Review Article

Participation in Sports and Leisure Activities of Young People with Known Cardiovascular Abnormalities

Hariton G. Antonakoudis, Leonidas E. Poulimenos, Georgios H. Antonakoudis

Department of Cardiology, Asclepeion Hospital, Athens, Greece

Key words:
Sudden cardiac death, sports, cardiovascular abnormalities.

Manuscript received:
January 11, 2008;
Accepted:
March 12, 2008.

Address:
Hariton G. Antonakoudis

14 Agiou Ioannou St., 163 41 Athens, Greece
e-mail:
antonakoud@yahoo.gr

Sudden death in young athletes comprises a real risk. Although it is very rare, it is a highly visible event, which consistently raises concern and ethical issues in both the lay public and the medical community.[1,2] Competitive sports activity enhances by 2.5-fold the risk of sudden death in adolescents and young adults. However it is necessary to make clear that sport activity is not *per se* a cause of increased mortality; it rather acts as a trigger of cardiac arrest in the presence of underlying cardiovascular diseases predisposing to life-threatening ventricular arrhythmias, potentially leading to sudden cardiac death (SCD). Sudden death in athletes is of cardiovascular origin in 90% of cases. Pre-participation screening aims to find these subjects and to exclude them from participation in order to reduce the risk of sudden death.[3]

On the other hand, exercise benefits are significant and well documented.[4] For example, it is believed today that coronary artery disease is almost entirely preventable and the contribution of exercise to such prevention and to life expectancy is significant. It is beyond the scope of this article to present the well documented exercise benefits. Nevertheless, it is useful to recall the most important of the so-called social benefits of exercise: improvement of perceived health; reduction of acute and chronic disease; enhanced quality of life; control of alcohol, tobacco and drug dependency; less passive smoking and fires; improved personal appearance; enhanced property values; enhanced employment; reduced employee turnover and absenteeism; reduced industrial injuries; reduced health insurance premiums; enhanced company image; reduced hospital and health care costs; fewer problems from social deviancy (vandalism, law enforcement, detention); and reduced geriatric dependency.[4]

Patients with known cardiovascular disease, even if they may really be at higher risk for SCD, cannot be completely excluded from any kind of sports participation, as long as a thorough evaluation is performed. The patients' right to exercise must be primarily kept in mind, but on the other hand any health risk (and SCD risk especially) should be ruled out. Obviously, a thorough pre-participation evaluation is needed.[5,6]

Classification of sports

According to the 36th Bethesda conference,[7] and to the recommendations of the International Olympic Committee,[8] an athlete is one who participates in an organised team or sport that requires regular competition against others as a central component, places a high premium on excellence and achievement, and requires some form of systematic and intense training—usually more than

10 hours per week. On the other hand, leisure activity is the physical activity that a person or a group chooses to undertake during their discretionary time.

An analytical classification of all kind of sports has been provided by the Committee on Sports Medicine and Fitness and is nowadays well accepted.[9,10] A proposed modified classification according to intensity is seen in Table 1, and in Table 2 according to contact risk.[5] The relative risk of exercise depends on the intensity of dynamic or static exercise needed for any sport activity.[12-15] A global classification of different sports according to the level of dynamic or static in-

Table 1. Classification of sports by intensity.

	High-to-moderate intensity		Low intensity
High-to-moderate dynamic and static demands	High-to-moderate dynamic and low-static demands	Low-dynamic and high-to-moderate static demands	Low-dynamic and low-static demands
Boxing	Badminton	Archery	Billiards
Crew/rowing	Baseball	Auto racing	Bowling
Cross-country skiing	Basketball	Diving	Cricket
Cycling	Field hockey	Equestrian	Curling
Downhill skiing	Lacrosse	Field events (jumping)	Golf
Fencing	Race walking	Field events (throwing)	Riflery
Football	Racquetball	Gymnastics	
Ice hockey	Soccer	Karate or judo	
Rugby	Squash	Motorcycling	
Running (sprint)	Swimming	Rodeo	
Speed skating	Table tennis	Sailing	
Water polo	Tennis	Ski jumping	
Wrestling	Volleyball	Water-skiing	
		Weightlifting	

Table 2. Classification of sports by contact.

Contact collision	Limited contact	Non-contact
Basketball	Baseball	Archery
Boxing	Bicycling	Badminton
Diving	Cheerleading	Bodybuilding
Field hockey	Canoeing/kayaking (white water)	Bowling
Football	Fencing	Canoeing/kayaking (flat water)
Flag	High jump	Crew/rowing
Tackle	Pole vault	Curling
Ice hockey	Floor hockey	Dancing
Lacrosse	Gymnastics	Discus
Martial arts	Handball	Javelin
Rodeo	Horseback riding	Shot put
Rugby	Racquetball	Golf
Ski jumping	Skating	Orienteering
Soccer	In-line	Power lifting
Team handball	Skiing	Race walking
Water polo	Cross-country	Riflery
Wrestling	Downhill	Rope jumping
	Water	Running
	Softball	Sailing
	Squash	Scuba diving
	Ultimate Frisbee	Strength training
	Volleyball	Swimming
	Windsurfing/surfing	Table tennis
	Weightlifting	Tennis
		Track

tensity needed, suggested by the 36th Bethesda conference,[7] is shown in Figure 1. Most SCDs occur in football players (35%), followed by basketball players (25%) and track and field athletes (15%).[5,7,10,11]

Sports eligibility according to underlying disease

The most common cardiovascular disease leading to SCD is hypertrophic cardiomyopathy (HCM) followed by congenital coronary artery anomalies, dilated cardiomyopathy, Marfan syndrome, right ventricular arrhythmogenic cardiomyopathy (RVAC), atherosclerotic coronary disease and rhythm/conduction diseases,[15] while some other not so uncommon and potentially dangerous cardiovascular diseases exist.[16-18] Furthermore, we should briefly mention the *commotio cordis*,[19] as the recognised cardiovascular risks on the athletic field are now extended to include cardiac arrest resulting from relatively modest, non-penetrating chest blows produced by projectiles (such as baseballs) or bodily contact, in the absence of underlying cardiac disease and without structural injury to the chest wall or heart. These uncommon, but usually fatal events seem to result when chest impact occurs precisely during the vulnerable phase of repolarisation and they may be reduced by use of softer projectiles.

