

Recommendations and cardiological evaluation of athletes with arrhythmias

Part 2

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Confronted with a competitive or recreational athlete, the physician has to discriminate between benign, parapsychological and pathological arrhythmias. Benign arrhythmias do not represent a risk for SCD, nor do they induce haemodynamic consequences during athletic activities. These arrhythmias are not markers for heart disease. Parapsychological arrhythmias are related to athletic performance. Long periods of endurance training induce changes in rhythm, conduction and repolarisation. These changes are fully reversible and disappear when the sport is terminated. Pathological arrhythmias have haemodynamic consequences and express disease, such as sick sinus syndrome, cardiomyopathy or inverse consequences of physical training. Arrhythmias can be classified as bradyarrhythmias and tachyarrhythmias. Conduction disorders can be seen in fast as well as in slow arrhythmias. (*Neth Heart J* 2004;12:214-22.)

Key words: arrhythmias, athletes, evaluation, recommendations

Bradyarrhythmias

Sinus bradycardia

Sinus bradycardia is defined as a rhythm lower than 50 beats/min. Related to the type of sporting activity 50 to 90% of athletes show sinus bradycardia with a mean heart rate of 50 beats/min.¹ Sinus bradycardias with rates of 25 beats/min are recorded especially at night. Sinus arrhythmia is a very common finding. There is a wide range in prevalence of sinus arrhythmia in athletes, ranging from 13 to 91%. Asymptomatic sinoatrial pauses of between 2 to 3 sec are commonly recorded in endurance athletes.^{2,3} A resting sinus rate below 40 beats/min that does not increase more than 100 beats/min during exercise is an abnormal finding and cardiological evaluation is indicated. ECG and Holter monitoring can be used and sometimes electrophysiology studies (EPS) in specific cases. Symptomatic pauses of more than 3 sec are abnormal⁴ and require cardiological evaluation including ECG, Holter monitoring, exercise testing, echocardiography and EPS.

Sick sinus syndrome

The prevalence of this disorder is not well defined. In the literature, a prevalence of 0.17% is mentioned by Kulbertus, et al.⁵ Several intrinsic as well as extrinsic factors can cause sick sinus syndrome. Longstanding isotonic loading causes volume stress of the myocardium and results in a dilatation of the heart chambers. It has been suggested that the process of dilatation of the atria and ventricles in combination with extreme vagal stimulation and changes in the collagen/fibrosis ratio could be a substrate for the development of sick sinus syndrome.⁶ The changes at atrial level could disrupt the interaction between pacemaker cells. It is likely that genetic predisposition contributes to this syndrome.

Recommendation

Athletes with presyncope or syncope should not participate in sports because loss of consciousness may

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be hazardous for the athlete himself or others. Athletes with a normal or structurally abnormal heart in which the bradyarrhythmia is asymptomatic and disappears with exercise and in which the (sinus) bradycardia rate increases appropriately during exercise may participate in all competitive sports. They have to be re-examined periodically to determine that training does not aggravate the bradyarrhythmia.

Athletes with symptoms such as impaired consciousness and fatigue related to the arrhythmia should be treated and should not participate in sports because loss of consciousness may be hazardous for the athlete himself or others. If the athlete is asymptomatic for a six-month period during treatment, he may participate in all competitive sports after cardiological re-examination. Athletes with symptomatic tachy-brady syndrome or an inappropriate increase of exercise heart rate should be treated. If asymptomatic for six months, they may participate in low-intensity competitive sports. Athletes with pacemakers should not engage in sports with danger of bodily collision. (Class IC: field hockey, soccer. Class IIA: diving, motorcycling. Class IIB: rugby. Class IIC: basketball, ice hockey, team handball. Class IIIA: bobsledding, karate, judo, water-skiing. Class IIIB: downhill skiing, wrestling. Class IIIC: boxing).

Disorders of atrioventricular conduction

In the athletic population AV conduction disorders are not uncommon and it is assumed that these disorders are related to the intensity and length of the training period.⁷

First-degree AV block

First-degree AV block is a benign conduction abnormality and the incidence is between 10 and 33% in athletes compared with 0.65% in the general population.³

Second-degree AV block

Second-degree AV block could be a sign of organic heart disease, but is not uncommon in endurance athletes. The incidence of type I second-degree AV block is between 23 and 40% in athletes compared with 5.7% in a normal population.³ Exercise-induced second-degree AV block is uncommon and clinically important because it can result in significant dyspnoea or syncope during sporting activities.

