate.

Risk of cardiac fatality rises exponentially with age. Even when they are not fatal, cardiovascular diseases may be sufficiently debilitating to seriously affect the functional ability of an individual or lead to a sudden incapacitating event. This is hard to assess without reliable morbidity data, but it may well be that 25-30% of cardiovascular disease burden arises from disabling sequelae of the heart disease. The available evidence suggests that the medical condition of a driver is not an important factor in a road traffic accident causing injury to other road users. Most road traffic accidents have a multi-

factorial cause. Studies in the UK and the USA have shown that approximately 95% of road traffic accidents involve human error. Of the 5% of the road traffic accidents which did involve medical conditions, cardiovascular conditions accounted for less than 5% and 75% of these cases were already diagnosed with cardiovascular disease(s). Precise data on cardiac disease as a cause of motor vehicle accidents is not known, but data from the US and Canadian studies suggest that less than 5% of accident involving commercial vehicles can be attributed to cardiovascular disease.

Several road safety reviews have highlighted the complexity involved in identifying the association between chronic medical conditions and the risk of crashes. The concept of acceptable risk in this report is based on the Risk of Harm Formula (developed by the Canadian Consensus Study 2003, and discussed in detail in subsequent chapters). This is because Risk of Harm Formula applied to individual conditions seems to be the best available tool to formulate a set of recommendations for fitness to drive. Above all, it creates consistency among all recommendations as this formula has also been used when setting fitness to drive standards in other areas e.g.in epilepsy and diabetes. The standards recommended for a Group 2 vehicle are more stringent due to the obvious reason of the greater size and weight of the vehicles and the duration of time spent behind the wheel by the Group 2 vehicle drivers.

While preparing this report, the members of the Group have considered the fact that there might be acceptable variations in the local clinical practices, duration of medical follow-up across various countries. Hence the Working Group has recommended the minimum safe interval for the follow-up reviews, at the same time it is important to remember that as cardiovascular diseases are generally of a progressive nature, it is important to have a regular medical follow-up especially in cases of Group 2 licences.

The recommendations in this report are guidelines for the minimum standards required for fitness to drive in applicants or drivers with cardiovascular disease(s). A medical doctor's assessment of a patient's fitness to drive should include consideration of the patient's level of knowledge and insight into their medical condition, ability to manage the condition, compliance with physician prescribed treatment and ability to modify driving activities to accommodate their medical condition. There is a lot of heterogeneity in the group of individuals with a particular heart condition (for example, stage and duration of disease, co-morbidities, risk factor profile and compliance to treatment). Ideally, these should be taken into account when assessing fitness to drive.

In the formulation of this report, account has been taken of the need to strike a fair balance between public road safety and individual rights and legitimate interests.

## **2 Psychosocial issues of driving restriction and adherence to recommendations**

Cardiovascular disease has in numerous studies been reported to affect the lives of patients and their families. However, specific research on psychosocial effects of driving restriction in patients is scarce. A qualitative study in ICD patients conducted in the United Kingdom (1) reports that driving restrictions are perceived as difficult for patients and their family and have an immediate consequence for their lifestyle. This entailed feelings of resentment and anger, increased dependence on others, lacking confidence in driving and imposed family sanctions when driving. Patients and their spouse stated that the imposed driving ban was the hardest part of having the ICD implanted. In addition to the psychological and societal impact, the driving ban may also pose a considerable impact on employment and education and thereby economic status. Driving is considered by many as a basic necessity. Following this, driving restrictions may have a substantial impact on the quality of life of patients with cardiovascular diseases.

The negative effects of driving restrictions have been of concern when outlining recommendations for driving for patients with cardiovascular diseases. Additional burden on recipients and their family's needs to be avoided. At the same time, adherence to advice given by health-care professionals needs to be maximised. As the driving restrictions can make the life situation of the patient and their families more difficult, this may affect adherence to the recommendations. Several studies (2-10) point in the direction of low adherence among recipients to the driving ban advised by health-care professionals. As there seems to be a gap between recommendations and patient adherence to these recommendations, an adequate education and follow-up of patients and family is pivotal. Hence, driving restrictions poses demands on health-care professionals in discussing alternative practical

solutions. Notably, studies have also identified that advice given to patients about when to resume driving is inaccurate (5) and differ between cardiologists (11). Improvement in standardized information given to patients is therefore desired.

Experiencing loss of consciousness while driving may result in death or injury to the patient, other passengers as well as members of the public. When recommendations that impose limitations on individuals driving privileges need to be considered, this also poses ethical issues. Whilst a driving ban imposes limitations on the lives of the patient and their family, their safety is also of concern. Similarly, public safety is of utmost importance. The aim of ethics as well as legislation is to ensure that the rights of the individual do not exceed the safety of fellow citizens and at the same time ensure that the rights of society to restrict individual action are limited.

### **Reporting non fulfilled health requirements**

If a patient is found not to fulfil the health requirements, the physician should inform the patient. The patient should feel obliged not to drive. General compliance to advice not to drive is pivotal to the safety effect of the health requirements. However, the degree of patient compliance with advice not to drive, has been found to be very low or zero (9). Thus, some countries have a legal obligation for physicians to report to the authorities when a patient no longer meets the requirements. However, the legislation varies considerably. In some states the physician is not allowed to report to the authorities, according to law or medical ethics code. In other states, the doctor is allowed, but not obliged, to report. The group recognised that the issue of reporting is governed by national legislation on other fields than road traffic legislation. There are reasons to support either of the strategies mentioned above. The group is of the opinion that reporting should be left to national legislation.

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## **3 The evidence base for driving and heart disease – Estimates and acceptance of risk**

### **3.1 The relationship between heart disease and accidents**

We are aware of only a small number of studies on the risk of accidents among people with various heart diseases. These studies have important limitations, such as:

- Reporting bias;

- Recall bias;
- Deceased subjects/drivers not included (in case control and retrospective cohort studies), even if obviously very relevant;
- Difficulty in deciding whether an accident was caused by a syncope or sudden death;
- Study outcome will be influenced by the existing driving regulations and by the advice given by the doctors to their patients;
- Health related accidents are probably relatively rare compared to other causes, and will therefore be difficult to measure in a reliable way;
- There is no systematic investigation of all traffic accidents, and thus difficult to know how prevalent health related accidents are;
- There are no disease registries to compare accident data to;
- Few of the studies meet modern quality criteria.

Thus, one has to build the recommendations on studies giving numbers for the rates of sudden (cardiac) incapacitation (SCI) in various patient groups.

### 3.2 Risk of sudden (cardiac) incapacitation (SCI)

Cardiovascular medicine has many prospective, controlled, randomised studies. However, most of these are estimating the risk of clinical end points like new myocardial infarctions, hospitalisation, death, need for revascularisation etc.. Only a few give data on SCI. Some give data on sudden death. But sudden death is usually defined as death within one hour or one day from onset of symptoms, and hence does not equal instantaneous death. The incidence of syncope is only rarely reported in the large studies of cardiovascular disease. An exception is of course studies on relapse rates of syncope in syncope patients. Also, some studies of ICD-patients report rates of syncope, arrhythmic death and sudden death, in addition to total mortality. In some diseases, dramatic and/or fatal outcomes like rupture of an aortic aneurysm will not always equal SCI, as there may be some time between symptom onset and loss of consciousness. Thus, with a few exceptions, the estimates of the risk of SCI, will depend on expert opinion or interpretation of subsidiary end points. This should be kept in mind when using the recommendations of this report to make legal regulations.

### 3.3 The likelihood that a disease state leads to third party personal injury – the “Risk of Harm Formula”

The risk of harm (RH) to other road users posed by the driver is assumed to be proportional to the:

- time spent behind the wheel (TD)
- type of vehicle (V)
- risk of sudden (cardiac) incapacitation (SCI)
- probability that SCI will result in a fatal or injury-producing accident (Ac) (1)

Thus:  $RH=TD*V*SCI*Ac$

The Canadian guidelines (1) state that a risk of third party death or injury of approximately 1/20,000 is acceptable. *This is a basic assumption of risk acceptance, not a calculated or derived value.*

Data cited in (1) suggests that if the risk associated with commercial driving of a large vehicle is 1, there is a risk of 0.28 associated with private car driving. Thus, V=1 for group 2 vehicles and 0.28 for group 1. Other cited data suggests that a commercial driver drives 25% of his/her time, while a private driver on average drives 4% of the time. Thus TD is 0.25 for group 2 and 0.04 for group 1 drivers. Obviously, there will be a certain amount of uncertainty of these figures. This may influence the calculations and results profoundly. This should be kept in mind when using the formula. “Ac”, the risk that a SCI leads to an accident, is a crucial number for the further use of the Risk of Harm Formula. It is stated to be 2%. Since this number is pivotal to all the following reasoning and calculations, and the risk assessments in the rest of this report, the evidence for this number will be discussed in some depth:

Öström et al. (2) presented 126 autopsy cases already judged to be sudden death during driving. Sixty-nine were car drivers, 35 bicyclists, 11 snowmobile riders, 6 mopedists, 4 riders of kick sleds (!) and one motorcyclist. Northern, mostly rural, Sweden 1980-85. Two passengers were injured. Sixty-six of the 69 car incidents were single vehicle accidents. No other road users were harmed. Thus, it may

support an estimate of “Ac” slightly below 2% (the passengers). Since the study included only cases which were on beforehand judged to be sudden death, the proportion of accidents caused by SCI cannot be estimated. Neither can the risk of an accident, given a SCI, since we don’t know the real number of SCIs. Death from arrhythmias will not be revealed at autopsy. Syncope or seizure with death or bodily damage to the driver, cannot be separated from other causes of accidents. Thus, the prevalence of sudden death or SCI as a cause of traffic accidents may be underestimated. Furthermore, northern, rural Sweden early nineteen eighties, may not resemble modern, urban traffic. Of 254 autopsies of road accident victims in Australia 1971-2, 102 were drivers (3). Eleven of these died “from natural causes”, as judged by autopsy. In 6/11 (=54%) a minor accident occurred, with no harm to others. Since cases where death did not cause an incident may not have been sent for autopsy, the frequency of crashes as a result of sudden death may have been overestimated. On the other hand, since causes of death or SCI not visible on autopsy, could not be included, the real number of sudden death or SCI as a cause of accidents probably is underestimated.

A “citation classic” (4) summarizes 92 cases where the authors’ patients told him that they had experienced a loss of consciousness while driving (“the hospital series”). He also collected 131 cases from press reports on presumed SCI at the wheel (“the published series”). The cause of the loss of consciousness was judged by the patient, the journalist and the author, on quite loose criteria. The unconventional method of case selection and classification limits the ability to draw conclusions from this paper. Nonetheless, it gives quite high numbers of accidents and injuries resulting from the events: In the “hospital series”, 19/39 fits lead to a collision, 5/39 involving another vehicle. No serious injury. In the “published series”, 3/8 fits resulted in a crash with another vehicle. One driver and five other persons were killed, giving an “Ac” of 5/8=63%. In the “published series”, 17/66 cases of presumed SCI led to a collision with another vehicle. This resulted in three “third party deaths”, Ac=4.5%, not counting personal injury other than death. In the “published series”, 38 incidents attributed to the driver falling asleep, killed 8 drivers and 51 other people, giving an “Ac” of 51/38=134% (more than one third party death per episode). This paper would suggest a fairly high “Ac”, of 4.5%, 63% and 134%, in the different parts of the study. Still it is cited as evidence to accept an Ac=2%.

In a retrospective study of necropsy reports where the victim had died in the driver’s seat of a car, 20 cases where judged to be sudden cardiac death (5). The following accidents happened:

- Collisions with parked vehicle: 4
- Collisions with property damage other than on vehicles: 5
- Collisions with operating vehicles: 3

This gives a 75% risk of accident if the driver dies suddenly. Since no persons other than the driver were injured, Ac=0%. In one case, an accident was prevented by a passenger on the bus who took over when the driver collapsed. So, the “Ac” could easily have been very high

*Thus, one should realise that the estimate of Ac=2% in the Risk of Harm formula, is not “evidence based” in the normal sense, and probably is a somewhat low estimate.*

In conclusion, if one uses the RH formula, and accepts the values of Ac=0.02, V=1 for group 2 and 0.28 for group 1, TD 0.25 for group 2 and 0.04 for group 1 and sets the acceptable RH at 1/20,000, the “allowable” yearly risk of an SCI will be 22% for group 1 and 1% for group 2.

### 3.4 Relative risk of an accident as a function of yearly risk of SCI

The relative risk is the risk of an accident in a person with the condition divided by the risk in a person without the condition. This is elaborated in the EU-report “Epilepsy and driving in Europe”, 2005, p 7-11. It is shown that if one accepts a relative risk R=2 – 3, the yearly risk of a SCI could be 20 – 37% in a group 1 driver. From a similar reasoning about TD and damage potential of large vehicles as in the RH formula, the acceptable yearly risk of a SCI is only 2% in group 2 drivers.

### 3.5 Acceptable risk of SCI in group 1 and group 2

The working group recognises the considerable degree of uncertainty of the assumptions behind the risk assessments according to the RH formula and the “relative risk” reasoning. This should be kept in



mind, especially when considering the possibility to grant exceptions from the rules in exceptional cases. The two methods yield similar estimates for acceptable yearly risk of SCI: 20-40% for group 1 and 2% for group 2. These numbers have formed the basis for the new EU requirements for epilepsy and driving. Thus, we will use the same numbers to assess criteria for fitness to drive in cardiovascular diseases. This means that if there is evidence, or expert opinion, that a condition carries a yearly risk of SCI below 2%, both group 1 and group 2 licences can be held. If the risk is judged to be between 2% and 20-40%, group 1 is allowed, but not group 2. If the risk is above 20-40%, neither group 1 or 2 can be held. It should be understood that these risk estimates apply to groups, and cannot be applied to single cases. Thus, the recommendations should identify groups of patients approximately corresponding to the risk levels mentioned. The risk levels cannot be used as such in the recommendations or the regulations.

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## **4 Definition of private drivers and professional drivers**

In the EU, the First Directive on driving licences (Directive 80/1263/EEC of 4 December 1980) proposed the establishment of a common European driving licence. The Second Directive on driving licences (Directive 91/439/EEC of 29 July 1991) further harmonised the standards at EU level. In its Annex III, the Directive distinguished between two groups of drivers:

Group 1 is formed by drivers of vehicles of categories A, A1, A2, AM, B, B1 and BE. This comprises drivers of e.g. motor cycles, passenger cars and other small vehicles with or without a trailer.

Group 2 is formed by drivers of vehicles of categories C, CE, C1, C1E, D, DE, D1 and D1E. This includes drivers of e.g. vehicles over 3.5 tonnes or vehicles designed for the carriage of more than nine passengers with including the driver.

For group 1, applicants shall be required to undergo a medical examination if it becomes apparent, when the necessary formalities are being completed or during the tests which they have to undergo prior to obtaining a driving licence, that they have one or more of the medical disabilities mentioned in Annex III. For group 2, applicants shall undergo medical examinations before a driving licence is first issued to them and thereafter they shall be checked in accordance with the national system in place in the Member State of normal residence whenever their driving licence is renewed..

This report will use the definitions used in the Driving licence Directive to distinguish between professional driving (group 2) and private driving (group 1). The experts in the Working Group strongly believe that, regardless of groups, particular consideration must be given to certain drivers such as drivers of taxis, ambulances and other professional drivers who spend many hours per day behind the wheel or carry passengers most of the time as they should be considered at higher risk. Under this perspective, the recommendations made for group 2 could also be applied to these drivers. Clinical judgement should prevail in borderline cases, for example for drivers of a small truck, where driving does not constitute the drivers' main activity or where it is for leisure activities.

## **5 Driving for specific cardiovascular diseases**

### **5.1 Arrhythmias**

#### **5.1.1 Introduction**

Arrhythmias, (disturbances of heart rhythm), are very common and give rise to a diverse range of symptoms. Although most arrhythmias may just cause symptoms like palpitations, dizziness or no symptoms at all, some may cause syncope or sudden cardiac death. (Syncope is, by definition, a relatively sudden, but short lasting and self-limited, loss of consciousness and postural tone, due to transient, global cerebral hypoperfusion. Syncope may be the result of many diseases/conditions, as discussed elsewhere in this report.) Hence arrhythmias have implications for road safety. Due to the diversity of the symptom profile, it is important to risk stratify the different arrhythmic conditions.

Data about the number of patients with supraventricular tachycardia suffering from syncope are scarce. Several studies demonstrated that syncopal symptoms may occur in 10-40% of patients and may be related to the heart rate during tachycardia or to the vasomotor tone. The problem with translating the results of these studies into a real world situation is that most studies only included patients referred for catheter ablation or highly symptomatic patients. Clinical experience suggests that most supraventricular tachycardias do not give rise to sudden syncope. There is lack of data on direct relevance of supraventricular tachycardia or symptomatic bradycardia on road safety issues.

In general symptomatic patients should be evaluated carefully, regardless of the underlying cause, to 1) establish the underlying disorder; 2) evaluate prognosis and risk of recurrence and 3) determine the best treatment strategy.

In this short report the most common cardiac rhythm disorders and recommendations with respect to driving relevant to these disorders are discussed. In general treatment should be based on guidelines, if available.

#### **5.1.2 Bradyarrhythmias**

##### **5.1.2.1 Sinus bradycardia**

Sinus bradycardia is common in athletes and causes symptoms only in extreme cases. Sinus bradycardia may be due to extra-cardiac diseases or drug treatment. In elderly people sinus bradycardia may cause fatigue and dizziness, but sudden syncope must be attributed to other aspects of sick sinus.