Guidelines are in place governing eligibility and disqualification criteria for competitive athletes with cardiovascular diseases.[20-28] However, similar systematic recommendations for the much larger population of patients who are not trained athletes, but nevertheless wish to participate in any of a variety of recreational physical activities and sports, have not been available to date. The practicing clinician is frequently confronted with the dilemma of designing non-competitive exercise programmes for athletes with a disease after disqualification from competition, as well as for those patients with such conditions who do not aspire to organised sports. Physical fitness is becoming increasingly emphasised in our society. Patients with some heart disease are living longer, healthier lives and may also desire sports participation. Indeed, many asymptomatic or mildly symptomatic patients desire a physically active lifestyle, with participation in recreational and leisure-time activities, to take advantage of the many documented benefits of exercise. This is why recently both the American Heart Association and the European Society of Cardiology, based largely on the experience

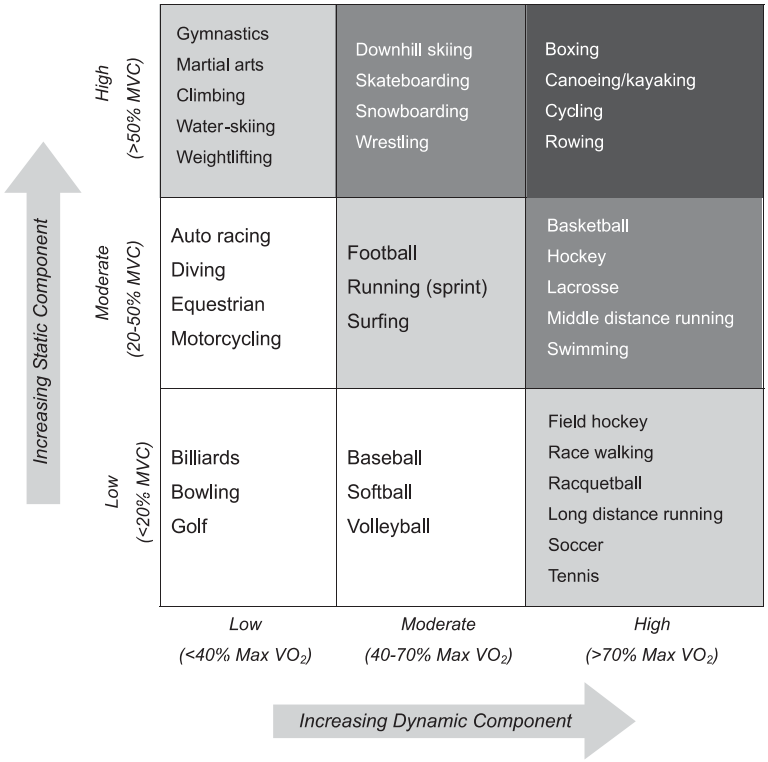

Figure 1. Classification of sports according to static and/or dynamic intensity.
MVC – maximal voluntary contraction.

and insights of the experts, gave some recommendations governing recreational exercise in cardiovascular diseases such as cardiomyopathies and channelopathies at risk of sudden death during sports and the potential of preventing fatal events.[29,30]

Genetic cardiovascular diseases

A group of relatively uncommon, but important genetic cardiovascular diseases are associated with increased risk for sudden death during exercise. They include HCM,[29,30] RVAC,[31-36] long-QT syndrome,[37] and Marfan syndrome.[38-44] These conditions, characterised by diverse phenotypic expression and genetic substrates, account for a substantial proportion of unexpected and usually arrhythmia-based fatal events during adolescence and young adulthood.

Although HCM has been repeatedly reported as a leading cause of athletic field deaths, these patients should not be excluded from participation in recreational activities.[5,16,20,29,30] However, specific prophylaxis is necessary and they must avoid some specific types of exercise, as shown in Table 3, while these precautions are mandatory in individuals with the high risk characteristics shown in Table 4.[7,29]

The same recommendations exist regarding the RVAC[31-35] and long-QT syndromes.[37] Special considerations apply to the not so uncommon Marfan syndrome.[38-44] In 1986, the sudden death of former Olympic volleyball player Flo Hyman from a ruptured aortic aneurysm highlighted the need for better detection of this syndrome in athletes. Its prevalence in the general population is estimated at 2 to 3 per 10,000, and it is a relatively common connective-tissue disorder that carries a significant risk of sudden death. Improved diagnostic awareness can substantially improve survival rates and reduce the risk.

After diagnosis is confirmed, active patients with Marfan syndrome should:[42]

- Participate only in sports with minimal physical demands, such as golf;
- Avoid sports with high static demands, such as weight lifting;
- Avoid sports that carry a risk of body collision, such as boxing, football or high diving;
- Wear protective cushioned spectacles when playing racket sports;
- Seek medical attention immediately in case of chest pain or fainting.