Recommendation

Athletes with normal hearts and no worsening or improvement of the AV block with exercise may participate in all competitive sports. Athletes with structural heart disease, in whom AV block disappears or does not worsen with exercise or recovery, may participate in all competitive sports, as determined by limitations of the cardiac abnormality. Athletes without symptoms, in whom the AV block initially appears to worsen with exercise or during the recovery period

should undergo cardiological evaluation. If the AV block is infranodal permanent pacing may be required.

Second-degree type II AV block

This type of AV block occurs in 8% of athletes.³ This abnormality is often related to a structural heart disorder. Evaluation of athletes with this abnormality is similar to athletes with type I AV block.

Recommendation

Athletes with intrahisian and infrahisian type II second degree AV block should be treated with permanent pacing before athletic activity.

Acquired complete heart block

Acquired complete heart block is rare in athletes. It can be permanent or transient. The incidence lies between 0.02 and 0.00017%. In a normal population the incidence is 0.0002%.³ If third degree AV block is present evaluation is necessary to exclude underlying heart disease. In some cases vagal over-stimulation plays an important role and a period of detraining could be successful in the management of a symptomatic athlete with a third degree AV block.

Recommendation

Athletes with normal hearts and asymptomatic transient complete AV block that disappears during exercise and who show an appropriate increase in heart rate may participate in all competitive sports. Athletes with symptoms of fatigue, near syncope or syncope should have a pacemaker implanted before they participate in competitive sports.

Atrioventricular escape and junctional rhythm

Atrioventricular escape and junctional rhythms are common arrhythmias in athletes.³ They are the result of training and vagal over-stimulation. Clinical evaluation and recommendations are the same as those for symptomatic athletes with sinus node dysfunction.

Recommendation

Athletes with normal hearts and normal heart rate response to activity without sustained AV nodal or AV junctional tachycardia may participate in all competitive sports. Athletes with structural heart disease may participate in competitive sports, depending on the limitations of the structural heart disease.

Bundle branch block

The mean QRS width in athletes is between 90 and 100 msec. Incomplete right bundle branch block (RBBB) is common in highly trained athletes. For male athletes the incidence is 16.7% and for female athletes it is 0.2%. The incidence of a complete RBBB in female athletes is 0.2% and for male athletes 1.2%.⁸ Complete left bundle branch block (LBBB) is uncommon in athletes and is probably more likely to be associated with underlying heart disease than RBBB.^{9,10} Exercise-induced

LBBB is a rare electrocardiographic observation in athletes. LBBB may represent heart disease and requires full examination.

Recommendation

Athletes with asymptomatic, incomplete RBBB and no underlying heart disease may participate in all competitive sports, as may athletes with an RBBB and an LBBB (with or without left-axis deviation), in whom no AV block develops with exercise and who have no symptoms. Athletes with a normal HV interval and a normal AV conduction response to pacing have no restrictions either. Athletes with significant prolongation of the HV interval (>90 ms) or with a His-Purkinje block should have a permanent pacemaker implanted before sport activity. Athletes with interventricular conduction abnormality and structural heart disease may participate in competitive sports, depending on the limitations of the underlying heart disease.

QT interval

In general the QT interval and the corrected QT is prolonged in athletes and is caused by the relative bradycardia.³ The QT interval in women is more prolonged at low heart rates than in men.¹¹ Pathological prolongation of the QT interval occurs in some electrolyte-related disorders, diet deficiencies and abuse of drugs. Some medications could induce torsade de pointes arrhythmias. A lack of appropriate shortening of the QTc interval during exercise could suggest LQTS. The predictive value of the QT dispersion in relation to ventricular arrhythmias in athletes is subject for debate.¹²

Recommendation

Athletes with a prolonged QT and QTc interval, in whom the interval prolongation persists during exercise, could be at risk for LQTS and should not participate in sporting activities even in the absence of documented ventricular arrhythmias.

Tachyarrhythmias

Sinus node reentry tachycardia and atrial tachycardia

Sinus node reentry tachycardia is an uncommon finding in athletes. The average heart rate is between 130 and 140 beats/min. Atrial tachycardia is extremely rare in athletes. The atrial rate is generally between 150 and 200 beats/min. Underlying heart disease could possibly be a cardiomyopathy. Automaticity or a reentry phenomenon can cause atrial tachycardia which develops after surgery. RF catheter ablation could be an effective therapeutic option. Evaluation can be done by ECG, Holter monitoring and echocardiography.