##### **5.1.2.2 Sick Sinus Syndrome (SSS)**

This consists of sinus bradycardia (above), sino atrial block, sino atrial arrest, chronotropic incompetence and tachy/brady-syndrome. Sino atrial block and arrest may cause pauses and syncope. Tachy/brady syndrome is the combination of any bradycardic component of SSS and supraventricular tachycardias, most often atrial tachycardias or atrial fibrillation.

##### **5.1.2.3 Atrio ventricular conduction block**

There are three degrees of AV conduction block:

- In first degree AV-block the conduction time from atria to ventricles is prolonged, but every atrial depolarisation gives rise to a ventricular depolarisation. There are no pauses.
- In second degree AV-block some atrial depolarisations are not followed by a ventricular depolarization. Thus, there will be pauses, usually of short duration. There are two subtypes: Type I (also called Wenckebach, WB) and Type II. In general, type I, WB, does not necessarily imply cardiac disease, and is often more benign than type II. Further discussion of this is beyond the scope of this text.
- In third degree AV-block there is AV-dissociation: No atrial depolarisations are conducted to the ventricles. The ventricular contraction is determined by "escape rhythm", which may be more or less adequate and reliable. Some subjects remain asymptomatic (especially those with congenital AV block), or experience only fatigue. In other, pauses are common, and these patients may have syncopal episodes.

"Intermittent high degree AV-block" covers intermittent spells of 2. or 3. degree AV-block giving pauses of at least 3 seconds or corresponding to two or more non-conducted P-waves. Syncopal symptoms are common.

#### **5.1.2.4 Bi- and trifascicular block**

In addition, there are other conduction defects that may be of relevance, as a marker of conduction system disease and risk of intermittent high degree AV-block: Right bundle branch block (RBBB), left bundle branch block (LBBB), left anterior hemiblock (LAH) and left posterior hemiblock (LPH). The combinations of these are often called bi- and trifascicular block. They are of relevance here, because they are markers of risk of advanced block and syncope.

#### **5.1.2.5 Bradycardia during atrial fibrillation, atrial flutter or atrial tachycardia**

Although atrial fibrillation, atrial flutter and atrial tachycardia episodes are in general associated with high ventricular rates, episodes with a low ventricular rate may occur especially in patients treated with drugs like beta-blockers, digoxin and/or calcium antagonists. These low ventricular rate episodes may cause syncope. Furthermore pauses may occur after the spontaneous cessation of these tachycardias.

### **5.1.3 Supraventricular arrhythmias**

#### **5.1.3.1 Sinus tachycardia**

Sinus tachycardia is relatively common. Generally symptoms are mild and syncope is rare. It may be caused by extra cardiac abnormalities or drug use, treatment of the extra cardiac disorder or change of medication will restore normal sinus rhythm in most patients.

#### **5.1.3.2 Atrial tachycardia**

Atrial tachycardias cover a wide spectrum of tachycardias where the impulses start in the atria. They may give rise to a spectrum of symptoms, from short paroxysms of palpitations, to incessant tachycardias which over time lead to heart failure. They may occur in otherwise normal heart, but are more prevalent in structural heart disease and toxic states. Treatment is by medication or ablation, or by treatment of the underlying condition. Syncope is rare. However patients with congenital heart disease after surgery are at risk of developing atrial tachycardia which may cause syncope and even sudden cardiac death.

#### **5.1.3.3 Atrial fibrillation**

Atrial fibrillation (AF) is the most common arrhythmia. It can be paroxysmal, persistent or permanent. It can occur in structurally normal hearts, but is often caused by hypertension, being overweight or heart diseases that strain the atria. The prevalence of AF increases with age, from 0.5% at 40–50 years, to 5–15% at 80 years. The prevalence in the population is about 1-2 %, and it is estimated that more than five million Europeans have AF. AF can cause syncope or dizzy spells if the heart rate is very high or very low, or if there are pauses when paroxysms stop (before the sinus rhythm takes over). The relative risk of syncope is low, unless there has been (pre-)syncopal symptoms in that individual already. AF carries a risk of embolic stroke. This risk is dependent upon age, blood pressure, whether there is underlying heart failure, diabetes, and previous cerebrovascular episodes. A scoring system called CHADS2 or CHA2DS2VASc is used to quantify the risk of a thromboembolic event, and determine if anticoagulation is recommended. There are detailed international guidelines on anticoagulation.

#### **5.1.3.4 Atrial flutter**

Atrial flutter is a macro re-entrant arrhythmia, where an impulse travels a certain path through one of the atria (usually the right). There are many sub-types. For the purpose of this text, it can be considered as equivalent to AF, although the treatment options and underlying causes are different. The risk of embolism is thought to be equivalent to that in AF, but the data are more sparse.

#### **5.1.3.5 AV nodal re-entrant tachycardia (AVNRT)**

AV nodal re-entrant tachycardia is a common disorder occurring more often in female patients than in male patients (2:1 predominance of female patients) and, although AV nodal re-entrant tachycardia may cause serious symptoms, syncope is relatively rare. However in a patient with syncope and known AV nodal re-entrant tachycardia, the arrhythmia may be the underlying disorder.

#### **5.1.3.6 AV re-entrant tachycardia (AVRT)**

AV re-entrant tachycardia is caused by an accessory pathway. In general these tachycardias with a heart rate of about 180 beats per minute do not cause syncope. As with AVNRT, AVRT may

deteriorate in atrial fibrillation, and if associated with high ventricular rates causes severe hemodynamic symptoms.

#### **5.1.3.7 Wolff Parkinson White Syndrome (WPW)**

WPW syndrome is the combination of pre-excitation (delta wave on the ECG) and supraventricular arrhythmias. The delta-wave may be intermittently present.

WPW patients may have increased risk of atrial fibrillation which may be associated with extremely high ventricular rates. This may cause ventricular fibrillation and sudden cardiac death. Still, the sudden cardiac death rate is low (1/1000 years of patient follow-up). In rare cases ventricular fibrillation may be the first manifestation of a WPW syndrome.

#### **5.1.3.8 Supraventricular Arrhythmias in patients with structural heart disease**

Most supraventricular arrhythmias will only cause limited hemodynamic compromise in patients without structural heart disease. In contrast, in patients with structural heart disease, the arrhythmia may cause hemodynamic compromise, with dizziness or syncope, with a high risk of recurrence.

Therefore in all patients with brady- and/or tachy- arrhythmias the presence of structural heart disease should be evaluated according to current international guidelines.

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#### **5.1.5 Recommendations**

It is assumed that there are no other disqualifying conditions. In case of other disqualifying conditions, the rules for that specific condition(s) apply.

Conduction Disorder/Arrhythmia	Group 1	Group 2
Sinus Bradycardia	no restriction unless dizziness or syncope	no restriction unless dizziness or syncope
Sick sinus syndrome	If the patient has had a history of syncope, driving must cease until the condition has been satisfactorily treated/controlled (most often by a pacemaker).	If the patient has had a history of syncope or sudden incapacitation, driving must cease until the condition has been satisfactorily treated/controlled (most often by a pacemaker). Driving can be resumed only after medical assessment.
AV conduction block (excluding bundle branch block and congenital AV-block, see below)	If history of syncope and/or sudden incapacity, driving must cease until a pacemaker has been implanted or the conduction block is eliminated by other means.	If history of syncope , driving must cease until a pacemaker has been implanted or the conduction block eliminated by other means. Driving can be resumed only after medical assessment.
Bi- and trifascicular blocks	If syncopal episodes have occurred, a pacemaker should be implanted before driving can be resumed.	If syncopal episodes have occurred, a pacemaker should be implanted before driving can be resumed. In alternating RBBB and LBBB, a pacemaker must be implanted, regardless of symptoms.
Congenital AV-block	If patients have had syncopal episodes or other significant symptoms, driving must cease until a pacemaker has been implanted	Driving is not allowed unless a pacemaker is implanted
Atrial fibrillation/atrial flutter/atrial tachycardia	Driving may continue provided no history of syncope. If history of syncope , driving must cease until the condition has been satisfactorily controlled /treated.	Driving may continue provided no history of syncope and anticoagulation guidelines are adhered to. If history of syncope, driving must cease unless the underlying cause is treated and the risk of recurrence is low . Rate control during tachycardia should be adequate. Driving can only be resumed after medical assessment
AVNRT, AVRT, pre-excitation and WPW	If history of syncope , driving must cease until the	Driving may continue provided no history of

	condition has been satisfactorily controlled /treated.	syncope or other significant symptoms (e.g. palpitations with dizziness) . If so, driving must cease until the underlying cause is treated so that the risk of recurrence is low. In case of pre-excitation, driving may only be allowed after specialist assessment.
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### 5.1.6 Ventricular arrhythmias

Ventricular arrhythmias cover a wide range of clinical and electrocardiographic manifestations, ranging from asymptomatic or symptomatic premature ventricular complexes (PVCs) and nonsustained ventricular tachycardia (NSVT) in normal subjects to SCD due to ventricular tachyarrhythmias in patients with and without structural heart disease. This recommendation will not cover the topic of ICD patients which is the subject of a specific recommendation.

#### 5.1.6.1 Ventricular arrhythmia in the absence of heart disease

##### 5.1.6.1.1 Introduction

The concept of a structurally normal heart does not always imply a normal heart, as the abnormalities can be at a molecular level. Among presumably normal individuals, estimates of the prevalence of premature ventricular contractions (PVCs) and non-sustained ventricular tachycardia (NSVT) vary according to the sampling technique used. In a healthy military population PVCs recorded on standard 12-lead electrocardiograms (ECGs) had a prevalence of 0.8%, with a range of 0.5% among the subjects under the age of 20 y to 2.2% of those over 50 y of age (1).

The prognosis in PVCs and monomorphic NSVT does not seem to be altered as observed in some old studies (2, 3) especially in young patients. After the age of 30 y PVCs and short runs of NSVT began to influence risk (4). Furthermore, if the ventricular tachyarrhythmia is polymorphic, even in the absence of structural heart disease, it is an indicator of an increased risk (5).

More recent studies provide conflicting implications regarding risk in asymptomatic subjects. In one study (6), asymptomatic ventricular arrhythmias in the absence of identifiable heart disease predicted a small increase in risk, while another study (7) suggested no increased risk. In contrast to the apparently non-life-threatening implication of PVCs at rest, PVCs elicited during exercise testing or during the recovery-phase, even in apparently normal individuals, appear to imply risk over time (8, 9). A selection bias, based on indications for stress testing, may have influenced these observations.

Sustained VT is rarely observed in normal hearts without “channelopathy” (see relevant chapter). In adults, these VTs are classified as adenosine-sensitive VT and verapamil-sensitive fascicular tachycardia. Their prognosis is good (10, 11). Patients with slower, stable VT may be asymptomatic but more frequently present with a sensation of rapid heart beating possibly accompanied by dyspnea or chest discomfort. The stability or tolerance of VT is related to the rate of tachycardia, presence of retrograde conduction, ventricular function, and the integrity of peripheral compensatory mechanisms. Hemodynamically unstable VT (i.e. associated with hypotension and poor tissue perfusion) is usually, but not exclusively, observed in patients with poor ventricular function.

##### 5.1.6.1.2 Recommendations

If there is structural heart disease, syncope or a “channelopathy”, or if there is an indication for an ICD according to international guidelines, the respective rules apply.

Ventricular arrhythmia in the absence of heart disease	Group 1	Group 2
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Premature ventricular contractions and non-sustained ventricular tachycardia (NSVT)	No driving restriction provided no disabling symptoms	No driving restriction for PVCs and asymptomatic monomorphic NSVT. Driving should cease in polymorphic NSVT regardless of symptoms, and in monomorphic NSVT with disabling symptoms until satisfactorily controlled and after specialist assessment. If there is an indication for an ICD, the respective rules apply.
Sustained ventricular tachycardia	Driving must cease if there are disabling symptoms. Driving is only allowed after specialist assessment and satisfactory control of the arrhythmia.	Driving must cease if there are disabling symptoms. Driving is only allowed after specialist assessment and satisfactory control of the arrhythmia. If there is an indication for an ICD, the respective rules apply.

### 5.1.6.1.3 References

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### 5.1.6.2 Ventricular arrhythmia in structural or electrical heart disease

For this section this includes: ischemic heart disease, dilated cardiomyopathy, hypertrophic cardiomyopathy, other more rare cardiomyopathies, and channelopathies. Evidence available for ischemic heart disease is discussed as below. For the other conditions, we refer to the other relevant chapters.

#### 5.1.6.2.1 Ventricular arrhythmia at the acute phase of myocardial infarction

Ventricular arrhythmias occurring during the acute phase of myocardial infarction do not imply continuing risk over time (1), so the driving restrictions should be those of the underlying disease.

#### 5.1.6.2.2 PVCs and NSVT

Among survivors of myocardial infarction (MI), frequent and repetitive forms of ventricular ectopic activity, accompanied by a reduced ejection fraction, predict an increased risk of SCD during long-term follow-up (2-4) but the power of risk prediction conferred by the presence of PVCs and NSVT appears to be directly related to the extent of structural disease as estimated by EF and to cardiovascular limitations as estimated by functional capacity (5). So in case of PVCs or NSVT, the assessment of the fitness to drive should be based upon the evaluation of the underlying ischemic cardiomyopathy.

### 5.1.6.2.3 Sustained VT

Stable and unstable VT seem to have the same prognosis with a sudden death risk of 2.4%/year in old studies from the pre-angioplasty era (6). In case of sustained VT, a reversible condition must be searched and treated (silent ischemia for example).

### 5.1.6.2.4 Recommendations

For all recommendations: If there is an indication for an ICD, the respective rules apply. The rules for reduced ventricular function do apply and will often be more restrictive than the rules below.

	Group 1	Group 2
Premature ventricular contractions and non-sustained ventricular tachycardia	No driving restriction provided there is no other disqualifying condition (cf heart failure and ischemic cardiomyopathy chapters)	PVCs and monomorphic NSVT: Driving allowed only if asymptomatic and no other disqualifying condition (cf heart failure and ischemic cardiomyopathy chapters). polymorphic NSVT: individual assessment needed.
Sustained ventricular tachycardia	Driving must cease until the arrhythmia is satisfactorily controlled and there is no other disqualifying condition (cf heart failure, ICD and ischemic cardiomyopathy chapters).	Driving must cease until the arrhythmia is controlled for at least 3 months provided there is no other disqualifying condition (cf heart failure, ICD and ischemic cardiomyopathy chapters).

### 5.1.6.2.5 References

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## 5.1.7 Implantable Cardioverter-Defibrillators

### 5.1.7.1 Introduction

Since the introduction of the implantable cardioverter-defibrillators (ICD) in the early 1980s multiple trials (1-11) have demonstrated the efficacy of ICDs for the prevention of sudden arrhythmic death. This resulted in a significant increase in the number of implanted ICDs and more recently in a shift from secondary prevention (treatment of patients who have survived a life-threatening arrhythmia) to primary prevention (treatment of patients at risk for life-threatening arrhythmias who have never had



sustained ventricular arrhythmias). Since the first implants it has been recognised that patients treated with an ICD have an on-going risk of sudden incapacitation that might cause harm to others while driving a car (16-30). It should be emphasized that the risk is mainly a consequence of the underlying condition carrying the risk of recurrent arrhythmias and not of the presence of the ICD. However, ICD discharges could induce driver incapacitation. National and International Societies of Cardiologists published scientific statements addressing this issue (31-39). In Europe the most recent 'Consensus Report: Recommendations for Driving of Patients with Implantable Cardioverter-Defibrillators' was published in 2009 (40). Recommendations for driving will be different for patients implanted for primary and secondary prevention. In this report we use following definitions: Secondary prophylaxis is defined as implantation after resuscitated cardiac arrest, or after spontaneous arrhythmias with hemodynamic compromise, including, but not limited to syncope. Primary prophylaxis is implantation for all other reasons.

### **5.1.7.2 Recommendations for Group 1**

#### **5.1.7.2.1 Risk of harm while driving in patients implanted for secondary prevention**

Patients implanted for secondary prevention have already experienced a spontaneous life-threatening arrhythmia. Factors that determine the risk of harming themselves and others in car accidents are the likelihood that patients will experience a recurrence of their arrhythmia, the likelihood that the arrhythmia while driving will impair consciousness, the probability that such an event will result in a car accident, and the probability that the accident will result in death or injury to other road users.

##### **5.1.7.2.1.1 Risk of recurrence of arrhythmia in patients implanted for secondary prevention**

In patients with a history of ventricular tachycardia (VT) or ventricular fibrillation (VF), the 5-year actuarial incidence of appropriate ICD shocks ranges between 55% and 70% (68-72). The time between ICD implantation and recurrent arrhythmias varies among studies. Tchou et al (73) reported a high incidence of first appropriate shock during the year following implant. Subsequently, the incidence dropped to a relatively steady rate with a rise during the fifth year. In a study of 65 ICD patients, Fogoros et al (74) showed a steadily increase in the cumulative incidence of appropriate shocks. Almost 30% of patients who did not have appropriate shocks during the first 2 years subsequently had appropriate shocks during the second two years. The actuarial incidence of appropriate shocks was 28% after 6 months, 33% after 12 months, 50% after 24 months and 64% after 48 months. Lubinski et al (75) reported data from the Polish registry of 2162 patients implanted for secondary prevention of sudden cardiac death. The probability of ICD intervention for VF or fast VT during 10 years of follow-up was 52.3%. The mean time to first intervention was  $344 \pm 416$  days. Fifty per cent of patients had an appropriate ICD intervention during the first 194 days after implantation. The probability of arrhythmic episodes was 1.9% in the first month, 3.3% in the second month and 3.7% in the third month. In the three months thereafter the added probability remained below 2% per month.