Table 3. Pre-participation preventive measures in patients with hypertrophic cardiomyopathy.

- Avoid "burst" exertion or sprinting
- Beware of environmental factors (temperature, humidity)
- Systemic exercise programmes are forbidden
- Avoid adrenergic-type activities
- Avoid intense static training (e.g. weightlifting.)
- Beware of risk of trauma
- Avoid extreme sports (bungee jumping, hang gliding ...)

Table 4. Patients with hypertrophic cardiomyopathy (HCM) who are at high risk.

- Patients with prior cardiac arrest
- Family history of sudden cardiac death due to HCM
- Unexplained syncope, especially after exercise
- Short episodes of ventricular tachycardia in ambulatory rhythm recordings
- Blood pressure drop during exercise stress test
- Extreme hypertrophy in echocardiogram (>30 mm)

Myocarditis – dilated cardiomyopathy

In these patients exercise avoidance is warranted. There is a great risk, and in some countries it is considered as the most common cause of SCD. Possible underlying myocarditis during the febrile phase of a viral illness is a reason to forbid any exercise in all patients with an acute illness. Acute myocarditis is a potentially devastating condition that is most commonly caused by viruses. Coxsackie B virus has been implicated in 50 percent of cases. Early symptoms, if present, may include exercise intolerance and congestive heart failure symptoms, with dyspnoea, cough and orthopnoea. Subtle clinical signs include tachycardia in the absence of fever, *pulsus alternans*, and other clinical signs of heart failure. (e.g. S_3 gallop, soft apical murmur, distended neck veins, peripheral oedema). Most patients with myocarditis present with sudden death secondary to a ventricular arrhythmia and have few, if any, prodromal signs or symptoms.[20,22,30,45]

Congenital and acquired coronary artery disease

Congenital coronary anomalies are multiple,[46-51] the most common being a misplaced aortic ostium, in which the left main and right coronary artery arise from the right sinus of Valsalva.[46] Myocardial bridges and inflammatory coronary artery aneurysms associated with Kawasaki's disease have also been reported as a cause of sudden death.[48-50] These conditions are difficult to

identify unless complaints of early fatigue, angina or exercise-induced syncope lead to a directed evaluation. In one review of 78 cases of sudden death thought to be secondary to autopsy-proven coronary anomalies, 62 percent occurred in asymptomatic persons.[46-51] Definite diagnosis is difficult. This abnormality must be suspected in cases of pain, arrhythmia or syncope during exercise and in these cases complete investigation is necessary before sports participation is permitted.

Tragically, acquired premature coronary artery disease can appear in athletes under the age of 30. Genetic predisposition plus other risk-factor prevalence can sometimes lead to coronary events resulting from typical atherosclerosis.[52] The prevalence was 2.6% in the Minneapolis Heart Foundation Institute survey,[2] while, paradoxically, in an Italian study a prevalence of 18.5% was detected.[23] Attention to risk factors and to the early symptoms of ischaemia, angina and other effort-related symptoms should be just as aggressively pursued in younger athletes as in older athletes.

Valvular heart disease

In athletes with mitral valve stenosis the extent of physical activity allowed is dependent on the size of the left atrium and concomitant valve regurgitation. They can participate in all types of sport associated with low and moderate isometric stress and moderate dynamic stress. Patients under anticoagulation should not participate in any type of contact sport.[53,54]

Asymptomatic athletes with mild aortic valve stenosis can participate in all types of athletic activities, as long as left ventricular function and size are preserved and there are no arrhythmias. Asymptomatic athletes with moderate aortic valve stenosis should only participate in sports with low dynamic and static stress, whereas exercise is not allowed in severe aortic stenosis and in aortic coarctation before definite surgical treatment. After operation exercise is allowed only if a gradient of less than <20 mmHg has been achieved.

Aortic valve regurgitation is often the result of connective tissue disease or a bicuspid valve. Athletes with mild aortic valve regurgitation, and with normal left ventricular end diastolic diameter and systolic function, can participate in all types of sport.[53,54]

Exercise increases the pressure gradient in patients with prosthetic aortic or mitral valves. The 26th Bethesda[22] conference recommended activity levels for athletes with prosthetic and bioprosthetic valves based on their anticoagulation status and left ventricular function. Patients who have a bioprosthetic mitral valve, who are not taking anticoagulants, and who have normal or near-normal left ventricular function, can participate in low-to-moderate static and low-to-moderate dynamic activities. Patients are also advised to avoid moderate static activities that carry a risk of bodily collision, such as auto racing, high diving, motorcycling or equestrian events. Patients who have an artificial aortic valve, are not taking anticoagulants and have normal function of both the valve and left ventricle, can participate in low-static and low-dynamic activities. Higher levels of activities may be permitted based on the athlete's abilities and haemodynamics. All patients taking anticoagulants should avoid sports that have a risk of collision.