Recommendation

Athletes with atrial tachycardia or sinus node reentry tachycardia should first be considered for EPS and RF catheter ablation. If there is no recurrence of the

arrhythmia after four to six months and if there is no structural heart disease, the athlete may participate in all competitive sports. Athletes with atrial tachycardia without structural heart disease, with ventricular rates comparable with those of an appropriate sinus tachycardia during physical activity with or without therapy, have no restrictions either. Athletes with atrial tachycardia and structural underlying heart disease should only participate in competitive sports consistent with the limitations of the heart disease.

AV nodal reentry tachycardia

The prevalence of this arrhythmia in the athletic population is equal to that in the normal population and accounts for about 50% of all cases for supra-ventricular tachycardia. Evaluation is carried out by ECG, echocardiogram to exclude structural heart disease and a stress test. EPS could be helpful if the diagnosis is uncertain. The preferred therapy is RF catheter ablation.

Recommendation

In the absence of structural heart disease, athletes with asymptomatic nonsustained episodes of this arrhythmia that are not induced by exercise and do not aggravate in duration during exercise may participate in all competitive sports. Athletes with symptomatic arrhythmias or with reproducible exercise-induced tachycardia should be treated with RF catheter ablation. If there is no recurrence four to six months after ablation all competitive sports are allowed. Athletes with structural heart disease after undergoing RF catheter ablation may only participate in competitive sports in accordance with the limitations of the heart disease.

Atrioventricular circus movement tachycardia, including Wolff-Parkinson-White syndrome

Atrioventricular circus movement tachycardia occurs in 40% of cases of supraventricular tachycardia. Orthodromic circus movement tachycardia occurs in 90 to 95% and antidromic circus tachycardia in 5 to 10% of cases. A concealed AV-bypass tract can be the cause of the circus movement tachycardia (30 to 40%). In case of an atrioventricular reentry tachycardia the extra pathway is usually located in the left lateral free wall (50%) (posteroseptal 30%, right anteroseptal 10% and right lateral 10%).¹³ The prevalence of atrial fibrillation in patients with atrioventricular reentry tachycardia is 40%.⁹ For evaluation, ECG, exercise test and 24-hour Holter during sport activities are indicated and echocardiography is used to exclude underlying heart disease.

Recommendation

Athletes with symptomatic atrioventricular reentry tachycardia should undergo EPS and RF catheter ablation of the accessory pathway regardless of its conduction properties. Athletes without structural heart disease and with atrioventricular reentry tachy-

cardia and concealed pathways have similar recommendations to those with atrioventricular nodal reentry tachycardia. Athletes with episodes of atrial fibrillation whose maximal ventricular rate at rest (without therapy) due to conduction over the accessory pathway is <240 beats/min and who have no episodes of syncope or near syncope and have no structural heart disease appear to have low risk of sudden cardiac death and may participate in all competitive sports. Athletes with syncope or near syncope or episodes of atrial flutter or atrial fibrillation whose maximal ventricular rate at rest (without therapy) due to conduction over the accessory pathway exceeds 240 beats/min, should be scheduled for ablation and are restricted to Class IA sports. After successful ablation, athletes who are asymptomatic and with no recurrence of the tachycardia for three to six months have no restrictions for competitive sports.¹⁴ Athletes with underlying heart disease may only participate in competitive sports in accordance with the limitations of the heart disease.

Atrial fibrillation and flutter

Prevalence of atrial fibrillation in athletes is higher than in the general population of the same age and is usually not related to structural heart disease.^{3,15,16} In most cases atrial fibrillation could be classified as lone atrial fibrillation. Long-term vigorous exercise, however, may trigger atrial fibrillation.¹⁷ Atrial fibrillation in young healthy athletes can be induced vagally and can be due to an overtraining syndrome. Atrial fibrillation could be the first symptom of a more serious underlying heart disease. There are data that lifelong endurance training induces more arrhythmias than a sedentary life.¹⁸ ECG, exercise test and 24-hour Holter during sport activity are used to evaluate atrial fibrillation/flutter and echocardiography can be performed to exclude underlying heart disease.