##### **5.1.7.2.1.2 Risk of syncope in patients implanted for secondary prevention**

Several studies evaluated the risk of having impairment of consciousness associated with an arrhythmia or ICD shock. In a study by Kou et al (76), approximately 10% of patients who experienced a shock during follow-up had syncope associated with the shock. In this study persons who experienced syncope associated with ICD discharge could not be reliably identified prospectively by any clinical criteria, including aetiology of heart disease, severity of ventricular dysfunction, presence or absence of syncope with presenting arrhythmia, or cycle length of VT induced at the time of electrophysiological testing. Freedberg et al (77) followed 125 ICD patients implanted for secondary prevention for  $408 \pm 321$  days. During the first ICD therapy, 14% of the patients had syncope and 18% near syncope. Clinical parameters predicting symptoms of first ICD therapy included presentation with cardiac arrest and inducible VT with cycle length less than 250 ms. Bansch et al (72) retrospectively analysed data on 421 patients with an ICD followed for  $26 \pm 18$  months. Of these patients, 229 (54.4%) had recurrent VT/VF, and 62 (14.7%) had syncope. Low baseline left ventricular ejection fraction (LVEF), induction of fast VT (CL <300 ms) during programmed stimulation and chronic atrial fibrillation (AF) were associated with an increased risk of syncope. In a study of 98 patients in France (78) syncope occurred in 16 % of patient who received ICD shocks. Abello et al (79) compared 26 patients with spontaneous syncopal VT with 50 patients with non-syncopal VT prior to ICD implantation. Patients who presented with syncopal VT were more likely to experience syncope at follow-up. The median time to recurrence of syncopal VT was 376 days.

##### **5.1.7.2.1.3 Risk of harm to patients and bystanders**

Most studies evaluating the incidence of motor vehicle accidents in patients with an ICD were conducted retrospectively or based on surveys and interviews. Conti et al (80) surveyed 82 patients who were followed 6 years. Fifty-two (63%) patients in this group had defibrillator shocks. Ninety per cent of the 52 patients who received an ICD discharge resumed driving and none experienced device discharge while driving during the follow-up time period. In the study of Lerecouvreur et al (78) none of the patients who received ICD shocks at the wheel had a traffic accident. Curtis et al (81) surveyed 742 U.S. physicians who followed defibrillator patients, 452 physicians responded, and a total of 30 motor vehicle accidents related to shocks from ICDs were reported over a 12 year period. The estimated fatality rate for patients with a defibrillator was 7.5 per 100,000 person years, significantly lower than for the general population (18.4 per 100,000 person years). Of 286 defibrillator discharges documented while driving, 10.5% resulted in an accident. Trappe et al (57) examined the driving behaviour of 291 ICD patients. Fifty patients had never driven. Fifty-nine per cent of 241 patients continued driving post implant and were followed for  $38 \pm 26$  months. No patients died while driving; there were 11 accidents, but only 1 caused by the driver with an ICD and none were related to syncopal symptoms or ICD therapy. Five per cent of all patients received ICD therapy while driving; 74% of these occurred more than 2 years post implant. No patient had syncope or an accident with this event. Akiyama et al (58) administered questionnaires regarding driving to 909 patients in the AVID study. Of the 758 patients who responded 627 drove in the year prior to their index episode of ventricular arrhythmia. Fifty-seven per cent of these drivers resumed driving within 3 months after randomization, 78% within 6 months, and 88% within 12 months. Two per cent of patients had a syncopal episode while driving, and 11% had dizziness or palpitations that required stopping the vehicle. Eight per cent of the patients with an ICD received a shock while driving. Of the 55 accidents during 1619 patient years after resumption of driving, 11% were preceded by any symptom of possible arrhythmia (0.4% per patient per year). The annual incidence of accidents in the ICD population of 3.4% per patient year was substantially lower than the accident rates in the general driving population in the United States of 7.1% per person year. In this study, there was no relationship between the duration of abstinence from driving after an episode of ventricular tachyarrhythmia and the subsequent risk of a motor vehicle accident.

These studies showed that the risk of symptoms that may lead to incapacity behind the wheel, with or without a defibrillator discharge, in patients with defibrillators implanted for secondary prophylaxis is very low. However, given the methodology, these studies had limitations including the possibility of underreporting. Therefore, most recommendations on driving in patients with ICDs have been based on a prospective study of 501 patients admitted to a hospital after resuscitation from sustained VT or VF by Larsen et al (82). Outcome events, which included syncope, sudden death, ICD discharge, recurrent VF or hemodynamically compromising VT, were analysed. At the end of 1 year of follow-up, 17% of patients had experienced an outcome event. Analysis of the monthly hazard rates during this first year of follow-up indicated that the highest hazard rate was seen in the first month after discharge from the hospital. Hazard rates for months 2 through 7 were moderate, after which they declined substantially. Because only 8% of the entire group was treated with an ICD, these results predominantly reflect the results of antiarrhythmic drug therapy, including class Ia and class Ic drugs in one third of patients. The authors suggested that survivors of VT or VF should refrain from driving during the first month after hospital discharge. The moderately elevated risk for months 2 through 7 supported restricting driving for most patients until the eighth month after hospital discharge. Based on these data most national societies recommended 6 months of restriction of driving for ICD patients in secondary prevention (31-38).

For a decade there was no compelling new evidence to question these recommendations. Recently Albert et al (60) reported the results of the TOVA study: a prospective case-crossover study comparing the risk of ICD shock for VT/VF both during and up to 60 minutes after an episode of driving. Of 1188 ICD patients followed, 73 % were implanted for secondary prevention. The majority of patients (80%) reported driving a car at least once a week. Participants reported spending a median of 3.8 h/week or 2.3% of their time driving a car. Over a mean follow-up of 562 days, there were 193 ICD shocks for VT/VF with data on exposure to driving before ICD shock. The absolute risk of ICD shock for VT/VF within 1 h of driving was estimated to be 1 episode per 25116 person-hours spent driving. The risk occurred primarily during the 30-min period after driving (RR 4.46, 95% CI 2.92 to 6.82) rather than during the driving episode itself (RR 1.05, 95% CI 0.48 to 2.30). The authors conclude that the risk for ICD shock for VT/VF was not elevated during driving and the absolute risk was low.

Mylotte et al (106), performed a prospective observational study of 275 patients implanted with an ICD in three centres in the Republic of Ireland. Over half of primary and a third of secondary patients drove

within 1 month of device implantation. The indication for ICD implantation was secondary prevention in 26%. During a mean follow-up of 26 months, 36% of these patients received an ICD shock. The median time to first shock was 9 months. Appropriate shocks accounted for 52%. In the entire cohort a total of 14 motor vehicle accidents occurred following ICD implantation, giving a road traffic accident rate of 2.6%. Eight patients (3.3%) received a shock while driving at a mean of 5.4 months following implantation. Three of four appropriate shocks led to loss of consciousness resulting in minor road traffic accidents. The annual risk of a shock while driving was 1.5%.

Thijssen et al (107), prospectively followed 2786 patients implanted between 1996 and 2009 for primary and secondary prevention. In the group of secondary prevention patients (38%), during mean follow-up of 1442 days, 32% of patients received an appropriate shock. The median time to first appropriate shock was 509 days. To calculate the annual risk of harm to other road users the authors assumed a 31% risk for syncope with an ICD shock (both appropriate and inappropriate). The annual risk of harm 1 month following implantation for secondary prevention was found to be 1.8 per 100000 ICD patients, below the accepted cut-off value of 5 per 100000.

Based on the evidence described above, the expert group decided to shorten the restriction time for private driving after a life threatening ventricular arrhythmia. Since patients resuscitated for cardiac arrest very often need extensive time to recover from the event, there was consensus not to reduce the restriction time shorter than three months. Patients should have an assessment of their functional class and cognitive functions before resumption of driving.

#### **5.1.7.2.2 Risk of harm while driving in patients implanted for primary prevention**

Patients with ICDs for primary prevention are generally considered at lower risk for sudden incapacitation while driving. This is based on mortality data, rates of sudden cardiac death and rate of ICD discharges reported from primary prevention trials (4-11). Annualized mortality rates range from 1.6% of patients per year in the MADIT II trial (7) to 12% of patients per year in the COMPANION trial (10) of patients with New York Heart Association III to IV congestive heart failure. Annualized mortality rates in the other 6 trials ranged from 4% to 8.5% of patients per year. The average annual mortality in the ICD arms of these trials was approximately 7% of patients per year. Rates of sudden cardiac or arrhythmic deaths ranged from 0.5% to 1.8% of patients per year, which can be considered low. In 2 trials that used earlier-generation ICDs, device discharge rates were high. In the CABG-Patch Trial (5), 50% of patients received a discharge during 1 year of follow-up; in MADIT I (4), 60% of patients received a discharge during 2 years of follow-up. In these trials the percentage of appropriate shocks is unknown since most ICDs were committed and did not have stored electrograms. The rates of ICD discharges in more recent trials were lower. In DEFINITE (8), discharges occurred at a rate of 7.4% of patients per year. A subsequent analysis reported that only 44.9% of shocks were appropriate. In SCD-HeFT (9), 259 (31%) of the 829 patients with ICDs received shocks for any reason, with 177 of these shocks being for VF or rapid VT. During 5 years of follow-up, the annual rate of appropriate ICD discharge was 7.5% per year. In an AHA/HRS scientific statement on personal and public safety issues related to arrhythmias that may affect consciousness, Epstein et al (39) calculated the risk of likelihood of an event while driving in ICD patients implanted for primary prevention. Based on data published by Conti et al (56) the authors assume that the average person with an ICD drives 8 to 20 miles per day for purely personal reasons, which is approximately 2% of the day. When coupling these data with results of trials of primary prevention, which demonstrated ICD discharge rates of 7.5% of patients per year, the likelihood of an ICD discharge while driving is in the range of 0.15% of patients per year. The authors conclude that no private automobile driving restrictions need be applied to patients who are asymptomatic from an arrhythmia standpoint. The results of these controlled clinical trials were recently confirmed in routine clinical practice. Alsheikh-Ali et al (83) reported on the incidence and time-dependence of appropriate ICD therapy in 525 patients implanted for primary prevention in a single institute. Appropriate therapy occurred in 115 (22%) patients. The incidence of appropriate therapy was 20% in the first year after implant, 12% in year 2 and 6 to 11% per year for up to 7 years post implant. The incidence of syncope was not reported.

In the study of Thijssen et al (107), 10% of 1718 patients implanted in primary prevention with a mean follow-up of 784 days received an appropriate shock. The median time to first appropriate therapy was 417 days. Of these patients 34% received a second appropriate shock after 66 days. The risk of harm to other road users was calculated to be 0.75 per 100000 ICD patients, one month after implantation, and was below the accepted cut-off risk of 5 per 100000.

Based on these data the expert group concludes that there is no need for driving restrictions in patients implanted for primary prevention after recovery from the procedure.

#### **5.1.7.2.2.1 Recovery from ICD implantation**

In the period after ICD implantation the patient needs to recover from the procedure and wound healing needs to take place to allow safe handling of the vehicle. Most implanting physicians advise their patients to refrain from vigorous exercise and extensive use of the arm at the side of the implantation for a few weeks after implantation. Complications like lead dislocation, pocket haematoma and perforation tend to occur in this period. Therefore the expert group recommends a waiting period of two weeks before resumption of driving.

#### **5.1.7.2.3 Risk of harm while driving after ICD therapy**

##### **5.1.7.2.3.1 Risk of harm while driving after appropriate ICD therapy**

When patients experience ICD therapy for a spontaneous ventricular arrhythmia during follow-up, the risk of harm while driving is determined by the probability of a subsequent arrhythmic event and by the likelihood of symptoms of impaired consciousness. In a study by Freedberg et al (77) of 125 patients implanted with an ICD for secondary prevention, 58 patients (46%) received ICD therapy after  $152 \pm 193$  days. Only 12 patients (21%) remained free of further ICD therapy. The median freedom from ICD therapy for the second shock was only 22 days, and all second shocks occurred within one year after the initial ICD therapy. The mean time to second ICD therapy was  $66 \pm 93$  days compared with  $138 \pm 168$  days for first ICD therapy. No correlation was found between time to the first and second ICD therapy. No clinical predictor for second ICD therapy was found. In this study symptoms were similar between first and second ICD therapy. Only 2 out of 30 patient who were asymptomatic at the time of the first ICD therapy had syncope with the second ICD therapy. The authors conclude that patients presenting with asymptomatic first ICD therapy were at low risk for future syncopal ICD therapy. A similar finding was described by Bänsch et al (72). In this study, patients with slow VT and absence of syncope during the first ICD therapy had a low risk of developing future syncope. However, in the study of Kou et al (76), the absence of syncope during the first ICD therapy did not predict the absence of syncope during subsequent shocks.

In patients implanted for primary prevention, little is published on the risk of recurrent arrhythmias after the first ICD therapy. However, it is known that patients included in the MADIT II trial (90) had an increased risk of death (hazard ratio 3.4) with a high frequency of heart failure after the first appropriate ICD therapy. Sesselberg et al (91) showed that MADIT II patients had a 17.8-fold increased risk of death in the first 3 months after electrical storm, defined as three or more episodes of VT or VF in 24 hours. A study of SCD-HeFT patients (92) showed a 5.7 fold increase in mortality, mostly due to progressive heart failure, after an appropriate shock. Following the development of congestive heart failure, patients have again an increased risk for VT or VF (hazard ratio 2.52) (93). These data indicate that patients in primary prevention who receive appropriate ICD therapy are at risk for clinical deterioration and subsequent arrhythmias.

Recent data were provided by Thijssen et al (107). After the first appropriate shock, 49% of patients in secondary prevention and 34% of patients in primary prevention received a second appropriate shock. The time between the first and second appropriate shock was respectively 400 days and 66 days. Risk calculation resulted in a risk of 8 per 100000 ICD patients after 1 month in primary prevention and 6.9 per 100000 in secondary prevention. The risk following appropriate shock declined below the accepted cut-off value after 4 months in the group of implanted in primary prevention and after 2 months in the group of implanted in secondary prevention.

Based on the data described above the expert group advises a restriction from driving of three months after appropriate ICD therapy, for patients implanted for primary and for secondary prevention, especially if the patient experienced symptoms of impaired consciousness.

##### **5.1.7.2.3.2 Risk of harm while driving after inappropriate ICD therapy**

Inappropriate ICD shock are caused by atrial fibrillation, supraventricular arrhythmias and inappropriate sensing, and occur in 11 to 32% of patients enrolled in major trials (94-97). In the MADIT II population patients experiencing an inappropriate shock had a mean number of  $2.2 \pm 2.5$  inappropriate shock episodes. In the study of Mylotte et al (106), 47 % of patients received inappropriate shocks. Four inappropriate shocks occurred during driving. Although none of them were associated with loss of consciousness, two resulted in minor road traffic accidents. In the study of Thijssen et al (107), 17 % of patients implanted for secondary prevention and 10 % of patients

implanted for primary prevention experienced inappropriate shocks. The recurrence rate was respectively 34% and 27%. The risk of harm to other road users remained below the accepted cut-off value in both patient populations. Measures to reduce inappropriate shocks and to prevent recurrence of inappropriate shocks are programming of SVT-VT discrimination algorithms, antiarrhythmic medication and in case of oversensing reprogramming of the device or electrode replacement in case of lead defects (98-101). The expert group recommends that patients after receiving inappropriate shocks are allowed to drive after measures are taken to prevent recurrence of inappropriate shocks.

#### **5.1.7.2.4 Patients refusing ICD implantation**

The issue of driving restriction is often discussed at the time the patient is offered an ICD and could be one of the reasons for a patient to refuse the ICD. It should be emphasized that not the presence of the device but the underlying heart condition results in the risk for incapacitating arrhythmias. Especially patients in secondary prevention who refuse an ICD are at continuous risk for recurrence of arrhythmias and impairment of consciousness. Although we have mortality data of the control groups in the secondary prevention studies, we don't have data on the risk of sudden incapacitation. Early data (1994) from Larson et al (82) hazard rates for recurrent ventricular arrhythmias or syncope were highest in the first month after discharge from the hospital and intermediate for months 2 through 7. A significant number of patients were treated with class Ic drugs. Since then medical treatment has evolved. In the study of Akiyama et al, there was no difference between the frequency of motor vehicle accidents among the patients who had been assigned to receive antiarrhythmic-drug therapy (mainly amiodarone) and that among the patients assigned to receive an ICD. The expert group considers the risk of sudden incapacitation for patients refusing an ICD after experiencing a ventricular arrhythmia equal to the patients implanted with an ICD for secondary prevention and recommends that driving privileges are withheld for three months. For patients in primary prevention, the risk for symptomatic ventricular arrhythmias while driving is described in chapter 5.2 and is considered low (39). Therefore, patients refusing an ICD for primary prevention should have no driving restriction for private driving.

#### **5.1.7.3 Recommendations for Group 2**

##### **5.1.7.3.1 Risk assessment for professional drivers**

For private drivers the risk of incapacitation while driving is considered low based on the studies described above. However, for professional drivers, the impact of the vehicle and the time spend behind the wheel combined with the risk of incapacitation due to occurrence or recurrence of VT/VF results in an unfavourable equation. Using the 'Risk of Harm' formula (31) a yearly risk of sudden cardiac incapacitation (SCI) of 1% should be considered the maximum accepted value. Data described above show a 5-year actuarial incidence of appropriate ICD shocks between 55% and 70% in secondary prevention (68-72) and yearly ICD discharge rates of 7.5% of patients in primary prevention trials (4-11). Therefore the study group recommends permanent prohibition of professional driving after ICD implantation for secondary and primary prevention.