Special consideration applies for the not so uncommon mitral valve prolapse.[55,56] Recently, a 3.7% prevalence of mitral valve prolapse was detected in competitive athletes in China.[56] In these patients even competitive sports are allowed. Exercise is forbidden only in patients with a personal or family history of syncopal attacks, documented severe arrhythmias, severe haemodynamic mitral valve regurgitation, or in patients with a history of embolic episodes. In some cases a continuous ECG recording should also be performed to detect significant arrhythmias.[54,57] However, a pre-participation consent form is suggested to avoid legal implications. Athletes with mild or moderate mitral valve prolapse do not present any significant differences in non-invasive cardiac electrophysiological indices, or in cardiopulmonary performance, compared to healthy ones. It seems that long-term exercise-induced adaptations can overcome any cardiac autonomic disorders in cases of mild-to-moderate mitral valve prolapse.[56]

Operated congenital heart disease

In operated aortic or pulmonary stenoses and operated septal defects exercise should be allowed. Patients suffering from Fallot tetralogy should be evaluated extensively (echo, stress test). In subjects with transposition of the great arteries exercise must be avoided because of the risk of arrhythmias.[26,27,29]

Brugada syndrome

This entity points to a role of the ECG in pre-participation screening. It also demonstrates that, in athletes with Brugada syndrome, repolarisation anomalies may be markedly attenuated during vigorous exercise and considerably increased immediately after exercise. The observed J-wave amplitude dynamics suggest enhance-

ment of pre-existing autonomic dysfunction through heavy exertion. Competitive athletics should be avoided and in cases of syncope an ICD should be implanted.[2,18]

Atrial fibrillation

Atrial arrhythmias are the most commonly encountered ones in professional athletes, particularly in basketball and football players. Ventricular arrhythmias, when not associated with cardiovascular abnormalities, do not convey adverse clinical significance. They appear to be an expression of athlete's heart syndrome and probably do not *per se* justify a disqualification from sports. The prevalence of atrial fibrillation in the general population is 0.04%, while in athletes it is 0.063%. The higher prevalence in athletes is due to exercise-induced atrial remodelling caused by volume or pressure overload and by angiotensin-1 receptor up-regulation. When there is satisfactory rate control sports participation is permitted. The treatment of choice is electrical cardioversion to sinus rhythm, as drugs may influence exercise performance. In the case of rare paroxysms of atrial fibrillation catheter ablation is probably not ideal. The physician has to be certain that there is no prolongation of the QT interval due to loss of electrolytes. As regards the selection of a good medication, amiodarone or propafenone are recommended.[60-63]

Wolff-Parkinson-White (WPW) syndrome

The risk of sudden death is 0.15-0.20% in asymptomatic and 2.2% in symptomatic patients with a WPW syndrome. In these individuals, intense physical activity or the practice of sports may not only trigger cardiac arrhythmias but also worsen their consequences and render them life-threatening. Moreover, severe episodes of pre-excited atrial fibrillation of WPW may even occur. Unfortunately, for one half of WPW patients who experience sudden cardiac arrest potentially leading to SCD, this is their first manifestation of the disease and usually occurs during exercise or emotional stress.

A complete electrophysiological study, including measurement of the anterograde refractory period of the accessory pathway, induction of atrial fibrillation and study of the effects of isoprenaline, seems to be indispensable to identify patients at greatest risk. When the risk of a potentially serious arrhythmia appears to be confirmed, catheter ablation of the accessory pathway may be the ideal solution. In general, radiofrequency ablation is necessary in symptomatic patients. In asymptomatic patients an electrophysiological study and probably ablation is suggested in high risk patients and in those requiring antiarrhythmic drug treatment, which may affect their performance. In low risk patients, exercise is permitted, especially if the pre-excitation disappears during a stress test.[64,65]

WPW syndrome represents the most important indication for radiofrequency ablation in athletes. Radiofrequency ablation is also recommended in other tachyarrhythmias, when they are disabling, related to exercise and therefore compromise an athlete's performance and sports career.[66] It is useful to explain to the athletes the possible recurrences with psychological traumatic consequences, the effective period to complete recovery, and the natural history of the tachyarrhythmias, which frequently disappear after interruption of the sports career.[67-69]

Patients with implantable cardioverter defibrillators (ICDs).

ICDs prevent recurrent arrhythmic death in sudden death survivors, or may prevent its development in physically active patients with an underlying risk for malignant ventricular arrhythmias.[70] Although ICDs do not substitute for the usual recommendations to refrain from intensive or competitive exercise because of any underlying arrhythmic disorder, they often provide a means for safe continuation of mild to moderate recreational sports activity. Long-term quality of life will be highly dependent on the prevention of inappropriate ICD shocks. This requires the choice of a durable lead device system, and careful programming tailored to the characteristics of the patient's physiological and pathological heart rhythms.[71-73] Fewer problems exist in patients with pacemakers.[74]

Syncope

Hippocrates (460-377 BC) stated: "It is common for subjects with frequent faints to die suddenly." Hippocrates' statement is well validated today given the fact that the incidence of SCD in individuals with exertional syncope can be as high as 17%. That is the reason for stopping exercise in these individuals for as much time as is needed to complete an extensive cardiological evaluation, and probably management. In young athletes, syncope in the past, before the initial pre-participation screening, has a neurally-mediated origin in most cases and shows a low recurrence rate. Exercise-related syncope is infrequent and is not associated with an adverse outcome in subjects without

cardiovascular abnormalities. The incidence of new syncope during competitive athletic activity is particularly low. On the other hand, post-exercise "syncope" is the mostly benign common faint due to causes reported as reflex syndrome in children (orthostatic hypotension, hyperthermia, "church" syndrome, hyperventilation, pain, etc.).[75,76]

Hypertension and sports

Children and adolescents who have systemic hypertension may be at risk for complications because exercise raises their blood pressure even higher. Athletes and other physically active patients should be screened for hypertension and given appropriate treatment.[77]

The American Academy of Paediatrics recommends:[78]

- The presence of significant hypertension in the absence of target organ damage or concomitant heart disease should not limit a person's eligibility even for competitive athletics. Athletes with significant hypertension should have their blood pressure measured regularly (every 2 months at the physicians office) to monitor the impact of exercise on blood pressure.
- Youths who have severe hypertension need to refrain from competitive sports and highly static (isometric) activities until their hypertension is under adequate control and they have no evidence of target organ damage. Complete prohibition of exercise may not be necessary for those patients.
- When hypertension and other cardiovascular diseases coexist, eligibility for participation in competitive athletics is usually based on the type and severity of other cardiovascular disease.
- The young athlete with hypertension, regardless of the degree of severity, should be strongly encouraged to adopt healthy lifestyle behaviours, including the avoidance of exogenous androgens, growth hormone, illicit drug abuse (especially cocaine), alcohol, use of tobacco and high sodium intake. The athlete should be advised that the use of diuretics and β-blockers has been prohibited by some athletic governing bodies. Diuretics must be avoided in high intensity athletes because of the risk of hypovolaemia or hypokalaemia. Drugs blocking the renin-angiotensin system along with calcium channel blockers are the drugs of choice.