Recommendation

Athletes with atrial fibrillation in the absence of WPW and other structural heart disease who maintain a ventricular rate comparable with that of an appropriate sinus tachycardia during physical activity with or without therapy may participate in all competitive sports. It is necessary to evaluate the arrhythmia therapy by Holter monitoring during sport activities. For evaluating possible proarrhythmic activity of the antiarrhythmic drugs, an exercise test should be done one week after starting the specific drug.

Athletes who have atrial fibrillation in combination with an underlying heart disease and appropriate increase in ventricular rate during activity may participate in sports dependent on the limitations of their structural heart disease.

Therapeutic remarks

Treatment of athletes with atrial fibrillation is initially no different from the treatment of nontrained subjects. The application of the recommendations for atrial

fibrillation is essentially the same for athletes and nontrained subjects.^{9,14,19} Underlying heart disease and causal factors such as rheumatic heart disease, mitral valve prolapse, hyperthyroidism, pulmonary embolism, alcoholism and drugs have to be determined. Medical therapy, anticoagulation, antiplatelet therapy and electrical cardioversion of atrial fibrillation are not different in an athlete population. Athletes who require anticoagulation should not participate in sports because of risk of bodily collision initiating serious bleeding.^{9,14} Class IC antiarrhythmic drugs are effective in vagal-induced atrial fibrillation while exercise-provoked atrial fibrillation could be successfully treated with a low dose of β -blocker or sotalol. In a substantial number of athletes sotalol has proved to be ineffective and in a subgroup of athletes it even worsens symptoms and provokes side effects. The bradycardia in some athletes is one of the problems and makes it difficult to choose the right therapy, if drug-therapy has failed. Focal ablation or Maze procedure is possibly a better option.

Training and atrial fibrillation

Atrial fibrillation can be seen as a manifestation of the overtraining syndrome and can disappear after interruption training. Training should be interrupted for three to five days. After this rest period the training schedule can be resumed with the restriction that the total volume of the training should be lowered to half the usual programme. Each training session is alternated with a rest day and one competition day should be substituted for a training session. In severe overtraining, the training has to be interrupted for several weeks and a low-intensity training could be advised.^{20,21} After an interruption of competition and training atrial fibrillation can disappear in some athletes. It is recommended to evaluate the effect after a period of eight weeks. With an adrenergically induced atrial fibrillation, high-intensity training bouts should be avoided.

Atrial flutter is uncommon in a young athletic population. Paroxysmal atrial flutter can occur in the absence of structural heart disease, while chronic atrial flutter is likely to be associated with underlying heart disease. In 20 to 30% of cases athletes also show atrial fibrillation. Atrial flutter can be classified as common and uncommon. The flutter rate of the common type is 250 to 350 beats/min. For the uncommon type the rate is between 350 to 450 beats/min. A rapid 1:1 ventricular response can occur with the use of IA and IC antiarrhythmic drugs that slow down the atrial flutter or by enhancing AV conduction with exercise. RF ablation is the preferred therapy for the common type of atrial flutter. The uncommon type could be treated medically but RF ablation is also possible. For evaluation, see atrial fibrillation.

Recommendation

Athletes with the common form of atrial flutter should be considered for RF catheter ablation. After ablation they may participate in low-intensity sports and if there

is no evidence of arrhythmia recurrence after four to six months all competitive sports are permitted. Athletes with the uncommon form of atrial flutter should be treated medically.

Athletes on medical treatment without structural heart disease and who maintain an appropriate ventricular rate during exercise may participate in low-intensity sports. Full participation in all categories is allowed if there is no evidence of recurrence arrhythmia for a period of four to six months. Athletes with atrial flutter and structural heart disease may participate in low-intensity sports depending on the limitations of the heart disease. Athletes on medical treatment should be examined annually.

Ventricular arrhythmias

Ventricular tachycardia is uncommon in athletes and frequently occurs in the presence of structural heart disease. Ventricular tachyarrhythmias can occur in the absence of (detectable) heart disease. Because of the preservation of normal cardiac function, these arrhythmias are generally well tolerated. These ventricular arrhythmias are usually associated with a favourable prognosis in normal individuals but they could have a clear impact on athletic performance and career. Occasional deaths reported in athletes with apparently idiopathic ventricular arrhythmias might not permit calling this arrhythmia benign.