##### **5.1.7.3.2 Professional drivers refusing ICD implantation**

Driving restriction could be one of the reasons for professional drivers to refuse an ICD implantation. As for private drivers it should be emphasized that not the presence of the device but mainly the underlying heart condition results in the risk for incapacitating arrhythmias. For professional drivers who survived a life threatening ventricular arrhythmia (ICD indication for secondary prevention) the risk of a recurrent arrhythmia in the next year is 17%. (Larsen et al) (82). In patients with a primary indication for ICD implantation (4-11) the yearly mortality rates range from 1.6% to 12%. Rates of sudden cardiac or arrhythmic deaths ranged from 0.5% to 1.8% of patients per year. These data exceed the maximum accepted yearly risk of sudden cardiac incapacitation (SCI) for professional drivers. Therefore professional drivers should not be allowed to drive if there is an indication for ICD implantation.

##### **5.1.7.4 Clinical Follow-up and Cardiac Rehabilitation**

Many of the patients implanted with an ICD have, apart from the risk for ventricular arrhythmias, underlying conditions that may impair their ability to drive. Singh et al (93) showed that in patients from the MADIT II population hospitalization for congestive heart failure was associated with an increased risk for VT or VF (hazard ratio 2.52). Interim hospitalization for coronary events was associated with an increased risk for VT, VF, or death (hazard ratio 1.66). These results show that worsening clinical condition and cardiac instability are subsequently associated with a significant increase in the risk for

appropriate ICD therapy and death. This emphasizes the need for continued clinical vigilance during the follow-up period after ICD implantation.

ICD patients can safely exercise and should be encouraged to participate in exercise based comprehensive cardiac rehabilitation programmes (102). Cardiac rehabilitation lowers the incidence of total and exercise-related shocks and psychosocial interventions that utilise cognitive-behavioural protocols will likely prevent or reduce anxiety problems and improve quality of life (103-104). Attention to the problem of driving restriction during the rehabilitation programs could result in better adherence to the recommendations.

### 5.1.7.5 Recommendation Summary

	Group 1	Group 2
ICD implantation for secondary prevention	driving must cease for 3 months	permanent driving ban
ICD implantation for primary prevention	driving must cease for 2 weeks	permanent driving ban
ICD therapy: appropriate	driving must cease for 3 months	not applicable (permanent driving ban)
ICD therapy: inappropriate	driving must cease until measures are taken to prevent subsequent inappropriate therapy	not applicable (permanent driving ban)

### 5.1.7.6 References

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## **5.2 Syncope**

### **5.2.1 Introduction and definitions**

Syncope is a transient loss of consciousness and postural tone due to transient global cerebral hypoperfusion, characterized by rapid onset, short duration, and spontaneous, complete recovery (1). Thus, it is not a synonym for loss of consciousness in general, although it is often erroneously used in that sense. It has to be separated from epileptic seizures, comatose states, functional “pseudosyncope”, transitory, ischaemic attacks (TIA) and other conditions. Syncope is very common. Approximately 30-40% of young adults have had a syncope, and the lifetime occurrence is approximately 50% (2). Thus, driving licence legislation on syncope may affect a large proportion of the population, and have huge social and economic consequences, if it is made too strict. On the other hand, as syncope may lead to sudden incapacitation, a too lenient policy may impose a traffic hazard.

There are many causes of syncope. Most of them are covered in other chapters of this report. This chapter covers syncope presumed to be of reflex origin (vasovagal syncope, carotid sinus syndrome, situational syncope etc.), or undetermined origin, without an underlying heart disease or arrhythmia. Syncope from causes covered by other parts of this report is not covered here.

A reflex syncope is a temporary, short lasting dysfunction of the body's hemodynamic regulatory functions, which leads to an inappropriate fall in blood pressure. The blood pressure fall is usually due to bradycardia and/or vasodilation. An orthostatic challenge with reduced venous return to the heart is usually, but not invariably involved. Mental processes, fear, grief or feeling sick, may be a trigger. There is often, but not invariably, a prodrome with autonomic features (like sweating, nausea, dizziness), and often also a phase of similar symptoms after the syncope. There is no postictal confusion, like in epilepsy. Involuntary movements may occur, but these are not bilateral, synchronous and vigorous, like in generalized epileptic seizures. A "situational syncope" is a syncope in conjunction with certain bodily functions involving the autonomous nervous system. The most common are micturition, defecation, cough and swallowing. The diagnosis of a reflex syncope is based on the history, and on excluding underlying structural heart disease and arrhythmic conditions. No medical test can with certainty exclude or confirm the diagnosis. The tilt-test, which has been in widespread use, is currently considered of borderline value (1), and it cannot be used for prognosis (1). There is no universally effective treatment. Patients are advised to increase salt and water intake, respond to prodromes with sitting or lying down, and increase muscle strength in the lower extremities. A pacemaker can be used for patients in whom bradycardia is an important part of the mechanism. The only double blinded study on this (8) has not supported its use. Still, clinical experience is that carefully selected patients remain virtually syncope free after having a pacemaker, if some efforts are put into programming the device.

### 5.2.2 The risk of recurrence of syncope

Reference (2) gives an excellent summary: The risk of a recurrence increases with the number of previous syncopal episodes:

**Table 2** Prognosis of patients <40 years with T-LOC of suspected syncopal nature (modified from Sheldon and Rose<sup>3</sup>)

Number of syncopes during life	Risk of recurrence of syncope after the index episode	
	Actuarial risk 1 year (%)	Actuarial risk 2 years (%)
1	10	10
2	19	29
≥3	40	54
≥6	43	60

**Table 3** Prognosis of patients with uncertain diagnosis and low risk >40 years according the number of syncopes during life

Number of syncopes during life	Risk of recurrence of syncope after the index episode		
	Actuarial risk 1 year (%)	Actuarial risk 2 years (%)	Estimated risk 4 years <sup>a</sup> (%)
1-2	15.4	19.7	28.2
3	36.5	41.7	52.2
4-6	37.0	43.8	57.4
7-10	37.5	43.7	56.2
>10	44.3	56.4	80.7

<sup>a</sup>Assuming a linear increase.

There is an even stronger effect of the number of syncopal episodes during the previous two years:

**Table 4** Prognosis of patients with uncertain diagnosis and low risk >40 years according the number of syncopes during the previous 2 years

Number of syncopes during last 2 years	Risk of recurrence of syncope after the index episode		
	Actuarial risk 1 year (%)	Actuarial risk 2 years (%)	Estimated risk 4 years <sup>a</sup> (%)
1-2	22.8	27.5	37.1
3	29.1	35.7	48.9
4-6	43.0	50.8	66.3
7-10	43.2	48.8	59.9
>10	85.6	98.1	100

<sup>a</sup>Assuming a linear increase.

From these numbers, a recurrent syncope (of reflex or unknown origin) would disqualify from a group 1 licence, if one adheres to the approximately 22% risk acceptance "limit" that is the basis for the rest of this report, in accordance with the RH formula and the relative risk calculations. Even a single syncope would disqualify from a group 2 licence. Adopting this without further refinements, would obviously lead to a very strict policy, affecting a large part of the population.

Similar recurrence rates are found in the original literature on syncope: The VASIS study (3) was a randomized, non-blinded, multicentre study of 42 patients with 3 or more syncopal episodes over the last 2 years and a cardio inhibitory response to tilt-test. The recurrence rates were: 1%/year in subjects treated with a pacemaker, and 50% in the first and 66% during the two first (cumulative) years in the non-pacemaker group. The VPS study (4) studied 54 patients with at least 6 previous syncopal episodes (lifetime history) and a syncope on tilt-test. Over a 15 month FU the recurrence rates were 70% in the non-pacemaker group and 22% in the pacemaker group. The SYDIT (5) studied patients having had >2 syncopal episodes during the last 2 years, and a tilt test with syncope and bradycardia (<60/min). The recurrence rate after 600 days of observation (read from figure 1) was approx. 8% in the PM group and 34% in the drug group. In the drug group it seems that about 2/3 of the recurrences occurred during the first year, giving a first year risk of recurrence of about 20%. In the ISSUE study (6) there was a 34% recurrence rate over the 3-15 months FU. The authors states that this recurrence rate is in keeping with the 30-40% one year recurrence rate that one can find in the literature. There was no difference between the tilt positive and negative patients, thus, tilt test cannot be used for prognosis (6). In the ISSUE 2 study (7) there was an approximately 30% one year recurrence rate in the total study population. The VPS II study (8) was the first double blind study of pacing in vasovagal syncope. 100 patients were randomized. The patients had had at least 6 syncopal episodes ever or at least 3 episodes in the last 2 years. The 6 month recurrence rate was 40% in the "pacemaker off" group, and 33% in the "pacemaker on" group. These studies did select high risk groups which might have resulted in higher recurrence rate than in the general population of reflex syncope.

In summary, as a rule of thumb, after one syncope, the one year recurrence risk is approximately 10%. After two, it is approximately 20%. After more than two syncopal episodes the risk is 30-40%. The strongest predictor of prognosis is the number of previous syncopal episodes, especially in the near history, and the time since the last episode (1,2).

### **5.2.3 The risk of a syncope during driving**

It is common clinical knowledge that many patients have a prodrome and many have only had reflex syncope in certain situations, most often when there is an orthostatic challenge (standing, more rarely sitting). Thus, it is not self-evident that one can use the reasoning from the RH formula that the risk of an incapacitation behind the wheel is directly proportional to the yearly risk of recurrence (SCI) times the time driven (TD). In VPS (4), 4/19=21% of the syncopal episodes in the no-PM group gave an injury. In SYDIT, 28,5% of the episodes resulted in injuries (5). However, although this is an indication of the suddenness of the episodes, not giving the subject time to sit or lay down, it may not be representative of the risk of a syncope during driving. There is mainly one well known study on syncope during driving (9). This was a retrospective analysis of 3877 patients referred to a tertiary centre (the Mayo Clinic), of which 381 had their index syncope during driving. Data on circumstances and recurrences were retrieved from patient records. Thus, the study is subject to referral bias, recall bias and depend on patient willingness to share sensitive information about loss of consciousness while driving. Obviously, it did not include patients severely or fatally injured during index syncope during driving. Furthermore, it does not take into account that subjects having had a syncope while driving, could have reduced their driving afterwards, for their own safety or according to driving licence advice from their doctors or the authorities. Still, the study may give valuable insights. 9,8% of the index syncopal episodes happened during driving. This is puzzling, as the RH formula assumes that private drivers spend 4% of their time at the wheel. There were only small and probably random differences in comorbidities and causes of syncope between the driving and the not driving group. There were some differences in the types and occurrence of prodromes, and in the rate and severity of injuries (table 2 in the paper). The actuarial recurrence rate after 6 and 12 months were 12% and 14% in the driving group and 12% and 17% in the non-driving group. In the driving group, the rate of recurrence during driving was 0.7% at 6 and 1.1% at 12 months. (This is slightly more than the 0.04xthe recurrence rate, which one would have expected using the RH formula.) This led the authors to conclude that syncope during driving does not increase the risk of another syncope during driving,

compared to if the index syncope did not occur during driving. However, they did not take into account that the index syncope during driving most probably influenced the driving habits of the subject. Of note, 28.6% of the drivers had an injury due to their syncope. Since an injury from a syncope during driving can almost exclusively be due to some kind of traffic accident (a syncope sitting in the seat of a parked car could not lead to an injury), this conflicts with the assumption of a 2% risk of injury (to others) from a SCI, that is made in the parameter  $A_c=0.02$  in the RH formula (see relevant chapter). There were no data on injuries to other people.

#### 5.2.4 From recurrence rates to guidelines for driving after syncope of reflex or unknown origin

A recent, evidence based, German position paper (10), after having summarized evidence similar to that given above, concludes that there should be a 6 month driving ban in group 1 and 12 months for group 2 (taxi 6 months). Recurrent syncopal episodes are, however, not explicitly mentioned. This is problematic, as these are the ones that give the highest rate of recurrence. The ESC syncope guidelines 2009 (1), treats the topic in section 4.3. The recommendations were, for group 1: No restrictions after “single, mild reflex syncope”. After “recurrent and severe reflex syncope” driving could be resumed after “symptoms are controlled”. After unexplained syncope, the recommendation was: “No restriction unless absence of prodrome, occurrence during driving or presence of severe, structural heart disease.” For group 2, the recommendations were: After “single, mild reflex syncope”, no restriction unless occurred during high risk activity”. After “recurrent and severe” reflex syncope, a “permanent restriction unless effective treatment has been established”, was recommended. After unexplained syncope, it was recommended to ban driving until “after diagnosis and appropriate therapy is established.”

These recommendations give rise to several questions: What is meant by “after symptoms are controlled”? There will usually be a completely symptom free interval of months to years before the next episode. What is meant by “effective treatment has been established”? There is no documented, effective treatment for reflex syncope (1), as explained previously. We think that these recommendations cannot be implemented “as is”.

Syncope and epilepsy resemble each other in that both lead to a relatively sudden loss of consciousness, and they both have a time dependent risk of recurrence (11). For epilepsy, the EU has chosen to give specific rules for observation times before driving can be resumed. There is no reason why one should not treat syncopal episodes in a similar way. However, since syncope is so common, quite often has a prodrome, and in many patients require an orthostatic challenge that will not occur during driving, the recommendations should take these factors into account. Since the nature and severity of the problem varies enormously from patient to patient, the national authorities should also be allowed to make judgements on an individual basis (good prognostic factors), to ensure that the regulations will not lead to an unduly strict policy. Furthermore, there are circumstances in which a syncope bears no reasonable risk of recurrence during driving, for example syncope/collapse during severe illness or injury and some of the situational syncope types. These should be specified.

#### 5.2.5 Suggested recommendations

These recommendations cover syncope of presumed reflex origin, and syncope of unknown cause with no evidence of underlying heart disease or associated with a disposition for arrhythmia. The minimum observational periods for the main types/circumstances of syncope are given in in the table below. The national authorities may allow driving to be resumed sooner if there is compelling evidence of a low risk of recurrence during driving. (Syncopal episodes from other causes are covered elsewhere.)

Condition	Group 1	Group 2
Syncope during severe illness or injury, with volume loss and/or strong vagal activity, even if recurrent under the same circumstances	No restriction	No restriction
Syncope during medical procedures, even if recurrent under the same circumstances	No restriction	No restriction

Reflex syncope of vasovagal or carotid sinus origin, single, not occurring during high risk activity	No restriction	No restriction. Special efforts should be taken to ensure the benign nature of the incident
Situational syncope due to micturition or defecation, even if recurrent under the same circumstances	No restriction	No restriction. Special efforts should be taken to ensure the benign nature of the incident
<u>Recurrent</u> reflex syncope of vasovagal or carotid origin	Driving allowed if no recurrence in 6 months	permanent ban
Recurrent or severe situational syncope due to coughing or swallowing	Driving allowed if no recurrence in 6 months	permanent ban

### 5.2.6 References

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### 5.3 Coronary Artery Disease (CAD)

It is well recognized that CAD is a progressive condition and individuals with CAD have an on-going risk of SCI, that may cause harm to public if it occurs whilst driving. However, there needs to be a risk stratification to identify the high risk groups and high risk time period for the occurrence of a sudden disabling event. There is a lack of evidence available with regards to restrictions to driving in CAD. We highlight relevant issues and data.

#### 5.3.1 Acute coronary syndrome, stable CAD/angina, Percutaneous Coronary Intervention, Coronary Artery Bypass Graft

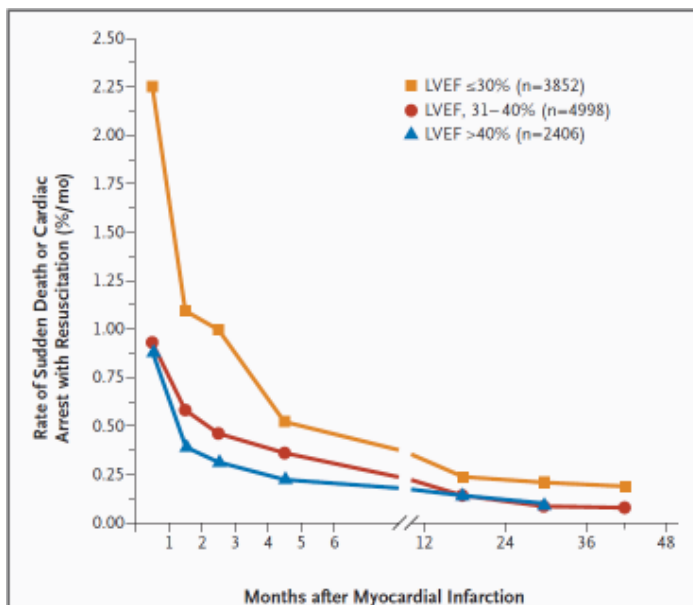
##### 5.3.1.1 Acute coronary syndrome (ACS)

This includes unstable angina, STEMI, NSTEMI. For extensive definitions, we refer to the ESC guideline (third universal definition of myocardial infarction, EHJ 2012). For the purpose of this text we describe the commonly used clinical terms:

- Unstable angina : symptoms of angina at rest with or without ECG-changes with no significant rise and fall of troponins.
- NSTEMI: Symptoms or signs of myocardial ischaemia, with or without ECG-changes, with rise and fall of troponins.
- STEMI: Symptoms or signs of myocardial ischaemia, with ST-elevation on ECG, with rise and fall of troponins.

Numerous studies are available evaluating different treatment strategies in patients admitted with ACS. However, variable endpoints are used. We selected four studies that were of particular interest to the risk of harm while driving. The Valiant trial (5) provides data on the importance of risk stratification based on the symptoms of heart failure or left ventricular function. The Valiant study (5), Olmsted County MI study (6) and the GRACE (7) study provide data on the risk for sudden death after ACS and on the high risk time period for sudden incapacitating events.