Blood pressure measurements must be made at least once a year in all athletes, and in hypertensives it is suggested that they not take medication on a game day. Furthermore, we have to know that non-hypertensive athletes usually have a wall thickness of 12 or 13 mm, which seems to be more or less within the range of normal for these individuals.[78,79]

Athletic heart

The issue of the physician's responsibility is of high importance in the screening and management of both athletes and leisure-time sportsmen, since in the case of sudden death the liabilities of the physician and the medical community must be considered. Athlete's heart is an important clinical challenge, as it is difficult to differentiate from HCM or other cardiomyopathies. Many criteria to distinguish HCM from athlete's heart have been suggested. The unusual pattern of left ventricular hypertrophy, a left ventricular cavity <45 mm, marked left atrial enlargement, bizarre ECG patterns, abnormal left ventricular filling, female gender, family history of HCM, favour HCM; whereas a left ventricular cavity >55 mm, maximum VO_2 >45 ml/kg/min or >110% predicted, and especially the reduction of thickness with deconditioning for a 3-6 month period, favour athlete's heart.[80-82]

In so called athlete's syndrome the presence of arrhythmias is not infrequent. Bradyarrhythmias are more frequent in sportsmen than in the general population. This fact is often the result of a "relative vagal hypertony" due to a training effect. Tachyarrhythmias are present in sportsmen in almost the same percentage as in sedentary people.[83] Paroxysmal supraventricular reciprocating tachycardias may present various clinical consequences: unimportant symptoms or severe haemodynamic effects on athletic performance, particularly during intrinsically high risk sports activity. The athletes must be evaluated by a clinical protocol which includes ambulatory rhythm monitoring, ergometric test, echocardiography study, thyroid function tests and electrophysiological study. Sometimes, arrhythmias may disappear after interruption of athletic activity because of modifications of electrophysiological conditions.[83-86]

Regarding the appropriate follow up, the American College of Sports suggests an annual history and a reevaluation every 2 years, while the National Basketball Association suggests an echocardiogram every 2 years.[15] However, there is no clear-cut and world-wide acceptable method for screening and follow-up in sports. It is strongly indicated that the follow up of these individuals should be performed by a sports cardiologist.

The need for an emergency action plan

Preparedness for proper management of sudden cardiac arrest in the athletic venue is anyway critical, but it is more important when it involves individuals with known cardiovascular abnormalities. Implementation of an emergency action plan is needed. This should be specific to each athletic venue and should encompass emergency communication, emergency equipment, medical emergency transportation and venue directions with map. Access to early defibrillation is essential. A target goal of under 5 minutes from time of collapse to first shock is strongly recommended. An automated external defibrillator should be part of standard emergency planning for coverage of athletic activities. Through development and implementation of such a plan, healthcare providers help to ensure that the athlete will have the best care provided when an emergency situation due to a cardiovascular disease or *commotio cordis* does arise.[87-89] It is worth mentioning again that obtaining informed signed consent before initiating an exercise training program helps to clarify the responsibilities and goals of both the physician and the subject.[90-92]

Conclusions

Practicing cardiologists are frequently asked to advise on exercise programmes and sport participation in young individuals with cardiovascular diseases. Indeed, many asymptomatic or mildly symptomatic patients aspire to a physically active lifestyle in order to take advantage of the many documented benefits of exercise. The present review article is intended to offer practising cardiologists and sport physicians a comprehensive overview of the recommendations governing safe participation in different types of competitive sport, as well as the participation in a variety of recreational physical activities and amateur sports in individuals with known cardiovascular diseases. Obviously, careful evaluation is needed in these patients along with an effective emergency action plan. It is suggested that written consent be obtained before pre-participation is permitted.