Right ventricular outflow tract tachycardia

Right ventricular outflow tachycardia (RVOT) is characterised by a repetitive monomorphic tachycardia with a left bundle branch block and inferior axis and has to be distinguished from arrhythmogenic right ventricular dysplasia. The athlete with RVOT tachycardia is often female, aged between 20 to 35.²² The arrhythmia starts under conditions of emotional stress or exercise and in most cases is well tolerated. It can occur during deceleration of the sinus rate following exertion. The arrhythmia is cyclic AMP mediated triggered activity and abnormal automaticity rather than reentry mechanism. To evaluate this condition, ECG, signal-averaged ECG, Holter monitoring during sport activity, exercise test, echocardiography, coronary angiography, magnetic resonance imaging and EPS can be used. The therapy of choice is ablation. Long-term antiarrhythmic therapy is not acceptable in this young population because of either side effects or dissatisfaction with the need for long-term medication. There is variable response to treatment with β -blockers.

Recommendation

Athletes with nonsustained or sustained ventricular tachycardia should not compete in sports until they are fully evaluated and treated. Athletes without structural heart disease and PVCs that disappear during exercise may participate in all forms of competitive sports. Should the PVCs increase in frequency during exercise and deteriorate to VT with symptoms of

impaired consciousness, extreme fatigue or dyspnoea, then the athlete should only participate in low-intensity sports. Athletes with symptomatic PVCs that are suppressed by drug therapy should only participate in low-intensity sports. Athletes with asymptomatic monomorphic nonsustained ventricular tachycardia (heart rates <150 beats/min) and no structural heart disease may participate in all competitive sports if exercise testing demonstrates suppression of the ventricular tachycardia or no significant worsening compared with baseline. Cardiological examination every six months is needed.

Athletes with no structural heart disease and treated for sustained or nonsustained VT should not compete in all sports for at least six months after the last episode of VT. In some cases participation in low-intensity sports is permitted. If there is no clinical recurrence and the VT is not inducible by exercise and/or EPS and the athlete has no structural heart disease all competitive sports are permitted.

For athletes with structural heart disease and ventricular tachycardias, moderate and high-intensity competitive sports are contraindicated regardless of whether the ventricular tachycardia is suppressed or not. Only low-intensity sports are allowed.

All moderate and high-intensity sports are usually contraindicated in athletes with an implantable cardioverter defibrillator.⁹

Left ventricular outflow tract tachycardia (LVOT)

These tachycardias show an RBBB with an inferior axis on the ECG. Some of these arrhythmias originate in the epicardial regions of the left ventricle at a level of the left main coronary artery.²² The behaviour is similar to that of RVOT compatible with cyclic AMP mediated triggered activity and delayed afterdepolarisations. ECG, signal-averaged ECG, Holter monitoring during sport activity, exercise test, echocardiography, coronary angiography, magnetic resonance imaging and EPS and indicated to evaluate these arrhythmias.

Recommendation

See recommendations for right ventricular outflow tract tachycardia.

Idiopathic left ventricular tachycardia (ILVT)

Athletes showing idiopathic left ventricular tachycardia or fascicular tachycardia are between 20 and 40 years old. There is a variable association with physical activity and the arrhythmia is not usually provoked by exercise. Athletes have symptoms of palpitations, syncope is uncommon and cardiac arrest is rare. The ECG has an RBBB and left superior axis morphology and a relatively narrow QRS duration. ILVT can be provoked by programmed stimulation and sometimes by isoproterenol infusion. Verapamil could terminate this arrhythmia. Catheter ablation is now the therapy of choice. The prognosis is in general benign. To evaluate ILVT, ECG, signal-averaged ECG, Holter monitoring

during sport activity, exercise test, echocardiography, coronary angiography, magnetic resonance imaging and EPS can be used.

Recommendation

See recommendations for right ventricular outflow tract tachycardia.

Bundle branch reentry ventricular tachycardia

Prognosis of athletes with this form of tachycardia is related to the severity of left ventricular dysfunction and the underlying disease (for instance HCM). The electrocardiographic features are LBBB morphology, although an RBBB variant is also described. In most cases the His bundle is involved in the disease process and the HV interval in sinus rhythm exceeds 80 ms during EPS. In general RF ablation abolishes the VT. Evaluation can be carried out using ECG, signal-averaged ECG, Holter monitoring during sport activity, exercise test, echocardiography, coronary angiography, magnetic resonance imaging and EPS.