The Valiant trial evaluated 14609 patients with left ventricular dysfunction, heart failure, or both after myocardial infarction to assess the incidence and timing of sudden unexpected death or cardiac arrest with resuscitation in relation to the left ventricular ejection fraction. Table 2 below shows the event rate and cumulative incidence of events during follow-up. The risk of sudden death was highest in the first month after myocardial infarction (1.4% per month) and decreased rapidly the next months. The risk of SCD was highest in the group of patients with LV EF < 30% at about 2,25%. In the third month the incidence of SCD in this group was 1%, in the 6<sup>th</sup> month it was 0.5% and 0.37% after 12 months. The risk of SCD in the group with EF 31- 40% and greater than 40% was half of that in the group with EF <30% especially in the first 6 months (Figure 2 below).



**Figure 2. Rate of Sudden Death or Cardiac Arrest with Resuscitation over the Course of the Trial in the Three Categories of Left Ventricular Ejection Fraction (LVEF).**

The analysis was restricted to patients for whom data on LVEF were available. The average rate (percentage per month) is shown at the midpoint of each period.

SUDDEN DEATH AFTER MYOCARDIAL INFARCTION

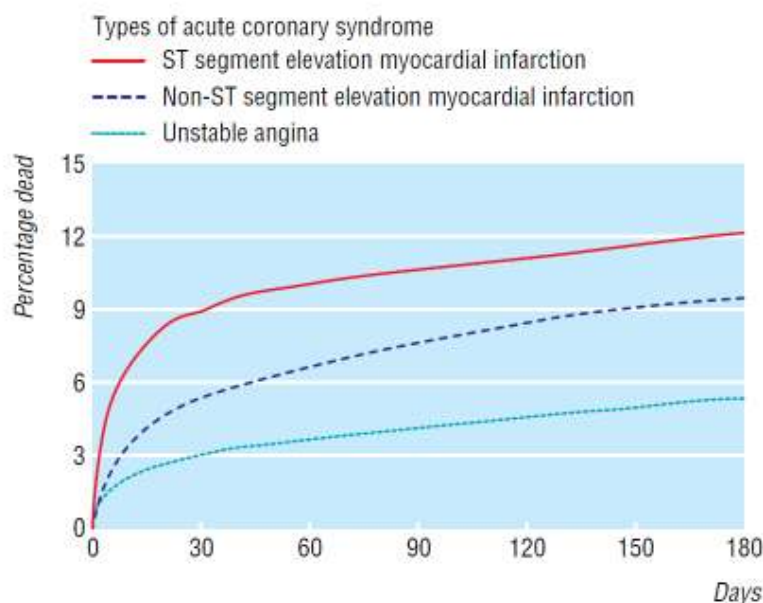
**Table 2. Event Rate and Cumulative Incidence of Events during Follow-up.\***

Time after Myocardial Infarction	No. at Risk at Beginning of Interval	No. Who Died of Any Cause during Interval	Sudden Death or Cardiac Arrest with Resuscitation		
			No. of Patients	Event Rate %/mo (95% CI)	Cumulative Incidence %
0–30 Days	14,609	589	198	1.4 (1.2–1.6)	1.4
>1–6 Mo	13,997	767	340	0.50 (0.45–0.55)	2.5
>6–12 Mo	13,157	509	211	0.27 (0.23–0.31)	1.6
>1–2 Yr	12,622	754	240	0.18 (0.16–0.20)	2.1
>2–3 Yr	7,926	244	75	0.14 (0.11–0.18)	1.7

\* CI denotes confidence interval.

An observational study in Olmsted county, USA (6), showed that within 30 days after acute MI SCD occurred in 1,2% of those affected. In the following years SCD was observed at 1,2% per year and remained constant thereafter. The risk of SCD increased significantly with onset of clinically manifest heart failure.

In the GRACE study (7) the risk of death and myocardial infarction in the first six months after presentation with acute coronary syndrome was evaluated in 43810 patients. 9% of the patients died (of which half were in hospital). In the first 15 days, mortality in patients with STEMI was 7-8% and in NSTEMI patients was 4-5%, (including in hospital mortality). Over the next two weeks there was a sharp decline in the mortality rate. By six months the risk of death in STEMI patients was similar to those with non-ST segment elevation myocardial infarction (NSTEMI) (Figure 1 below). Predictors of mortality were heart failure symptoms, advanced age and cardiac arrest on admission. We are not aware of any data of SCI after acute MI, but from the Dynamite and Iris study we infer that the sudden cardiac death rate (SCD) was about half of the total mortality. From the 'Petch assumption' (9) the SCI rate is double the SCD rate. Therefore we assume the SCI rate in this scenario equals the mortality rate.



**Fig 1** Risk of death from admission to hospital to six months after discharge (patients separated into unstable angina, non-ST segment elevation myocardial infarction, and ST segment elevation myocardial infarction)



### **Recommendations:**

Based on the above data and figures we propose following recommendations on driving after ACS:

#### **Group 1:**

Driving may be allowed provided free of symptoms. In case of significant myocardial damage driving is allowed after four weeks.

#### **Group 2:**

Driving may be allowed six weeks after the acute event provided free of symptoms and exercise or other functional test requirements can be met.

### **5.3.1.2 Stable CAD, stable angina**

For the sake of clarity, we use the following definition: history of angina with CAD but no ongoing symptoms.

The annual rate of death in stable angina is 1.5%. SCD, defined as death within 1 hour after symptom onset, occurs between 0.32%-1% per year in cases with stable angina (10,11). The PEACE investigators (8) studied SCD in patients with stable coronary disease and preserved left ventricular systolic function (EF > 40 %). The risk of SCD in this group of patients (55% with old MI) with EF 40-50%, was 0.7% per year. This shows that LV EF is a good prognostic indicator and must be an essential criterion in the assessment of fitness to drive, especially for the group 2 licence. Further data can be deducted from the 4S study. Patients with stable coronary disease (status post MI) with an elevated cholesterol were followed for a median of 5.4 years. There were 78 instantaneous deaths among the 4444 subjects during the study (data not in the 4 S paper, personal communication by dr. Terje Pedersen, first author), that is 1.7% over the years the subjects were followed. If we divide this by the median follow up time of 5.4 years, the annual rate of instantaneous death is 0.3%.

Although the risk of sudden cardiac death or syncope is low in this patient population, the symptoms of angina (e.g. chest pain) could result in an inability to safely handle the vehicle. Hence for group 2 licence driving, functional tests are required to show that symptoms do not recur with mild exercise.

### **Recommendations:**

Based on the above data we propose following recommendations on driving with stable coronary disease or stable angina:

#### **Group 1:**

Driving licences will not be issued to, or renewed for, applicants or drivers if symptomatic of angina at rest or whilst driving. Driving may be allowed to resume after treatment if it is proven that symptoms do not recur with mild exercise.

#### **Group 2:**

Driving licences will not to be issued to, or renewed for, applicants or drivers if symptomatic of angina. The functional test requirements need to be met.

### **5.3.1.3 Percutaneous Coronary Intervention (PCI)**

After PCI with stent implantation there is risk of sudden stent thrombosis. In a large meta-analysis, rate of abrupt stent closure with risk of sudden arrhythmia was 0.5% in the first 30 days after PCI. (12-15) After 30 days the risk of abrupt stent closure was only 0.3% in the following year.

### **Recommendations:**

Based on the above data we propose following recommendations on driving with PCI:

#### **Group 1:**

Driving may be allowed after elective PCI, if good clinical outcome.

#### **Group 2:**

Driving may be allowed four weeks after elective PCI if good clinical outcome provided the applicant or driver is free of symptoms, and that the functional test requirements are met.

#### **5.3.1.4 Coronary Artery Bypass Graft (CABG)**

Car driving is one of the most perceived problems by patients after cardiac surgery. Unfortunately very limited data on the capacity to drive or impairment of driving performance after a cardiac surgical procedure exist. In one small study of 74 patients (active drivers) undergoing cardiac surgery, 1 out of 3 patients stopped car driving, particularly among female gender; 1 out of 7 patients refrained from wearing a seatbelt and about half of them claimed for exemption following procedures not codified (17).

Another small study addressed impairment of neurocognitive function after coronary artery bypass grafting and its effect on an on-road driving test. Patients underwent neuropsychological examination, a standardized on-road driving test and a test in an advanced driving simulator before and 4-6 weeks after intervention. Patients deteriorated after surgery in the cognitively demanding parts like traffic behaviour ( $P=0.01$ ) and attention ( $P=0.04$ ). No deterioration was detected in the driving simulator after surgical intervention. Patients with a cognitive decline after intervention also tended to drop in the on-road driving scores to a larger extent than did patients without a cognitive decline. This study indicated that cognitive functions important for safe driving may be influenced after cardiac surgery at least temporarily (16). If there is clinical evidence of cognitive impairment, this should be assessed accordingly. So far no guidelines with regards to driving after cardiac surgery exist. Neither the European Society of Cardiology nor the European Association of cardiothoracic Surgery (EACTS) have issued guidelines or recommendations with regards to this particular topic (15).

According to a more recent publication the risk of sudden cardiac death after CABG surgery is 0.3%, 1.0%, and 1.5% at 1, 2, and 3 years (18). Since patient undergoing CABG have coronary artery disease with or without LV dysfunction, relevant regulations apply.

#### **Recommendations:**

Based on the above data we propose following recommendations on driving with CABG:

##### Group 1:

Driving may be allowed after sufficient wound healing and clinical recovery.

##### Group 2:

Driving may be allowed after sufficient wound healing, clinical recovery and functional test requirements are met.

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## **5.4 Peripheral vascular disease**

### **5.4.1 Carotid artery stenosis**

Extra-cranial cerebrovascular disease is a marker of systemic atherosclerosis. As atherosclerosis is a systemic disease, patients with extra-cranial carotid or vertebral atherosclerosis frequently have atherosclerosis elsewhere, notably in the aorta, coronary arteries and peripheral arteries. Patients with ECVD are at increased risk of myocardial infarction and death attributable to cardiac disease, such that many patients with carotid stenosis face a greater risk of death caused by MI than of stroke. Coronary atherosclerosis is prevalent in patients with fatal stroke of many origins and occurs more frequently in those with carotid or vertebral artery atherosclerosis. In 803 autopsies of consecutive patients with neurological disease, 341 patients had a history of stroke in whom the prevalence of atherosclerotic coronary plaque, more than 50% coronary artery stenosis, and pathological evidence of MI were 72%, 38% and 41% respectively as compared with 27%, 10% and 13% respectively in the remaining 462 patients with neurological diseases other than stroke (all p less than 0.001). This does indicate the high prevalence of coronary atherosclerotic disease in individuals who had stroke as compared to the individuals who had died due to other neurological diseases. Two thirds of the cases of MI found at autopsy had been clinically silent. The frequency of coronary atherosclerosis and MI was similar in patients with various strokes of types, but the severity of coronary atherosclerosis was related to the severity of the ECVD. Risk factors associated with ECVD such as cigarette smoking, hyper-cholesterolaemia, diabetes and hypertension, are the same for atherosclerosis elsewhere, although difference exist in the relative contribution to the risk in various vascular beds. A population – based study of all TIA and non-disabling stroke in Oxfordshire, UK, OXVASC study (1) looked at annual event rates (MI, MI and SCD) in these patients. It showed that in patients with no history of Coronary artery disease (angina, MI, previous coronary intervention), the annual rate of MI and SCD was about 1% in patients with maximum carotid stenosis of 30-49% and less than 1% in those who had maximum carotid stenosis <30%. In those who had maximum carotid stenosis > 50%, the annual event rate was > 2%.

**Recommendations:**

*Group 1:*

no restriction on driving. In case of stroke/TIA, the neurology section shall apply.

*Group 2:*

If significant carotid artery stenosis, driving can be allowed if the cardiac functional test requirements are met. In case of stroke/TIA, the neurology section shall apply.

**5.4.2 Thoracic and abdominal aortic aneurysm**

**5.4.2.1 Risk assessment for aortic aneurysms**

Aortic aneurysms can result in a sudden rupture which may lead to inability to drive. Usually the rupture causes a sharp pain, which may allow sufficient time to stop the vehicle. The risk of rupture increases with the diameter of the aneurysm. The natural history of aortic aneurysm is influenced by aneurysm size (diameter), rate of expansion, location, associated factors (bicuspid aortic valves, concurrent hypertension, smoking, COPD), and underlying causes (Marfan syndrome, Ehlers-Danlos syndrome, infection, inflammation, or atherosclerosis). Aneurysms carry the following complications (aortic insufficiency, rupture, dissection, thrombosis).

**5.4.2.2 Thoracic aortic aneurysm**

Thoracic aortic aneurysms grow progressively at an approximate average overall rate of 1.2 mm/year. Larger aneurysms expand faster than smaller ones, the descending aorta expands faster (0.19 cm/year) than the ascending aorta (0.07 cm/year), and the abdominal aorta expands the fastest (3-3.2 mm/year)(2,3). Therefore, it is only an issue of time before aneurysms achieve a size of clinical risk. Larger aneurysms experience faster rate of expansion. Should the patient not die of other causes in the intervening period, the patient will eventually die of the aneurysm (4). Because of a higher risk of rupture for any size of the aorta in Marfan syndrome, a lower threshold to surgical intervention is used (Table 1 below)(5).

Location of Aneurysm	Patients without Marfan Syndrome	Patients with Marfan Syndrome
Ascending aorta	>5.5 cm <sup>3</sup>	>5.0 cm <sup>3</sup>
	>5.0-5.5 cm <sup>4</sup>	>4.5 cm <sup>4</sup>
Arch	>5.5-6.0 cm <sup>4</sup>	
Descending aorta	>6.5 cm <sup>3</sup>	>6.0 cm <sup>3</sup>
Thoracoabdominal	>5.0-6.0 cm <sup>4</sup>	

Table 1. Threshold to Surgical Intervention is Lower in Patients with Marfan Syndrome.

In 2007, the validity of the 55-mm threshold for establishing unacceptable risk of acute aortic dissection of the ascending aorta was challenged; among nearly 600 cases of acute aortic dissection, the majority occurred with ascending aortic dimensions of less than 55 mm.(5)

Threshold for intervention (predicted on clinical stability of the aneurysm) is 5.5 cm for the ascending aorta (non-Marfan patient), 6.5 cm for the descending aorta. For Marfan’s disease or familial thoracic aortic aneurysm, it is recommended earlier intervention at 5.0 cm for the ascending and 6.0 cm for the descending aorta. Symptomatic aneurysms must be resected regardless of size.

Elefteriades A. John et al. (6) started by analysing the lifetime risk of rupture or dissection. Analysis revealed sharp “hinge points” in the aortic size at which rupture or dissection occur. These hinge points are seen at 6 cm in the ascending aorta and 7 cm in the descending aorta (Figure 1 below)(3,6). It is important to intervene before the aorta reaches these hinge-point dimensions. Specifically, as seen in the figure, an individual with thoracic aortic aneurysm incurs a 34% lifetime risk of rupture or dissection by the time that his or her ascending aorta reaches a diameter of 6 cm. As seen in Figure1 (below), the descending aorta does not rupture until a larger dimension.

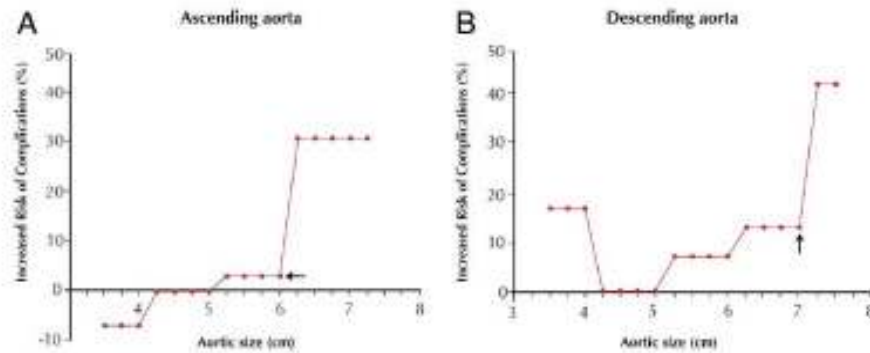


Figure 1. Depiction of “Hinge Points” for Lifetime Natural History Complications at Various Sizes of the Aorta. The y-axis lists the probability of complication; complication refers to rupture or dissection. The x-axis shows aneurysm size. (A) The ascending aorta. (B) The descending aorta. Arrows indicate discrepant diameter (2,5).

To calculate yearly growth rates required even more robust data, which have now become available in their database. This analysis reveals that the incidence of rupture, dissection, or death increases in a roughly linear fashion as the aorta grows, reaching maximal levels at an aortic dimension of 6 cm (Figure 2 below)(7). There is something special about the dimension of 6 cm, which we shall see is important mechanically as well as clinically. We have known for some years that dissections do occasionally occur at small aortic sizes (Figure 2 below) (7).

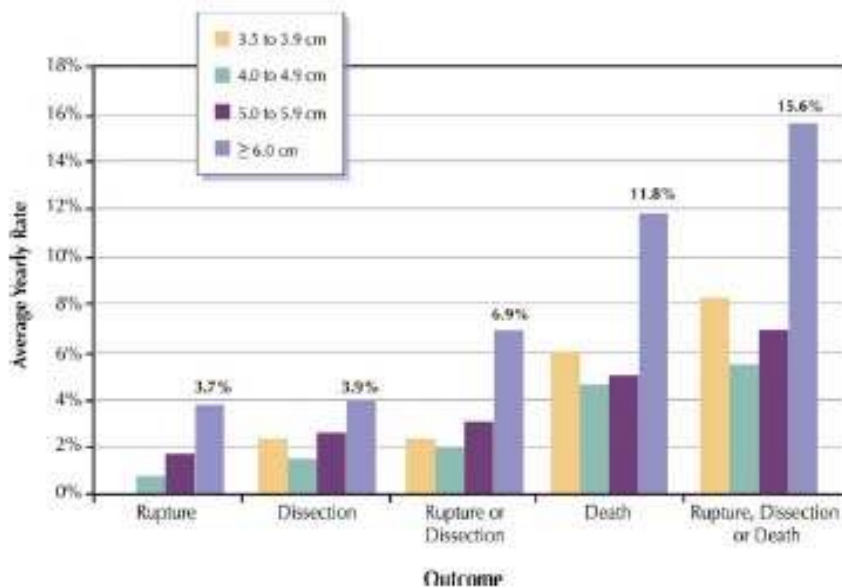


Figure 2. Yearly Rates of Rupture, Dissection, or Death Related to Aortic Size (6)

Note that the likelihood of rupture, dissection, or death within the coming year also jumps sharply for aneurysms that reach 6 cm or larger. The rates indicated for ‘rupture or dissection’, and for ‘rupture, dissection or death’, are lower than the sum of the rates in individual categories because patients with multiple complications were counted only once in the combined categories. These data underlie the conclusion that aneurysms in the ascending aorta need corrective surgery when the artery balloons to 5.5 cm (7).