References

1. O'Connor FG, Kugler JP, Oriscello RG. Sudden death in athletes: screening for the needle in a haystack. Am Fam Physician. 1998; 57: 1-12.
2. Maron BJ. Sudden death in young athletes. N Engl J Med. 2003; 349: 1064-1075.
3. Karvouni E, Anastasakis A, Spiliopoulou C, et al. Sudden death in the young in Greece. Hellenic J Cardiol. 2000; 41: 359-370.
4. Shephard RJ. Costs and benefits of an exercising vs a non exercising society. In: Exercise, fitness and health. Human Kinetic Publishers; 1990. p. 50-60.
5. Committee on Sports Medicine, Fitness. Medical conditions affecting sports participation. Pediatrics. 1994; 94: 757-760.
6. Maron BJ, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles. JAMA. 1996; 276: 199-204.
7. 36th Bethesda Conference Eligibility Recommendations for Competitive Athletes With Cardiovascular Abnormalities. J Am Coll Cardiol. 2005; 45: 1312-1375.
8. Bille K, Figueiras D, Schamasch P, et al. Sudden cardiac death in athletes: the Lausanne Recommendations. Eur J Cardiovasc Prev Rehabil. 2006; 13: 859-875.
9. Mitchell JH, Haskell WL, Raven PB. Classification of sports. J Am Coll Cardiol. 1994; 24: 864-866.
10. Mitchell JH, Haskell W, Snell P, et al. Task Force 8: classification of sports. J Am Coll Cardiol. 2005; 45; 1364-1367.
11. Lawless C, Lampert R, Olshansky B. Sudden cardiac death in athletes: rates of defibrillation. J Am Coll Cardiol. 2006; 47 (Suppl): 165A.
12. Corrado D, Basso C, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden cardiac death? J Cardiovasc Med. 2006; 7: 228-233.
13. Corrado D, Basso C, Rizzoli G, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden death in adolescents and young adults? J Am Coll Cardiol. 2003; 42: 1959-1963.
14. Maron BJ, Araujo CG, Thompson PD, et al. Recommendations for pre-participation screening and the assessment of cardiovascular disease in masters athletes: an advisory for healthcare professionals from the WG of the World Heart Federation, the International Federation of Sports Medicine, and the American Heart Association Committee on Exercise, Cardiac Rehabilitation, and Prevention. Circulation. 2001; 103: 327-334.
15. Bove AA, Block PC. Cardiac Problems in Athletes. 2007 American College of Cardiology [Internet]. Available from: http://www.medscape.com/viewarticle/552101
16. Maron B, Ackerman MJ, Nishimura RA, et al. Task Force 4: HCM and other cardiomyopathies, mitral valve prolapse, myocarditis, and Marfan syndrome. J Am Coll Cardiol. 2005; 45: 1340-1345.
17. Maron BJ, Zipes DP. Introduction: eligibility recommendations for competitive athletes with cardiovascular abnormalities - general considerations. J Am Coll Cardiol. 2005; 45: 1318-1321.
18. Esperer HD, Hoos O, Hottenrott K. Syncope due to Brugada syndrome in a young athlete. Br J Sports Med. 2007; 41: 180-181.
19. Maron BJ, Estes NA 3rd, Link MS. Task Force 11: Commotio cordis. J Am Coll Cardiol. 2005; 45; 1371-1373.
20. Kaminer SJ, Hixon RL, Strong WB. Evaluation and recommendations for participation in athletics for children with heart disease. Curr Opin Pediatr. 1995; 7: 595-600.
21. Corrado D, Basso C, Schiavon M, et al. Screening for hypertrophic cardiomyopathy in young athletes. N Engl J Med. 1998; 339: 364-369.
22. Maron BJ, Isner JM, McKenna WJ. 26th Bethesda conference: recommendations for determining eligibility for compe-