Recommendation

Athletes may participate in class IA sports but no competitive sports.¹⁴

Polymorphic VT

Usually the disorder emerges at an early age, but more adult onset has been described. Patients present with recurrent syncope triggered by exercise. Exercise may evoke polymorphic VT/VF in patients with catecholamine sensitive polymorphic VT/VF. It typically occurs with isolated atrial and ventricular ectopy at a certain (rate) threshold, followed by bi-directional doublets and more complex polymorphic VT or VF. QT interval is normal. A family history for SCD is common. EPS with isoproterenol infusion provokes this type of arrhythmia. In some cases it is a primarily

electrical autosomal dominant inherited disorder (LQTS, Brugada syndrome, etc) or there could be an underlying structural heart disease (cardiomyopathy or ARVD). Evaluation is with ECG, signal-averaged ECG, Holter monitoring during sport activity, exercise test, echocardiography, coronary angiography and EPS.

Recommendation

Athletes with polymorphic ventricular tachycardias, with structural heart disease in combination with PVCs, nonsustained and sustained VT, may only participate in low-intensity sports (class IA) whether the VT is suppressed or not. In athletes with ICD, all moderate and high-intensity sports are contraindicated. These athletes may participate in class IA sports.⁹

Summary

For diagnosis of structural heart disease, screening of all athletes who may be a risk factor for sudden cardiac death is not feasible and therefore not advisable. From a practical point of view the physician has to make a selection in order to pursue the best protection for the individual athlete. The physician should take into account the age-frequency distribution of causes of sudden cardiac death (figure 1). A good specific medical history addresses chest discomfort, dyspnoea, light-headedness, palpitations and early fatigue out of proportion to the level of exercise, which can be suggestive of underlying heart disease. A history of near syncope, and syncope during exercise or at rest in athletes warrants further evaluation. Syncope may be benign but can also be the first sign of a serious structural heart disease and could be a predictive sign of sudden cardiac death. A family history of sudden cardiac death is very useful in identifying athletes at risk. Physical examination can sometimes identify risk-bearing athletes or diseases, such as Marfan's

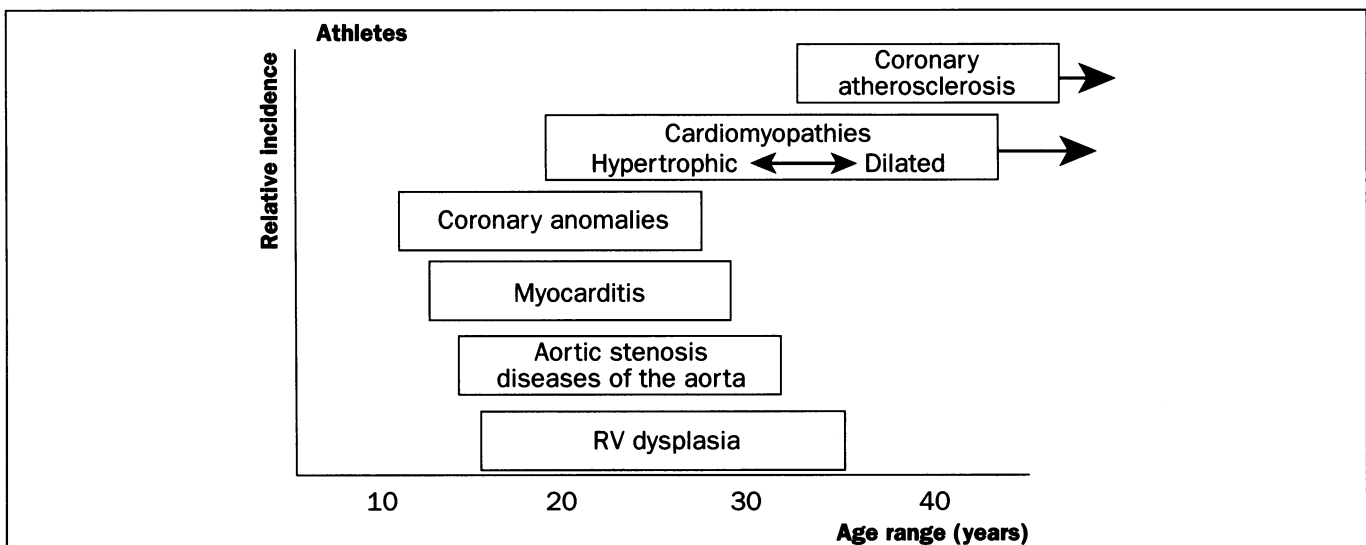


Figure 1. Age frequency distribution of various aetiologies of sudden cardiac death (adapted from reference 23).