The annual risk of rupture or dissection of a thoracic aortic aneurysm with a diameter > 6 cm is 6.9% per year (9), but the risk in patients with bicuspid aortic valve and additional aortic valve stenosis is higher (10).

### 5.4.2.3 Abdominal aortic aneurysm

In current clinical practice, aneurysm diameter is one of the primary criteria used to decide when to treat a patient with an abdominal aortic aneurysm (AAA). The current threshold for treatment is 5.5 cm

(11); however, many surgeons have come across gigantic AAAs (e.g., 11 or 12 cm) that have not yet ruptured, as well as small aneurysms <5.5 cm that have. There is evidence that the simple association of aneurysm diameter with the probability of rupture is not sufficient, and presumably other parameters play a role in causing an aneurysm to rupture or protecting it from rupture.

In abdominal aortic aneurysms with a diameter > 5.5 cm a risk of rupture of about 10% can be expected per year, whereas in aneurysms with a diameter of 4.0 to 5.5 cm, the annual rupture rate is 0.7-1.0% (11,12) (Table 2 below).

AAA Diameter (cm)	Rupture Risk (%)
3.0-3.9	0
4.0-4.9	1 %
5.0-5.9	1.0-1.1%
6.0-6.9	1.0-2.2 %
>7.0	3.0-3.3%

Table 2. 12-month AAA rupture risk by diameter. (10)

The reported average growth rate of AAAs between 30 and 55 mm ranges from 0.2 to 0.3 cm per year. Larger AAA variation between patients has been reported consistently.

Size of AAA is associated with risk of rupture (Figures 3 and 4 below) (13,14). AAA rupture is the initial presentation in a substantial number of cases (12). Factors associated with increasing likelihood of rupture include larger AAA diameter, presence of hypertension, presence of COPD or smoking, and aneurysm body diameter more than two times the diameter of the neck (15). The actual trigger of the rupture event is unknown (15). Rupture cannot be accurately predicted in all cases (13,14).

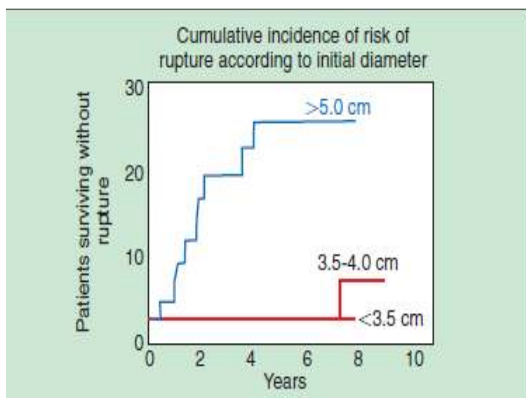


Figure 3. Risk of Rupture of Aneurysm According to the First Measured Aortic Diameter.

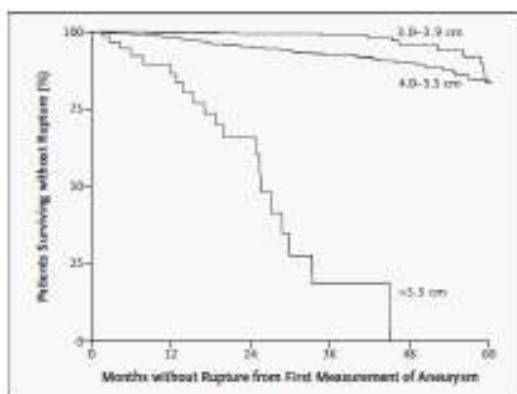


Figure 4. Risk of Rupture of Aneurysm According to the First Measured Aortic Diameter.

**recommendations:**

*Group 1:*

Driving licences will not be issued to, or renewed for, applicants or drivers if the maximum aortic diameter is such that it predisposes to a significant risk of sudden rupture and hence a sudden disabling event.

*Group 2:*

Driving licences will not be issued to, or renewed for, applicants or drivers if the maximum aortic diameter exceeds 5.5 cm.

#### **5.4.2.4 References**

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## **5.5 Heart failure**

### **5.5.1 Heart failure**

#### **5.5.1.1 Classification of heart failure**

Mild, moderate, or severe heart failure (HF) is used as a clinical symptomatic description, where mild is used for patients who can move around with no important limitations of dyspnoea or fatigue, severe for patients who are markedly symptomatic and need frequent medical attention, and moderate for the remaining patient cohort. Two classifications of the severity of HF are commonly employed. One is based on symptoms and exercise capacity [the New York Heart Association (NYHA) functional classification]. The other describes HF in stages based on structural changes and symptoms. The NYHA functional classification has proved to be clinically useful and it is employed routinely in most randomized clinical trials.

#### **5.5.1.2 Different types of heart failure**

A common description of HF distinguishes different types of the syndrome according to its mechanism (systolic vs. diastolic), its aetiology (ischaemic vs. non-ischaemic) or its clinical presentation (acute vs. chronic).

### **5.5.1.3 Epidemiology of heart failure in Europe**

HF is one of the most important causes of morbidity and mortality in the industrialized world. According to the European Society of Cardiology, within 51 European countries representing a population of 900 million, it is estimated that there are at least 15 million patients with HF. The prevalence of HF is estimated at 1–2% in the Western world, and the incidence is estimated 5–10 per 1000 persons per year, with a mean age of HF population of 74 years. In a recent US population-based study, the prevalence rate of HF was 2.2% (95% CI 1.6–2.8), increasing with age: 0.7% in persons aged 45–54 years, 1.3% in persons aged 55–64 years, 1.5% in persons aged 65–74 years, and 8.4% for those aged 75 years or older (11). Similar increasing prevalence trends were reported in the Rotterdam study: from 1% in persons aged 55–64 years to 13% in those aged 75–84 years. The rise in the incidence and prevalence of HF globally is the result of improved care of acute myocardial infarction combined with the ageing of the population and the emerging pandemic of cardiovascular disease in the developing countries. HF hospitalization represents 1–2% of all hospital admissions, which makes it the leading cause of hospitalization for patients older than 65 years. HF has a huge impact on health-related quality of life (29). It is estimated that 1–2% of all healthcare expenditure is devoted to HF in developed countries.

### **5.5.1.4 Causes and co-morbidity of heart failure**

HF is associated with ischaemic heart disease (from 46 to 68%), arterial hypertension (from 53 to 66%), diabetes (from 27 to 38%), arrhythmia, especially atrial fibrillation (from 21 to 42%), and renal insufficiency (from 17 to 53%).

### **5.5.1.5 Heart failure mortality and prognosis**

Mortality rates among groups of HF patients are highly variable and range from 5% to 75% per year. The results of both the Framingham Heart Study and a population-based study in Olmsted County, Minnesota suggested decreases of age-adjusted mortality rates in patients after the onset of HF in the last decades. However, 5-year age-adjusted mortality rates after onset of HF remained high in those two studies, with higher rates in men (50% in men vs. 46% in women for the Olmsted County population based study). The vast majority of patients with HF die from cardiovascular causes; estimates vary from 50% to 90%, depending on the HF population studied. Furthermore, the mode of death is also divergent, in that some patients die suddenly (many of ventricular arrhythmia) and others die of progressive failure of cardiac function (pump failure). Among cardiovascular causes of death, sudden cardiac death poses a major threat, with up to 50% of HF patients dying of sudden cardiac death. Patients with a nonischaemic HF aetiology have a better prognosis than those patients with an ischemic cause of HF.

### **5.5.1.6 Risk of sudden incapacitation (SCI) in heart failure according to NYHA class, EF and other risk factors**

#### **5.5.1.6.1 Heart Failure with NYHA I**

NYHA I patients are at the lowest risk for an incapacitating cardiac event and are therefore acceptable for private driving. For those patients with an EF less than or equal to 35% there is an approximately 5% annual risk of death and a 2.5% annual risk of sudden cardiac death. Commercial driving therefore is not recommended for patients with an EF less than 35%, since the acceptable annual risk for commercial drivers has been set at 1%. In patients with a LVEF above 35%, commercial driving may be allowed based on an individual decision.

#### **5.5.1.6.2 Heart Failure with NYHA II and III**

EF<35%, no sustained ventricular arrhythmia or resuscitated sudden death

The SCD-HeFT trial (1) included 2521 patients with well controlled congestive heart failure in NYHA II and III, from ischaemic and non-ischaemic aetiology. The EF had to be below 35%. The median EF was 25%, with an interquartile range of 20%-30%. Subjects were randomized to placebo, amiodarone or ICD. The main findings were that ICD treatment decreased the relative risk of death (all cause) by 23%. There was no statistically significant effect of amiodarone. A substudy (2) presents the mode of death in more detail. Over a median follow up time of 45.5 months, the proportion of subjects who died suddenly from a tachy- or bradyarrhythmia, was 4.6%, (0.9% per year) in the ICD group, 9.5% (1.9% per year) in the amiodarone group and 11.6% (2.3% per year) in the placebo group. (Annual rates obtained by dividing the total by 5 years, which probably leads to a small underestimation, since all subjects will not have been followed for the entire period.). Similar numbers are found in other studies: The annual rate of sudden death was 2.4% in the CASS-registry and 2.3% in the CABG-Patch trial (3).



Thus, patients with moderate to severe heart failure (NYHA II-III), has a 2-3% annual rate of instantaneous or sudden cardiac death, without an ICD implanted.

### 5.5.1.6.3 The rate of syncope

In SCD-HeFT, the annual rate of syncope post randomization was 8.7% in the ICD group, 7.2% in the amiodarone group and 8.6% in the placebo group (annual rate obtained by dividing the 2.5 year Kaplan-Meier rate by 2.5) (5). Only 14% of the syncopal episodes were orthostatic, and thus irrelevant to driving. The median time from randomization to syncope was 15 months. The risk of syncope was higher in NYHA III than in NYHA II subjects, higher when the QRS was broader than 120 msec and lower if beta blocker was used. The risk of having an appropriate shock from the ICD and the risk of death, were higher among subjects who had a syncope than among those without. Thus, it seems clear that the annual rate of SCI, in a population with heart failure in NYHA II and III, and an EF below 35%, is higher than 2% but lower than 22%.

### 5.5.1.6.4 Heart failure NYHA I-III, with resuscitated sudden death, symptomatic sustained VT or unexplained syncope + VT

Data from the three secondary prophylactic ICD studies, AVID, CIDS and CASH, are summarized in a meta-analysis (4). The annual rates of arrhythmic death were:

	AVID	CASH	CIDS
Amiodarone	7.4%	5.1%	4.5%
ICD	3.0%	1.5%	3.0%

No data were available on syncope in these groups. However, there should be no reason to believe that the syncope rates should be lower than in the SCD-HeFT population, of about 8%. (The syncope rate is then 1-2 times the annual rate of arrhythmic death, in keeping with the assumption that the SCI-rate is approximately the same as the annual cardiovascular mortality rate (6).) Thus, heart failure patients with an EF below 40%, and in addition resuscitated cardiac death, sustained, symptomatic VT or syncope probably due to VT, have annual rates of SCI above 2%, but below 22%.

### 5.5.1.6.5 Heart Failure with NYHA IV :

The annual total mortality risk in NYHA IV patients or unstable NYHA III patients is ranging between 13 and 25%. Driving is restricted for private drivers as well as for commercial driving because the symptoms are so severe that the driver is not able to focus on the traffic.

### 5.5.1.7 Implications for driving licence policy

Based on these data, one can safely assume that the annual rate of SCI is below 22%, even in relatively ill heart failure patients, even if they have had VT or VF. Thus, there should be no need to restrict driving in group 1 for heart failure patients in general. If the EF is below 35%, the annual rate of SCI is above 2%. Thus, having heart failure and an EF below 35%, should debar from having a group 2 licence.

	Group 1	Group 2
NYHA I	No restriction	No restriction, if LVEF >35%
NYHA II	No restriction	No restriction, if LVEF >35%
NYHA III	No restriction	Disqualified
NYHA IV	Disqualified	Disqualified

### 5.5.1.8 References

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## 5.5.2 Heart transplantation

### 5.5.2.1 Sudden death in cardiac transplant recipients

It has been claimed (by references to other studies), that 0,5%-15% of cardiac transplant (TX) recipients experience SCD (1). However, these references are not very solid evidence. The first one referenced is (2). This was a retrospective analysis of all 512 adult TX recipients in one centre operated during the years 1984 to 1994. 257 patients died during this period, that is a total mortality of 50%. Of these, 25 had SCD, defined as instant, unexpected, natural death out of hospital. Thus, 10% of all deaths were SCD. Over the study period, 5% of the subjects experienced SCD. However, even if the mean FU time was given (3,8 years), the risk of SCD per annum cannot be extrapolated reliably, since there are no survival curves, and observation time is likely not to be evenly distributed among survivors and non-survivors. There are no data on syncope.

An abstract (4) cited in the study of Tsai et al (1) showed that in a retrospective review of 532 adult heart TX recipients from 1990-9, 139 died (total mortality 26%). Of these, 58% were SCD, defined as witnessed sudden, unexpected death. The rest of the abstract gives the characteristics of these patients and the presumed underlying cause of death. From this abstract, the SCD-rate seems to be quite high. However, the material has not appeared later as a published paper (searching the first authors publications from 2001 to Oct. 2011). One of the studies cited in (1), was (5). The results of heart transplantations in a Spanish centre, and in the total Spanish registry, were given. The cause of death was given in 27,1% of the patients. SCD occurred in 0,8%. Thus, SCD accounted for 3% of the mortality mentioned. There are K-M curves for mortality, but not SCD. This paper would indicate a very low proportion of deaths being sudden, especially compared to the studies referenced here. This might be due to different definitions of SCD.

Citation # 7 in (1) is (6): This is a 100% complete follow up of 295 patients from one centre. Of the 18 patients having an "early death", only one was SCD. (There is no precise definition of "early death", but from the context, it seems they mean peri-operative.) Of the "late deaths", none of the 60 that occurred were classified as SCD. The definition of SCD was not given. No data on syncope were given. The actuarial survival was 86% one year, 74% five year and 59% ten year. This study could thus indicate a very low rate of SCD.

One more recent, probably more relevant study was identified (3) by Medline search. This was a retrospective analysis of all heart TX recipients at UCLA Medical Centre between 1994 and 2004 (10 year more recent than (2)). The mean FU was 76+/-55 months. 194/628 patients died (mortality 34%, considerably lower than 50% in the 10 year older material in (2)). Mode of death was known in 116/194, 60%. Of these, 35% had SCD, defined as cardiac death within 24 hours from onset of symptoms, i.e. not instantaneous, so not directly comparable to (2). It was specifically noted that sudden non-cardiac death was not included. The paper discusses the mechanism and underlying causes. There is a K-M graph of "freedom from SCD" (figure 2 B). It can be read out of this that after 25 months 5% had had SCD. Thus, the annual SCD-rate would be approximately 2,5%. No data on syncope are given. No data on sudden death from presumed non-cardiac causes. The problem with this paper is the definition of SCD, which definitely does not equal instantaneous death. On the other hand, syncope rate will have to be added to get the "true" SCI rate.

By references to other studies, a German position paper states that the yearly risk of sudden cardiac death, is between 0,9 and 1,6% (7). Thus, the paper states that group 2 can be held until five years after the operation. After five years, the risk is considered to be increased. However, the rate of syncope is not taken into account. If one assumes a syncope rate equal to the SCD rate (so the SCI rate is 2 x the SCD rate) (8), then the conclusion would be different.

### 5.5.2.2 Conclusion

There are conflicting data on the SCD rate in heart transplant recipients, and no data on syncope rates. Thus, the evidence base for our recommendations, is not very solid, and the group of patients is

heterogeneous. Significant problems can occur like for example transplant vasculopathy, acute rejection, electrical storm. The yearly risk of SCI is likely to be in the range of 2%. Therefore there is no restriction in group 1. Hence it would be reasonable to have an individual assessment before group 2 licence can be allowed.

### 5.5.2.3 References

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### 5.5.3 Cardiac assist devices

Patients carrying cardiac assistive devices are presumed to have a significant risk of SCI. Due to advances in the technology, the risk might decrease. Given the lack of available evidence directly related to SCI we recommend individual assessment for drivers or applicants in group 1. We recommend a permanent driving ban for group 2 licences.

## 5.6 Valvular heart disease

In the recent years the spectrum of valvular disease in industrialized countries has changed with a decrease in the incidence of rheumatic valve disease, and an increase in the incidence of degenerative valvular diseases. The incidence of endocarditis remains stable and other causes of valve disease are rare (1,2). Because of the predominance of degenerative valve disease, the two most frequent valve diseases are now calcific aortic stenosis (AS) and mitral regurgitation (MR), whereas aortic regurgitation (AR) and mitral stenosis (MS) have become less common (3).