tition in athletes with cardiovascular abnormalities. Task Force 3: hypertrophic cardiomyopathy, myocarditis and other myo-pericardial diseases and mitral valve prolapse. Med Sci Sports Exerc. 1994; 26: S261-267.
23. Pelliccia A, Fagard R, Bjornstad HH, et al. Recommendations for competitive sports participation in athletes with cardiovascular disease. Eur Heart J. 2005; 26: 1422-1445.
24. Maron BJ. Hypertrophic cardiomyopathy. Circulation. 2002; 106: 2419-2425.
25. Maron BJ, Shen WK, Link MS, et al. Efficacy of implantable cardioverter-defibrillators for the prevention of sudden death in patients with hypertrophic cardiomyopathy. N Engl J Med. 2000; 87: 342-365.
26. Pelech AN, Neish SR. Sudden death in congenital heart disease. Pediatr Clin North Am. 2004; 51: 257-263.
27. Graham TP Jr, Bricker JT, James FW, et al. 26th Bethesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. Task Force 1: congenital heart disease J Am Coll Cardiol. 1994; 24: 867-873.
28. Cheitlin MD, Douglas PS, Parmley WW. 26th Bethesda conference: recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. Task Force 2: acquired valvular heart disease. J Am Coll Cardiol. 1994; 24: 874-880.
29. Maron BJ, Chaitman BR, Ackerman, et al. Recommendations for physical activity and recreational sports participation for young patients with genetic cardiovascular diseases. Circulation. 2004; 109: 2807-2816.
30. Pellicia A, Corrado D, Bjørnstad HH, et al. Recommendations for participation in competitive sport and leisure-time physical activity in individuals with cardiomyopathies, myocarditis and pericarditis. Eur J Cardiovasc Prev Rehabil. 2006; 13: 876-885.
31. Dalal D, Nasir K, Bomma C, et al. Arrhythmogenic right ventricular dysplasia: A United States experience. Circulation. 2005; 112: 3823-3832.
32. Thiene G, Nava A, Corrado D, Rossi L, Pennelli N. Right ventricular cardiomyopathy and sudden death in young people. N Engl J Med. 1988; 318: 129-133.
33. Fontaine G, Fontaliran F, Frank R, Tonet JL, Lascault G, Grosgogeat Y. Right ventricular dysplasia. An overlooked cause of tachyarrhythmias and sudden death. Cardiologia. 1990;35: 61-68.
34. Marcus FI, Fontaine GH, Guiraudon G, et al. Right ventricular dysplasia: a report of 24 cases. Circulation. 1982; 65: 384-398.
35. Corrado D, Basso C. Thiene G: Arrhythmogenic right ventricular cardiomyopathy: diagnosis, prognosis and treatment. Heart. 2000; 83: 588-595.
36. Katsouras GE, Margos PN, Livanis EG, Theodorakis GN, Kremastinos DT. Contribution of electroanatomical mapping to the diagnosis of arrhythmogenic right ventricular cardiomyopathy in a patient with sustained ventricular tachycardia. Hellenic J Cardiol. 2006; 47: 184-189.
37. Ackerman MJ. The long QT syndrome. Pediatr Rev. 1998; 19: 232-241.
38. Braverman AC. Exercise and the Marfan syndrome. Med Sci Sports Exerc. 1998; 30: S387-395.
39. Demak R. Marfan syndrome: a silent killer. Sports Illustrated. 1986; 64: 3-35.
40. Silverman DI, Burton KJ, Gray J, et al. Life expectancy in the Marfan syndrome. Am J Cardiol. 1995; 75: 157-160.
41. Hirata K, Triposkiadis F, Sparks E, et al. The Marfan syndrome: abnormal aortic elastic properties. J Am Coll Cardiol. 1991; 18: 57-63.
42. Salim MA, Alpert BS. Sports and Marfan Syndrome. The physician and sports medicine. 2001; 29: 1-9.
43. Schroeder LE, Lavallee ME. Ehlers-Danlos syndrome in athletes. Curr Sports Med Rep. 2006; 5: 327-334.
44. Siniawski A, Szyszka A, Gwizdala A, Plonska E, Cieslinski A. The risk of mitral pathology in marphanoid female basketball players. Pol Merkur Lekarski. 2006; 20: 299-301.
45. Bresler MJ. Acute pericarditis and myocarditis. Emerg Med. 1992; 24: 35-51.
46. Davis JA, Cecchin F, Jones TK, Portman MA. Major coronary artery anomalies in a pediatric population: incidence and clinical importance. J Am Coll Cardiol. 2001; 37: 593-597.
47. Iskandar EG, Thopmson PD. Exercise-related sudden death due to an unusual artery anomaly. Med Sci Sports Exerc. 2004; 36: 180-182.
48. Bestetti RB, Costa RS, Kazava DK, et al. Can isolated myocardial bridging of the left anterior descending coronary artery be associated with sudden death during exercise? Acta Cardiol. 1991; 46: 327.
49. Taylor AJ, Rogan KM, Virmani R. Sudden cardiac death associated with isolated congenital coronary artery anomalies. J Am Coll Cardiol. 1992; 20: 640-647.
50. Corrado D, Thiene G, Cocco P, et al. Non-atherosclerotic coronary artery disease and sudden death in the young. Br Heart J. 1992; 68: 601-607.
51. Basso C, Maron BJ, Corrado D, et al. Clinical profile of congenital coronary artery anomalies with origin from the wrong aortic sinus leading to sudden death in young competitive athletes. J Am Coll Cardiol. 2000; 35: 1493-1501.
52. Lefkou E, Fragakis N, Varlamis G. The inflammatory process during childhood and the risk of developing atheromatous disease in adult life: the role of C-reactive protein. Hellenic J Cardiol. 2006; 47: 184-189.
53. Burke AP, Farb A, Virnami R. Causes of sudden death in athletes. Cardiol Clin. 1992; 10: 303-317.
54. Mellwig KP, van Buuren F, Gohlke-Baerwolf C, Bjørnstad HH. Recommendations for the management of individuals with acquired valvular heart diseases who are involved in leisure-time physical activities or competitive sports. Eur J Cardiovasc Prev Rehabil. 2008; 15: 95-103.
55. Dollar AL, Roberts WC. Morphologic comparison of patients with mitral valve prolapse who died suddenly with patients who died from severe valvular dysfunction or other conditions. J Am Coll Cardiol. 1991; 17: 921-931.
56. Koutlianos NA, Kouidi EJ, Metaxas, et al. Non-invasive cardiac electrophysiological indices in soccer players with mitral valve prolapse. Eur J Cardiovasc Prev Rehabil. 2004; 11: 435-441.
57. Garson A Jr. Arrhythmias and sudden cardiac death in athletes. American College of Cardiology, 16th Bethesda Conference. Pediatr Med Chir. 1998; 20: 101-103.
58. Ma JZ, Dai J, Sun B, Ji P, Yang D, Zhang JN. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death in China. J Sci Med Sport. 2007; 10: 227-233.
59. American Academy of Pediatrics Committee on Sports Medicine and Fitness. Mitral valve prolapse, athletic participation in children and adolescents. Pediatrics. 1995; 95: 789-790.
60. Mont L, Sambola A, Brugada J, et al. Long-lasting sport practice and lone atrial fibrillation. Eur Heart J. 2002; 223: 477-482.