Table 1. Cardiovascular screening questionnaire.

One positive answer justifies a specialised cardiovascular evaluation and advice.

- 1 Have you ever undergone surgery to the heart or great vessels?
- 2 Have you ever been diagnosed with a cardiac anomaly?
- 3 Have you ever been treated for a cardiovascular disease?
- 4 Are you taking any cardiovascular medication? (Antihypertensive drugs, anticoagulation, antiarrhythmic drugs)
- 5 Has any member of your family died suddenly before the age of 35?
- 6 Do you have a family history of hypertrophic cardiomyopathy?
- 7 Is there anyone in your family aged <35 with an ICD or pacemaker implant?
- 8 Do you have chest pain during exercise which rapidly disappears after cessation of exercise?
- 9 Have you ever had palpitations (see*)?
- 10 Have you ever had presyncope or a syncopal episode (see **)?
- 11 Have you ever been diagnosed with hypertension?
- 12 Is there a blood pressure difference of more than 15 mmHg between both arms?
- 13 Are the femoral pulses absent or asymmetric? Has anyone ever noticed a murmur in the groin?
- 15 Is a fourth heart sound present?
- 16 Is a diastolic heart murmur present?
- 17 Is a systolic heart murmur present that does not seem to be physiological?
- 18 Is a fixed splitting of the second heart sound present?
- 19 Is there a resting heart rate of less than 40 beats/min on clinical examination?
- 20 Did you find any stigmata of Marfan's disease on clinical examination?

(*) Palpitations: Inappropriate rapid heartbeat of more than 130 beats/min, regular or irregular, variable in duration, with or without associated symptoms (vertigo, chest pain, shortness of breath, syncope or presyncope.) Sudden onset? Regular or irregular? Frequency? Duration of this episode? How often are palpitations present? Precipitating factors? How did it stop?

(**) (Pre)syncope: Sudden decrease or loss of consciousness often preceded by acute vertigo or loss of vision.

syndrome (leptosome athletes), HCM or aortic stenosis and constitute a substantial risk for sudden death.

Proposals for recommendations and protocols

The following recommendations are made for cardiologists in order to support and guide the selection

of asymptomatic and symptomatic athletes in the assessment of eligibility for competitive sports. Pre-syncope or syncope and/or sudden cardiac death are more likely to occur in the presence of underlying heart disease. A syncope is a very important event and should be considered as an aborted sudden death until proven otherwise. The underlying mechanism

Table 2. Stress testing protocol for screening athletes with suspected arrhythmias.

Cyclist

Warming up for 3 minutes with a loading of 2 watt/kg.

After three minutes increase the loading every minute by 20 watt until exhaustion.

Three minutes rest.

Sprint protocol: increase loading in 2 minutes to 1000 watt; this sprint protocol should be done twice. Between the 2 sprints, 2 minutes rest.

Two-minute loading of 85 to 90% of the maximal power between the 2-minute periods, 1 minute with a loading of 100 watt.

The 2-minute loading can be repeated 2 or 3 times.

Running athlete

Warming up for 2 minutes at a speed 8 km/h.

After the 5-minute period increase the speed by 1 km/h every minute until exhaustion.

Three minutes rest.

Sprint protocol: Increase the speed in 2 minutes until maximum speed. Between the 2 sprints a 3-minute rest should be scheduled.

Two-minute loading of 85 to 90% of the maximal speed, between the loadings a 2-minute period with a speed of 8 km/h is scheduled. The 2-minute loading can be repeated 2 or 3 times.

Table 3. Indications for EPS.

All supraventricular reentry and focal tachycardias:

- WPW syndrome asymptomatic, at risk, or symptomatic
- Atrial flutter
- Idiopathic, right or left ventricular tachycardia
- Ventricular tachycardia in relation to structural heart disease, such as ARVD, DCM, etc

has to be elucidated. If there is a suspicion of arrhythmias, great pains should be taken in provoking and monitoring the cardiac rhythm. The cardiologist must be aware that arrhythmias can be benign but can also be the first sign of an underlying heart disease. The behaviour of the arrhythmia during sport activity should be assessed. Both bradyarrhythmia and tachyarrhythmia can be incompatible with athletic performance and could induce non-acceptable high risks for the athlete himself or others. Still worse, the initial innocent arrhythmia can provoke a dangerous new one.