### 5.6.1 Aortic regurgitation

Very few studies address the natural history of conservatively treated AR. Most of them focus on mortality rates and morbidity, not on sudden incapacitation risk (3-7). But this risk is low as syncope is not specifically associated to AR (8) and mortality is mainly due to heart failure. In moderately severe (III/IV) and severe (IV/IV) AR, the yearly mortality rate from cardiac cause is 3.6%, with sudden cardiac death accounting for 18.2% of these cardiac causes. The annual incidence of sudden cardiac death in moderately severe and severe AR can so be estimated to 0.7% (9). Patients with AR can develop heart failure. When in NYHA IV group 1 driving is not allowed. When in NYHA III or IV, group 2 driving is not allowed.

### 5.6.2 Aortic stenosis

Aortic stenosis (AS) is the most frequent type of valvular heart disease in Europe. The most frequent aetiology is calcific AS in adults of advanced age and the second aetiology, which dominates in the younger age group, is congenital. AS is a progressive disease and patients remain asymptomatic for years, though the stenosis can be hemodynamically severe. In symptomatic patients, the first symptom is usually exertional shortness of breath, then angina and syncope. Sudden cardiac death is a frequent cause of death in symptomatic patients but appears to be rare in the asymptomatic even in case of severe AS ( $\leq 1\%$  per year) (10-12). The severity of AS is assessed by echocardiography. Transvalvular pressure gradients and measurement of valve area are the two parameters used to quantify AS, but both can have their potential inaccuracies. Thus, AS severity has to be assessed by a combination of flow rate, pressure gradient and ventricular function, as well as functional status. AS with a valve area  $< 1.0 \text{ cm}^2$  is considered severe; however, indexing to BSA, with a cut-off value of  $0.6 \text{ cm}^2/\text{m}^2$  BSA is helpful, in particular in patients with either unusually small or large BSA.

Severe AS is unlikely if cardiac output is normal, and there is a mean pressure gradient  $< 50 \text{ mmHg}$ . In the presence of low flow, usually due to depressed LV function, low pressure gradients may be

encountered in patients with severe AS. As soon as mean gradient is <40 mmHg, even a small valve area does not definitely confirm severe AS since mild-to-moderately diseased valves may not open fully, resulting in a 'functionally small valve area' (pseudosevere AS) (13). Stress echocardiography using low-dose dobutamine may be helpful in this setting to distinguish truly severe AS from the rare cases of pseudosevere AS (14).

### 5.6.3 Mitral regurgitation

MR is now the second most frequent valve disease after AS. Leaflet abnormality is the primary cause of the disease, in opposition to ischaemic and functional MR, in which MR is the consequence of LV disease. Acute MR is poorly tolerated and obviously not compatible with driving. With chronic MR patients can remain asymptomatic for a long time, depending on the severity of the regurgitation and on the left ventricle function.

The severity of the MR is assessed by the combination of several echocardiographic parameters:

- specific signs: vena contracta width  $\geq 0.7$  cm with large central MR jet (area >40% of LA) or with a wall impinging jet of any size, swirling in LA, large flow convergence, systolic reversal in pulmonary veins, prominent flail mitral valve or ruptured papillary muscle
- supportive signs: dense, triangular continuous wave doppler MR jet, E-wave dominant mitral inflow ( $E > 1.2$  m/s), enlarged left ventricle and left atrium size
- quantitative parameters: regurgitant volume  $\geq 60$  ml/beat, regurgitant fraction  $\geq 50\%$ , effective regurgitant orifice area  $\geq 0.40$  cm<sup>2</sup>.

Left ventricular dysfunction is defined by an EF <60% and/or end systolic diameter > 45mm measured by echocardiography (15). MR usual symptoms are those of heart failure. Syncope is not primarily a MR symptom. Nevertheless some observational studies have shown an important yearly risk of sudden death, mainly depending on the functional class. Yearly linearized risk of sudden cardiac death of NYHA I, II and III and IV patients are respectively 1%, 3.1% and 7.8%. Reduced systolic function and atrial fibrillation are also associated with a higher risk (16, 17).

### 5.6.4 Mitral stenosis (MS)

Although the prevalence of rheumatic fever has greatly decreased in industrialized countries, MS still results in significant morbidity and mortality worldwide (1). Symptoms of MS are mainly related to exertion (dyspnea). One of the main complications of MS is atrial fibrillation which can lead to systemic embolism. Syncope usually occurs as a complication of severe pulmonary hypertension. No study evaluates accurately the syncope rates and the risk factor, so the recommendations we can provide are at the expert opinion level.

### Recommendations:

	Group 1	Group 2
Aortic Regurgitation	Driving must cease in NYHA IV	Driving must cease in NYHA III or IV
Aortic stenosis	Driving should cease when syncope or NYHA IV	Driving should cease in asymptomatic patients with echocardiographic signs of severe AS or when syncope or NYHA III and IV
Mitral regurgitation	Driving must cease in NYHA IV.	Driving must cease in NYHA III and IV patients and in all patients with EF below 35%
Mitral stenosis	Driving must cease in syncope patients	Driving must cease in NYHA III and IV patients, and in patient with severe pulmonary hypertension

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### 5.6.5 Valvular heart surgery

There are no available data on SCI after valvular surgery, but it is well known that disturbances of cardiac rhythm and thromboembolic events can occur. Heart failure may result from the corrected valvular disease or can be a post-operative complication. Therefore heart failure regulations apply. There should be satisfactory post-operative recovery, including wound healing to ensure safe control of the vehicle.

## **Recommendations:**

Valvular heart surgery	Group 1	Group 2
	driving allowed after sufficient wound healing, on stable anticoagulation if indicated. In case of heart failure, relevant regulations apply	driving allowed after sufficient wound healing, on stable anticoagulation if indicated. In case of heart failure, relevant regulations apply

## **5.7 Arterial hypertension**

### **5.7.1 Background**

Hypertension is a risk factor for coronary events, stroke, heart failure, peripheral artery disease and end stage renal disease. But it does not seem to be linked to road traffic accidents, as among drivers responsible for accidents, hypertension rate is not higher than in the general population<sup>1, 2</sup>. For the purpose of assessing the fitness to drive, attention must be focused on the potential relationship between hypertension and sudden collapse, functional impairments affecting vision, brain function/cognition or physical abilities/movement.

First of all, the concept of hypertension must be defined. Cardiovascular morbidity and mortality bear a continuous relationship with both systolic and diastolic blood pressures so cut-off values are difficult to establish. Though, the classification of the 2013 ESC/ESH guidelines is widely agreed upon (table 1)<sup>3</sup>. In order to find a link between hypertension and loss of consciousness, we made an extensive Pubmed search, but no direct relationship between arterial hypertension and syncope has been described. Only over treated hypertension can lead to syncope, especially in the elderly<sup>4</sup>.

But hypertension can be responsible of driver incapacitation in case of malignant hypertension. This condition embraces a syndrome of severe elevation of arterial blood pressure (diastolic blood pressure usually > 140 mmHg) with vascular damage especially retinal haemorrhages, exudates and/or papilloedema, which can lead to the sudden onset of a blurred vision. Malignant hypertension can also be complicated by cerebral haemorrhages that can affect cognition or physical abilities. So it seems reasonable to restrain these patients from driving.

Apart from this extreme condition, grade 3 hypertension is strongly and proportionally linked to stroke occurrence with an annual incidence of above 0.3% when systolic blood pressure is > 180mmHg<sup>5</sup>. So, professional drivers should not be allowed to drive until their hypertension is under control. Grade 1 or 2 hypertension can be considered as a cardiovascular risk with no immediate consequence for driving safety. It should be treated but should not be a cause of driving restriction.

**Table 3** Definitions and classification of office blood pressure levels (mmHg)<sup>a</sup>

Category	Systolic		Diastolic
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension	≥140	and	<90

<sup>a</sup>The blood pressure (BP) category is defined by the highest level of BP, whether systolic or diastolic. Isolated systolic hypertension should be graded 1, 2, or 3 according to systolic BP values in the ranges indicated.

Table: classification of hypertension used in the 2013 ESH/ESC Guidelines

### 5.7.2 Recommendations

Because of the lack of published data about hypertension and driving incapacitation, it is impossible to provide evidence-based guidelines. So the following recommendations are based on expert opinion.

	Group 1	Group 2
Malignant hypertension	No driving until under control	No driving until under control
Grade 3 hypertension (DBP ≥ 110 and/or SBP ≥ 180 mmHg)	No restriction	No driving until under control
Grade 1 or 2 hypertension (DBP < 110 and SBP < 180 mmHg)	No restriction	No restriction

### 5.7.3 References

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## **5.8 Congenital heart disease**

### **5.8.1 Background**

Congenital heart surgery has led to a major improvement in survival of patients with CHD. There is a continuously growing number of “GUCH” ie grown up congenital heart disease patients, in particular those with more complex disease. Precise data on the size and composition of the GUCH population are lacking. The 32nd Bethesda Conference report in 2001 estimated that there were 2800 adults with CHD per 1 million population, with more than half of them having moderate or high complexity of their defect.(1)

More adults than children are living with congenital heart disease, and this population is estimated to be growing at 5% per year (2). This young population wants to lead a normal life but a small part of this population (mainly those with complex defect) is threatened by arrhythmia and sudden death. In European, Canadian and American (3,4,5 ) recommendations, there is no focus on fitness to drive.

### **5.8.2 Arrhythmias and sudden cardiac death**

Arrhythmias are the main reason for admission of GUCH and they are an increasingly frequent cause of morbidity and mortality.(6 ) Risk stratification, investigation, and choice of treatment are often different from those applied to the normally formed heart. For example atrial flutter can be responsible for syncope and sudden death in atrial switch for transposition of the great arteries (7, 8,9).

The five defects with the greatest known risk of late SCD are Tetralogy of Fallot, Transposition of the great arteries (TGA), corrected TGA, aortic stenosis (AS), and univentricular heart.(10,11,12,13 ) Several risks factors in each defect have been described for sudden death, but rarely are an indications for ICD in asymptomatic patients.(3)

For example, operated Tetralogy of Fallot population is now well described but frequency of sudden death is reported as 2% by decade (14 ,15).

Indications for implantation of an implantable cardioverter defibrillator (ICD) for primary prevention have so far not been well established (3). When ICD is implanted, the recommendations for patients with ICDs can be used.

### **5.8.3 Recommendations**

In conclusion, specific data are lacking in this growing population. In symptomatic patients, a specialized work up should be suggested to assess risk factors for acute events.

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## **5.9 Structural and electrical cardiomyopathies**

### **5.9.1 Hypertrophic cardiomyopathy (HCM)**

Most individuals with HCM are asymptomatic and sudden cardiac death (SCD) can occur as the initial disease presentation (1). The annual mortality from HCM has been estimated in community-based studies in the range of 1% or less (2–7). But a minority of patients will be at high risk of SCD. Features suggesting higher risk of SCD have been derived from observational studies (2, 8, 9–17). In one study of 480 patients with a mean follow-up of 6.5 y, LV wall thickness, especially when greater than or equal to 30mm, is strongly and independently related to SCD with an annual incidence of SCD of 1.8% (18) but the independence of this criterion is not confirmed in another study in which other risk factors were also evaluated (19). The degree of outflow obstruction can predict cardiovascular death (14, 18) but not SCD (18). A history of SCD in one or more family members has been considered to signify higher risk (20), suggesting that certain specific genetic abnormalities have been associated with increased risk of SCD, but the role of genetic testing as a predictor of SCD is poor (21). Syncope has been associated with increased risk of SCD (22-25). The severity of other symptoms such as dyspnoea, chest pain, and effort intolerance has not been correlated with increased risk of SCD (26). A flat or hypotensive response to upright or supine exercise testing in patients younger than 40 y has been shown to be a risk factor for SCD, although the positive predictive value of this finding is low (27). A normal blood pressure response to exercise identifies a low-risk group (27,28). The presence of NSVT on Holter monitoring has been associated with a higher risk of SCD (29). VT induced in the EP laboratory is of limited relevance because of its lack of specificity (30).

The combination of LV wall thickness and other clinical risk factors (NSVT, abnormal blood-pressure response during upright exercise defined as a failure of systolic blood-pressure to rise by more than 25mmHg or a fall of more than 10 mmHg of the maximum blood-pressure during exercise, a family history of sudden death, recurrent or unexplained syncope) seemed to be more relevant for SCD prediction in a 5 y follow-up study of 630 patients (19).

No published dataset addresses the risk of non-fatal loss of consciousness. Syncope occurring while driving (so at rest) is most likely to be due to paroxysmal atrial fibrillation with a rapid ventricular response. Considering the lack of published data on prediction of AF in HCM patients, it would be

reasonable to consider that individuals with enlarged atria, with either palpitation or non-sustained supraventricular tachycardia on Holter are at increased risk.

**Recommendations:**

	Group 1	Group 2
Hypertrophic cardiomyopathy	Driving may continue provided no history of syncope. If history of syncope, driving must cease until the condition has been satisfactorily controlled /treated.	Driving may continue provided no history of syncope. Driving must cease when two or more of the following conditions are present : LV wall thickness > 3 cm, non-sustained ventricular tachycardia, a family history of sudden death (in a first degree relative), failure of increase of blood pressure with exercise.

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### 5.9.2 Long QT syndrome

LQTS is an inherited channelopathy, i.e. a defect in one of several ion channels, affecting the electrophysiological properties of the heart. There are several variants, of which LQT1 and LQT2 are the most prevalent. LQT3 is less common, while the other variants (LQT4-LQT7, short QT syndrome and others), are rare. LQTS is diagnosed by measuring the QT interval on an ECG (in lead II, V5 or V6) and by genetic testing. The QT is corrected for heart rate by the Bazett formula, and the QTc is a key parameter used to risk stratify. The QT can vary with time, and it is the longest QT interval measured in the individual that is used for risk assessment. LQT gives a risk of polymorphic ventricular tachycardia, called “torsades de pointes” (TdP) in this context, which can lead to syncope or sudden death. An excellent review is given in (1). There is a vast amount of literature on the risk of ventricular arrhythmias, syncope and death in various groups of LQT patients. Here we have tried to concentrate on data on the risk of sudden cardiac incapacitation (SCI), which is relevant to driving. The cited papers are selected and summarized due to perceived relevance and quality, and in the interest of keeping it short.

In an Italian cohort of consecutive LQTS-genotyped patients (n=335) on beta blockers, the five year cumulative risk of cardiac events (syncope or (aborted) sudden death) was slightly below 20% in LQT1, approx. 30% in LQT2 and slightly above 40% in LQT3 subjects (2). (Numbers read from fig. 1 in the paper.) Thus the annual risk would be estimated to approx. 6%, over all. However, many of these patients were symptomatic on beforehand, and many were children. It is well known that the risk of an event is highest early in life and higher among subjects who have already had one or more events.

According to the review paper cited above (1), the lifetime risk of (aborted) sudden death is approx. 10% for men and 20% for women with LQTS. The authors suggested that one could risk stratify as follows:

- No prior syncope and QTc < 500 ms: 5 year risk = 0.5 %
- Either a syncope or QTc > 500 ms: 5 year risk = 3%
- Having already had torsades de pointes ventricular arrhythmia or being resuscitated from cardiac arrest: 5 year risk= 14%

Note that these numbers apply to the risk of sudden death only, not the risk for syncope or the combined end point of SCI.

Many papers use data from the International LQTS Registry. One paper specifically gives the risk of the combined end point of syncope and (aborted) cardiac arrest (=SCI), in subjects between 18 and 40 years (3). This is very relevant in the context of driving licence legislation. Over all, the cumulative risk over 22 years was estimated to be approx. 10% for men and 40% for women (read from fig. 1 C in the paper). The risk (over 22 years) varied with the QTc (read from fig. 1A in the paper):

- < 5% if QTc < 440 ms
- 30% if QTc 470-499 ms
- 50% if QTc > 550 ms.

The risk of SCI also depended on the number of syncopal episodes before the age of 18 (fig. 1 B in the paper):

- Slightly below 20% if no previous episodes
- Slightly above 30% if one previous episode
- Above 50% if 2-10 episodes, and
- Well above 80% if the subject had had more than 10 syncopal episodes.
- 

This risk also depended on LQT type: LQT2 approx. 40%, LQT1 approx. 25% and LQT3 approx. 15%. Thus, the annual risk was 0,5% to 5%, depending on these risk factors. In addition, beta blockers lowered the risk compared to no beta blocker. It should be noted that this population consisted of both symptomatic subjects and subjects without any symptoms and with normal QTc on ECG (diagnosed by family screening). This clearly gives lower risk estimates than if only symptomatic subjects or subjects with prolonged QTc had been studied. It might also explain the lower numbers in this paper than in (2).

Trying to gather the information in this paper into a practical conclusion, it could be stated that if the QTc never has been measured to >500 ms and the subject has been free of syncopal events or ventricular arrhythmia, the annual risk of a SCI is below 1%. Conversely, if the subject has had any syncopal episode, or the QTc has been measured >500 ms, the risk is above 2%. Subjects with repeated syncopal episodes has an annual risk for SCI of > 20%.

The risk of (aborted) cardiac arrest is lower if the subject has survived, symptom free, to the age of 40, according to a paper specifically addressing this (4). This paper focused on the risk of sudden death, not the combined end point of SCI (i.e. including syncope), and is therefore not further discussed.

### **Recommendations:**

Even though the literature is very detailed, the recommendations have to be practical, if the rules are going to be adhered to. Only patients identified with LQT by genetic testing or ECG should be covered. It is not recommended to differentiate among the LQT subgroups or sexes, even if there are reasons to do so in the literature (see above). Young patients with the condition should be advised not to start a professional driving career, due to the consequences of a later syncope. All subjects must be aware of medications and circumstances that could provoke an arrhythmia.