61. Biffi A, Piovano G, Verdile L, et al. Prognostic evaluation on supraventricular arrhythmias in athletes. In: Pelliccia A, Caselli G, Bellotti P, editors. Advances in sport cardiology. Berlin-New York-Milan: Springer; 1997. p. 40-44.
62. Furlanello F, Bertoldi A, Fernardo F, et al. Competitive athletes with arrhythmias. Classification, evaluation and treatment. In: Bayes de Luna A, Furlanello F, Maron BJ, Zipes DP, editors. Arrhythmias and sudden death in athletes. Dordrecht: Kluwer Academic; 2000. p. 89-105.
63. Milliez P, Haggui A, Maltret A, Leenhardt A. Recommendations for sports participation in patients with arrhythmia. Arch Mal Coeur Vaiss. 2006; 99: 992-1002.
64. Saxena A, Chang CJ, Wang S. Wolff-Parkinson-White syndrome in athletes. Curr Sports Med Rep. 2006; 5: 254-257.
65. Sarubbi B. The Wolff-White electrocardiogram pattern in athletes: how and when to evaluate the risk for dangerous arrhythmias. The opinion of the pediatric cardiologist. J Cardiovasc Med (Hagerstown). 2006; 7: 271-278.
66. Group on Sports Cardiology of the European Association for Cardiovascular Prevention, Rehabilitation. Recommendations for participation in leisure-time physical activity and competitive sports in patients with arrhythmias and potentially arrhythmogenic conditions. Part I: supraventricular arrhythmias and pacemakers. Eur J Cardiovasc Prev Rehabil. 2006; 13: 475-484.
67. Mezzani A, Giovannini T, Michelucci A, et al. Effects of training on the electrophysiologic properties of atrium and accessory pathway in athletes with Wolff-Parkinson-White syndrome. Cardiology. 1990; 77: 295-302.
68. Furlanello F, Bertoldi A, Bettini R, et al. Supraventricular reentry tachycardia and athletic fitness. Cardiologia. 1991; 36: 117-120.
69. Furlanello F, Bertoldi A, Inama G, et al. Catheter ablation in competitive athletes: indication. J Intern Cardiol. 1995; 8 (6 Suppl): 837-840.
70. Kallergis EM, Vardas PE. Primary prevention of sudden cardiac death: apart from the defibrillator, what is important in patients with myocardial infarction or heart failure? Hellenic J Cardiol. 2007; 48: 89-93.
71. Heidbuchel H, Corrado D, Biffi A, et al. Recommendations for participation in leisure-time physical activity and competitive sports of patients with arrhythmias and potentially arrhythmiogenic conditions. Part II: ventricular arrhythmias, channelopathies and implantable defibrillators. Eur J Cardiovasc Prev Rehabil. 2006; 13: 676-686.
72. Lampert RL, Cannom D, Olshansky B. Safety of sports participation in patients with implantable cardioverter defibrillators: a survey of Heart Rhythm Society members. J Cardiovasc Electrophysiol. 2006; 17: 11-15.
73. Choi GR, Porter CB, Ackerman MJ. Sudden cardiac death and channelopathies: a review of implantable defibrillator therapy. Pediatr Clin North Am. 2004; 51: 1289-1303.
74. Heidbüchel H, Panhuyzen-Goedkoop N, Corrado D, et al. Recommendations for participation in leisure-time physical activity and competitive sports in the patients with arrhythmias and potentially arrhythmogenic conditions Part I: Supraventricular arrhythmias and pacemakers. Eur J Cardiovasc Prev Rehabil. 2006; 13: 475-484.
75. Colivicchi F, Ammirati F, Santini M. Epidemiology and prognostic implications of syncope in young competing athletes. Eur Heart J. 2004; 25: 1749-1753.
76. Colivicchi F, Ammirati F, Biffi A, et al. Exercise-related syncope in young competitive athletes without evidence of structural heart disease. Clinical presentation and long-term outcome. Eur Heart J. 2002; 23: 1125-1130.
77. O'Connor FG, Meyering CD, Patel R, et al. Hypertension, athletes and the sports physician: implications of JNC VII, the Fourth Report and the 36th Bethesda Conference Guidelines. Curr Sports Med Rep. 2007; 6: 80-84.
78. Committee on Sports Medicine, Fitness. Athletic participation by children and adolescents who have systemic hypertension. Pediatrics. 1997; 99: 637-638.
79. Niedfeldt MW. Managing hypertension in athletes and physically active patients. Am Fam Phys. 2002; 52: 457-468.
80. Maron BJ. Distinguishing hypertrophic cardiomyopathy from athlete's heart: a clinical problem of increasing magnitude and significance. Heart. 2005; 91: 1380-1382.
81. Oakley D. The athlete's heart. Heart. 2001; 86: 722-726.
82. Pellicia A, Maron BJ, Di Paolo FM, et al. Prevalence and clinical significance of left arterial remodelling in competitive athletes. J Am Coll Cardiol. 2005; 46: 690-696.
83. Swanson DR. Atrial fibrillation in athletes: implicit literature-based connections suggest that overtraining and subsequent inflammation may be a contributory mechanism. Med Hypotheses. 2006; 66: 1085-1092.
84. McGovern BA, Liberthson R. Arrhythmias induced by exercise in athletes and others. S Afr Med J. 1996; 86: C78-82.
85. Link MS, Wang PJ, Estes NA 3rd. Ventricular arrhythmias in the athlete. Curr Opin Cardiol. 2001; 16: 30-39.
86. Huston TP, Puffer JC, Rodney WM. The athletic heart syndrome. N Engl J Med. 1985; 313: 24-32.
87. Courson R. Preventing sudden death on the athletic field: the emergency action plan. Curr Sports Med Rep. 2007; 6: 93-100.
88. Zangwill SD, Strasburger JF. Commotio cordis. Pediatr Clin North Am. 2005; 51: 347.
89. Maron BJ, Strasburger JF, Kugler JD, Bell BM, Brodkey FD, Poliac LC. Survival following blunt chest impact-induced cardiac arrest during sports activities in young athletes. Am J Cardiol. 1997; 79: 840-841.
90. Fletcher FG, Balady JG, Amsterdam AE, et al. AHA Scientific Statement, Exercise Standards for Testing and Training. Circulation. 2001; 104: 1694-1740.
91. Paterick TE, Paterick TJ, Fletcher GF, Maron BJ. Medical and legal issues in the cardiovascular evaluation of competitive athletes. JAMA. 2005; 294: 3011-3018.
92. Burke AP, Farb A, Virmani R, et al. Sports-related and non-sports-related sudden cardiac death in young adults. Am Heart J. 1991; 121: 568-575.