To evaluate eligibility for (competitive) sport in symptomatic or asymptomatic athletes, the arrhythmological evaluation can be divided into three levels.

Level I includes:

- The cardiovascular screening questionnaire (table 1).²⁴
- A physical examination with special attention to stigmata of Marfan's syndrome. Is there a difference in blood pressure of more than 15 mmHg in both arms? On cardiac auscultation, is there a murmur in diastole or a nonphysiological systolic murmur? Any abnormal splitting of the second heart sound? Heart rate below 40 beats/min? Pulsations in both femoral arteries symmetric?
- ECG (rest and exercise).

Level II includes:

- ECG.
- Echocardiography M-mode, 2D and colour-flow Doppler.
- Holter monitoring should be performed during periods of intense physical activity and preferably whenever the athlete is performing her or his specific sport activity.
- 12-lead stress tests (sport specific) with a special protocol (table 2.) A normal stress test protocol for screening coronary artery disease in a young athletic population is not adequate.
- Routine blood test.

Level III includes:

- Signal-averaged ECG.
- Echocardiography M-mode, 2D, and colour-flow Doppler.
- Magnetic resonance imaging (MRI).
- Left and right catheterisation, left and right ventriculography and coronary angiography.
- Head-up tilt testing.
- EPS (table 3.)
- Nuclear stress test.

In the assessment of eligibility for competitive sport the cardiologist should take into account the age of the athlete (figure 1), level of athletic performance and impact of different sports on the cardiovascular system.

Protocol for asymptomatic athletes (all ages)

Cardiological evaluation level I is applied if there is a negative specific medical history, no abnormalities on physical examination, and no abnormal findings in the ECG and no history of sudden cardiac death no further evaluation is necessary and the athlete may participate in all competitive sports.

Level II evaluation should be done in case of abnormalities on the ECG to exclude HCM or other structural heart diseases.

Table 4. Cardiac and noncardiac causes of sudden cardiac death.¹⁷

HCM	Arteriovenous anomaly
CAD	WPW
ARVD	Myocardial bridging
CAA	Coronary aneurysm
LVH	Subvalvular aortic stenoses
Myocarditis	LQTS
Conduction disorder	Idiopathic VF
Mitral valve prolapse	DCM
Valve disease	Cerebral embolus
Aorta dissection	Pulmonary embolus

HCM=hypertrophic cardiomyopathy, CAD=coronary artery disease, ARVD=arrhythmic right ventricular cardiomyopathy, CAA=congenital coronary artery anomalies, LVH=left ventricular hypertrophy, WPW=Wolff-Parkinson-White syndrome, LQTS=long-QT syndrome, VF=ventricular fibrillation, DCM=dilated cardiopathy.

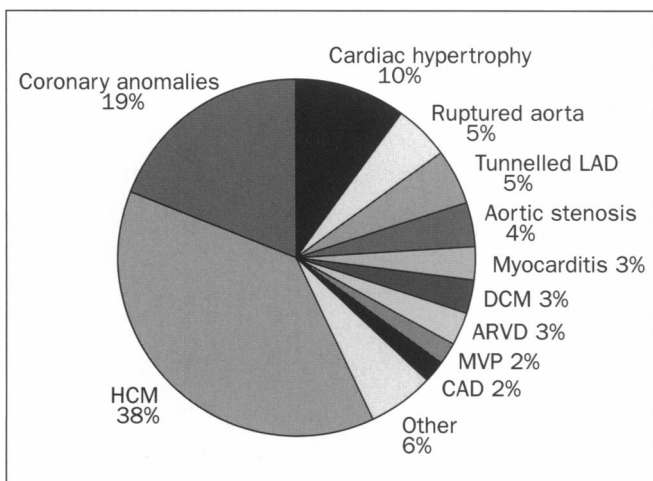


Figure 2. Estimated prevalence of sudden cardiac death in athletes younger than 35.

If there is a suspect family history of sudden cardiac death and/or structural heart disease, level II and in certain cases level III cardiological evaluation is indicated.

Protocol for symptomatic athletes age <35 year

Full cardiac evaluation is indicated with special attention to the most prominent cardiac disorders in relation to sudden cardiac death (figure 2 and table 4).

Protocol for symptomatic athletes age >35 year

Full cardiac evaluation is indicated with special attention to coronary artery disease. ■

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