LQT syndrome	Group 1	Group 2
No previous syncope or TdP, and QTc never > 500 ms	No restriction	No restriction
History of syncope, TdP <u>or</u> QTc > 500 ms	Restriction until adequate therapy has been started and is judged to be effective by specialist opinion. If ICD, rules for this also applies.	Permanent restriction

### **References:**

1. Goldenberg I, Moss AJ: Long QT syndrome. State of the art paper. JACC 2008; 51, p 2291-300.
2. Priori, SG et al: Association of LQTS Loci and Cardiac Events Among Patients Treated With Beta-blockers. JAMA 2004; 292, p 1341-44.
3. Sauer JA et al: Long QT Syndrome in Adults. JACC 2007; 49: p 329-37.
4. Goldenberg I et al: Long-QT Syndrome After Age 40. Circulation 2008; 117: p 2192-2201.

### 5.9.3 Brugada syndrome

Brugada-syndrome is the combination of certain ECG-abnormalities and a risk of malignant ventricular arrhythmia (giving syncope or SCD), due to (inherited) malfunction of the sodium channels of the heart. There are several variants, of which the so called type 1 is the only one with accepted clinical relevance. A short, good review is given in (1). The prevalence (of type 1) is probably about 0,02% in Europe and 0,1% worldwide (1). Diagnosis is mainly made by ECG, but this is sometimes difficult, even for specialists. The ECG pattern can vary over time in one individual (1). Several other conditions and disease states may mimic the Brugada ECG (1). Genetic testing may be useful. Persons with Brugada syndrome should avoid certain drugs (with sodium channel blocking properties) (2). Symptomatic subjects are often treated with an ICD. As the syndrome carries a risk of syncope and sudden, arrhythmic death, it may be relevant to driving. However, there is a tendency that these events occur during sleep or other vagal activity. This may reduce, but not eliminate, the risk of an event during driving.

The largest and most recent cohort of patients followed, is the FINGER registry (3), with 1029 subjects. Of these, 64% had been asymptomatic, 6% had had SCD and 30% had had a syncope. All were treated according to physician discretion, - 42% got an ICD. An event was defined as SCD or appropriate ICD-therapy. Note that syncope was not included in this definition of an event. Thus, the event rates are not equivalent to SCI (sudden cardiac incapacitation); they will be lower than the expected rate of SCI. However, in contrast to LQTS, where the syncope rate is several times the SCD rate, it is often said that Brugada patients have a higher risk of having a lethal arrhythmia as their first symptom. (In the FINGER registry, the syncope rate before inclusion was five times the rate of SCD (see above) (3). Since SCD subjects will have a lower likelihood of getting into the registry (only live persons were included), the ratio between syncope and SCI in unselected Brugada patients can still not be assumed to be five.) The follow up was for 14-54 months, median 32. The mean annual event rate was 1,6% over all. It was 7,7% among those who had survived a previous SCD, 1,9% in the previous syncope group, and 0,5% in the previously symptom free group. Thus, a history of symptoms was a strong predictor of risk. Family history of SCD, inducibility on electrophysiological study and sex were not statistically significant predictors.

A meta-analysis of prospective studies (4) used SCD, syncope or ICD shock as the end point. Based on studies with a total of 1545 patients, the overall event rate was 10% over 2,5 years. The relative risk was higher with a history of syncope or SCD, higher in men than in women (the disease has a male preponderance) and higher if the ECG had a spontaneous type 1 Brugada pattern. Annual event rates are not given for the subgroups, but from the relative risks given and the overall event rate, the event rates seem to be in reasonable agreement with the ones given in (3), especially since syncope was included in (4) but not (3).

A smaller, but recent study (5) enrolled 166 patients, of whom 3% had survived SCD, 35% had had a syncope and 62% had been asymptomatic. The end point was syncope, SCD or ICD shock. Over a follow up (FU) of 30 +/- 21 months, the rates were:

	Event rate over FU	Per year*
All subjects	5,4%	2,2%
Previous SCD	60%	24%
Previous syncope	8,6%	3,4%
Previously asymptomatic	1%	0,4%

\*Event rate over FU divided by mean FU time (which may lead to some underestimation, as all subjects will not have been followed for the whole period, especially the ones reaching an end point. However, the mean FU in the 9 subjects with an end point was 29,2 months).

#### **Conclusion: Risk for SCI in Brugada syndrome**

On the basis of these studies, it seems reasonable to conclude that in subjects with a Brugada ECG, but no symptoms, the annual risk of SCI is < 1%. In individuals having had one syncope, the risk is 3-4%. In individuals having had SCD, the risk of SCI is approx.10-20%.

Brugada syndrome has been diagnosed	Group 1	Group 2
No previous syncope or aborted SCD	No restriction	No restriction

Any syncope, aborted SCD or an ICD is judged to be indicated according to specialist opinion	Restriction until ICD implanted. Rules for ICD apply thereafter	Permanent restriction
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### **References**

1. Fowler SJ, Priori SG: Clinical spectrum of patients with a Brugada ECG. *Curr Opin Cardiol* 2008;24;74-81.
2. Postema PG et al: Drugs and Brugada syndrome patients: Review of the literature, recommendations, and an up-to-date website ([www.brugadadrugs.org](http://www.brugadadrugs.org)).
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4. Gehi AK et al: Risk stratification of individuals with a Brugada ECG: A meta analysis. *J. Cardiovasc Electrophysiol* 2006;17:577-583.
5. Giustetto C et al: Risk stratification of the patients with Brugada type electrocardiogram: a community-based prospective study. *Europace* 2009;11:507-13
6. Petch MC: Heart disease, guidelines, regulations and the law. *Heart* 2002; 87: 472-9
7. Veltmann C et al: Risk stratification in electrical cardiomyopathies. *Herz* 2009;34:518-27.

### **5.9.4. Other cardiomyopathies**

The risk of sudden incapacitating events shall be evaluated in applicants or drivers with well described cardiomyopathies (e.g. arrhythmogenic right ventricular cardiomyopathy, non-compaction cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia and Short QT syndrome) or with new cardiomyopathies that may be discovered. The regulations in sections 5.1.4, 5.1.5, and 9.1.6 apply. A careful specialist evaluation is required. The prognostic features of the particular cardiomyopathy should be considered.

## 6 Final recommendations: Proposition of legal text

DIRECTIVE 2006/126/EC OF THE EUROPEAN PARLIAMENT AND OF THE COUNCIL of 20  
December 2006 on driving licences

### ANNEX III MINIMUM STANDARDS OF PHYSICAL AND MENTAL FITNESS FOR DRIVING A POWER-DRIVEN VEHICLE

#### CARDIOVASCULAR DISEASES

9. Cardiovascular conditions or diseases can lead to a sudden impairment of the cerebral functions that constitutes a danger to road safety. These conditions represent grounds for establishing temporary or permanent restrictions to driving. Disease-specific guidelines are provided in the text. When more than one disease coexists, the more restrictive recommendation prevails. All cardiovascular diseases in this section carry the risk of progression, and therefore in all cases the issue or renewal of the licence may be subject to periodic assessment especially if there is risk of deterioration.

#### 9.1 DISTURBANCES OF CARDIAC RHYTHM

##### 9.1.1. Brady-arrhythmias: sinus node disease and conduction disturbances

###### Group 1:

Driving licences will not be issued to, or renewed for, applicants or drivers with a history of syncope or syncopal episodes due to arrhythmic conditions. Driving licences may be issued, or renewed, after the condition has been effectively treated and subject to competent medical authorization.

###### Group 2:

Driving licences will not be issued to, or renewed for, applicants or drivers with a history of syncope or syncopal episodes due to arrhythmic conditions. Driving licences will not be issued to, or renewed for, applicants or drivers with second degree AV block Mobitz II or with third degree AV block or alternating bundle branch block. Driving licences may be issued to, or renewed, after the condition has been effectively treated and subject to competent medical authorization.

##### 9.1.2. Tachy-arrhythmias: supraventricular and ventricular arrhythmias

###### Group 1:

Driving licences will not be issued to, or renewed for, applicants or drivers with a history of syncope or syncopal episodes due to arrhythmic conditions. Driving is not allowed for applicants or drivers with structural heart disease and sustained VT. Driving licences may be issued, or renewed, after the condition has been effectively treated and subject to competent medical authorization.

###### Group 2:

Driving licences will not be issued to, or renewed for, applicants or drivers with a history of syncope or syncopal episodes due to arrhythmic conditions. Driving licences will not be issued to, or renewed for, applicants or drivers with polymorphic nonsustained VT, sustained ventricular tachycardia or with an indication for a defibrillator. Driving licences may be issued to, or renewed, after the condition has been effectively treated and subject to competent medical authorization.

##### 9.1.3. Permanent pacemakers

###### Group 1:

Driving licences may be issued to, or renewed for, applicants or drivers after pacemaker implantation or replacement. Adequate pacemaker function and satisfactory wound healing is to be confirmed by a competent medical authority. Regular medical assessment is required.

###### Group 2:

Driving licences may be issued to, or renewed for, applicants or drivers two weeks after pacemaker implantation or replacement. Adequate pacemaker function and satisfactory wound

healing is to be confirmed by a competent medical authority. Regular medical assessment is required.

#### 9.1.4. Automatic defibrillators

##### Group 1:

Driving licences may be issued to, or renewed for, applicants or drivers after defibrillator implantation or replacement. When the defibrillator is implanted for secondary prevention, a minimum period of three months of driving cessation is imposed. When the defibrillator is implanted for primary prevention a minimum period of two weeks of driving cessation is imposed. Adequate defibrillator function and sufficient wound healing is to be confirmed by a competent medical authority. After an appropriate defibrillator shock a minimum period of three months of driving cessation is imposed. After an inappropriate defibrillator shock driving cessation is imposed until measures to prevent subsequent inappropriate therapy are taken and confirmed by a competent medical authority. Regular medical assessment including defibrillator checks is required.

##### Group 2:

Driving licences will not be issued to, or renewed for, applicants or drivers with a defibrillator.

#### 9.2. SYNCOPE

Syncope is a transient loss of consciousness and postural tone, characterized by rapid onset, short duration, and spontaneous recovery, due to global cerebral hypoperfusion. This section applies to syncope of presumed reflex origin, of unknown cause, with no evidence of underlying heart disease. National authorities may allow drivers with recognized good prognostic indicators to drive sooner than recommended below.

##### Group 1:

Driving licences may be issued to, or renewed for, applicants or drivers after a single syncope or recurrent syncopal episodes occurring in known low risk circumstances. In all other cases of recurrent syncopal episodes a minimum period of six months of driving cessation is imposed.

##### Group 2:

Driving licences may be issued to, or renewed for, applicants or drivers after a single syncope or recurrent syncopal episodes occurring in known low risk circumstances. Special efforts should be taken to ensure the benign nature of the incident by competent medical authorization. All other cases of recurrent syncopal episodes a permanent driving.

#### 9.3 CORONARY ARTERY DISEASE

Coronary artery disease may cause myocardial damage. If this results in a left ventricular ejection fraction below 35% or in functional ability estimated to be NYHA II to IV, the recommendation on heart failure should be taken into account.

##### 9.3.1. Acute coronary syndrome

##### Group 1:

Driving may be allowed provided free of symptoms. In case of significant myocardial damage driving is allowed after four weeks.

##### Group 2:

Driving may be allowed six weeks after the acute event provided free of symptoms and exercise or other functional test requirements can be met.

##### 9.3.2. Stable Angina

##### Group 1:

Driving licences will not be issued to, or renewed for, applicants or drivers if symptomatic of angina at rest or whilst driving. Driving may be allowed to resume after treatment if it is proven that symptoms do not recur with mild exercise.

##### Group 2:



Driving licences will not to be issued to, or renewed for, applicants or drivers if symptomatic of angina. The functional test requirements need to be met before Group 2 licence can be considered.

#### 9.3.3. Percutaneous coronary intervention (PCI)

##### Group 1:

Driving may be allowed after elective PCI, if good clinical outcome.

##### Group 2:

Driving may be allowed four weeks after elective PCI if good clinical outcome provided the applicant or driver is free of symptoms, and that the functional test requirements are met.

#### 9.3.4. Coronary artery bypass graft surgery (CABG)

##### Group 1:

Driving may be allowed after sufficient wound healing and clinical recovery.

##### Group 2:

Driving may be allowed after sufficient wound healing and functional test requirements are met.

### 9.4 PERIPHERAL VASCULAR DISEASE

#### 9.4.1. Carotid artery stenosis

##### Group 1:

Driving may be allowed. In case of stroke/TIA, the neurology section shall apply.

##### Group 2:

If significant carotid artery stenosis, driving can be allowed if the cardiac functional test requirements are met. In case of stroke/TIA, the neurology section shall apply.

#### 9.4.2. Thoracic and abdominal aortic aneurysm

##### Group 1:

Driving licences will not be issued to, or renewed for, applicants or drivers if the maximum aortic diameter is such that it predisposes to a significant risk of sudden rupture and hence a sudden disabling event.

##### Group 2:

Driving licences will not be issued to, or renewed for, applicants or drivers if the maximum aortic diameter exceeds 5.5 cm.

### 9.5 CONGESTIVE HEART FAILURE

#### 9.5.1. Heart failure

##### Group 1:

Driving licences may be issued to or renewed for applicants or drivers in NYHA I, II, III (if stable). Driving licences shall not be issued to or renewed for applicants or drivers in NYHA IV.

##### Group 2:

For applicants or drivers in NYHA I and II, driving licences may be issued or renewed provided that the left ventricular ejection fraction is at least 35%. Driving licences shall not be issued to or renewed for applicants or drivers in NYHA III and IV.

#### 9.5.2. Heart transplantation

##### Group 1:

Driving may be allowed for applicants or drivers if in NYHA I, II, clinically stable, and on stable immunotherapy.

##### Group 2:

Driving may only be allowed after individual assessment.

#### 9.5.3. Cardiac assist devices

##### Group 1:

Driving licences shall only be issued to /renewed after individual assessment.

Group 2:

Driving licences shall not be issued or renewed.

## 9.6 VALVULAR HEART DISEASE

### 9.6.1. Valvular heart disease

Group 1:

Driving licences shall not be issued to or renewed for applicants or drivers with aortic regurgitation, aortic stenosis, mitral regurgitation or mitral stenosis if functional ability is estimated to be NYHA IV or if there have been syncope episodes.

Group 2:

Driving licences shall not be issued to or renewed for applicants or drivers in NYHA III or IV or with EF below 35%. Driving licences shall not be issued to or renewed for applicants or drivers with mitral stenosis and severe pulmonary hypertension and for applicants or drivers with severe echocardiographic aortic stenosis or aortic stenosis causing syncope.

### 9.6.2. Valvular heart surgery

Group 1 and 2:

Driving may be allowed if satisfactory wound healing, clinical recovery and on stable anticoagulation if indicated.

## 9.7. ARTERIAL HYPERTENSION

Malignant hypertension is defined as elevation in systolic blood pressure  $\geq 180$  mmHg or diastolic blood pressure  $\geq 110$  mmHg associated with impending or progressive organ damage, such as major neurological changes, hypertensive encephalopathy, cerebral infarction, intracranial haemorrhage, acute heart failure, acute pulmonary oedema, aortic dissection, renal failure, or eclampsia.

Group 1:

Driving licences may not be issued to or renewed for applicants or drivers with malignant hypertension until treatment resolves the symptoms described above.

Group 2:

Driving licences may not be issued to or renewed for applicants or drivers with malignant hypertension or with grade III blood pressures (diastolic blood pressure  $\geq 110$  mmHg and/or systolic blood pressure  $\geq 180$  mmHg) until treatment resolves the symptoms and ensures that the blood pressure remains stable below these thresholds.

### 9.8. Congenital heart disease

Group 1 and 2:

Driving licences may be issued to or renewed for applicants or drivers with congenital heart disease with or without surgical correction. Individual assessment is necessary, taking into account the complexity of the defect and the higher risk for complications (e.g. arrhythmia and sudden cardiac death).

## 9.9 STRUCTURAL AND ELECTRICAL CARDIOMYOPATHIES

### 9.9.1. Hypertrophic cardiomyopathy

Group 1:

Driving licences may be issued to or renewed for applicants or drivers without syncope.

Group 2:

Driving licences shall not be issued to, or renewed for, applicants or drivers with history of syncope or when two or more of the following conditions present : LV wall thickness  $> 3$  cm, non-sustained ventricular tachycardia, a family history of sudden death (in a first degree relative), no increase of blood pressure with exercise.

### 9.9.2. Long QT syndrome

#### Group 1:

Driving licences shall not be issued to or renewed for applicants or drivers with previous syncope, Torsade des Pointes or QTc has ever been > 500 ms. Driving may resume if therapy has been started and judged to have brought the yearly risk of sudden incapacitating event below 22%. In case of therapy with automatic defibrillator, relevant recommendations shall also apply.

Group 2: Driving licences shall not to be issued to, or renewed for, applicants or drivers with previous syncope, Torsade des Pointes and QTc has ever been > 500 ms. This restriction is permanent.

### 9.9.3. Brugada syndrome

Group 1: Driving licences shall not be issued to or renewed for applicants or drivers with previous syncope or aborted sudden cardiac death. In case of therapy with automatic defibrillator, section 9.1.4 shall apply.

#### Group 2:

Driving licences shall not to be issued to, or renewed for, applicants or drivers with previous syncope or aborted sudden cardiac death. This restriction is permanent.

### 9.9.4. Other cardiomyopathies

The risk of sudden incapacitating events shall be evaluated in applicants or drivers with well described cardiomyopathies (e.g. arrhythmogenic right ventricular cardiomyopathy, non-compaction cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia and Short QT syndrome) or with new cardiomyopathies that may be discovered. The regulations in sections 9.1.1, 9.1.2 and 9.1.4 apply. A careful specialist evaluation is required. The prognostic features of the particular cardiomyopathy should be considered.