Pre-participation screening in patients with known heart disease[1]

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Summary

Adapted physical exercise is a well-established cornerstone of therapy in almost any cardiovascular disease, even severe chronic heart failure. On the other hand, physical exercise can be a trigger for sudden cardiac arrest (SCA) or even sudden cardiac death (SCD). Although the yearly incidence of exercise-related SCD in young athletes (age <35 years) is relatively rare (about 1–3 SCD in 100 000 young athletes per year), the incidence of fatal events undoubtedly increases in specific subgroups. Indeed, in older and untrained, previously sedentary individuals the incidence may be as high as nearly 7 deaths in 100 000 individuals per year. In cases of known underlying cardiac disease the risk of exercise-related SCD is fundamental. Currently, detailed European as well as North-American recommendations serve as reliable guidelines through most of the known cardiovascular diseases. In this review, the two expert panel consensus statements are highlighted by discussing the role of physiological adaptions during sports in general and by introducing some specific cardiovascular pathologies and their impact on competitive sports.

Key words: exercise-related sudden cardiac death; pre-competition screening; known cardiovascular disease

General considerations

Some call it "the paradox of sports": A term that prescribes the fact that regular physical training leads to indisputable and striking health benefits whereas sport is considered to be a clear trigger for sudden cardiac death (SCD), particularly in athletes with underlying heart disease. Nevertheless, recent studies provided data that regular intensive exercise may also be a promotor of structural cardiac changes which may thereafter lead to potentially harmful arrhythmias [1–6, 19–21]. But can this really be called "a paradox"? Apparently not, as the positive benefits outreach the potential harm of physical exercise by far [7]. Stressing history, it was 16th century philosopher "Paracelsus", who had stated that all things (even sports) are poison and only the dose permits something not to be poisonous. Contemporary opinion leaders, for example one well-known American Football coach established terms that refute the so-called paradox as such, "The will to win is important but the will to prepare is vital." Focussing on patients with underlying heart disease where screening is beneficial, thorough, with qualified risk stratification and guidance makes everyone capable of performing physical training safely and with great benefit.

Considering physical activity, one has to clearly differentiate between "competitive" and "non-competitive" or pure leisure-time sports. A fundamental risk for sports-associated SCD has clearly been detected in "competitive athletes" [1–4, 7]. However, both categories are not always easy to distinguish. According to a 2004 Scientific Statement of the American Heart Association, a competitive athlete is defined as one who participates in an organised team or individual sport that requires systematic training and regular competition against others and that places a high premium on athletic excellence and achievement [9]. This definition is quite vague and technically includes the majority of physically active individuals (particularly young individuals). Therefore, the differentiation between "competitive" and "non-competitive" athletes is only reasonable, if "non-competitive" sports would be equivalent to "leisure sports", whereby people perform physical exercise for pure pleasure and without any physical and/or psychological (di-)stress. Most likely, such individuals would not need a specific cardiac screening [4]. Nevertheless, pre-competition assessment and screening

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Table 1
Physiologic response to dynamic and static exercise.

	dynamic (= isotonic)	static (= isometric)
VO_2	▲▲	▲
CO	▲▲	▲
HR	▲▲	▲
SV	▲▲	–
SBP	▲	▲▲
DBP	▼	▲
P Res	▼▼	–

VO_2 = maximal oxygen uptake; CO = cardiac output; HR = heart rate; SV = stroke volume; SBP = systolic blood pressure; DBP = diastolic blood pressure; P Res = peripheral resistance.
▲ = increase; ▼ = decrease; – = no relevant changes.

should immediately be provided if such "leisure-sports-persons" suddenly and spontaneously perform in competitive situations (e.g., charity sport-events, marathons, etc.). Recently, these events constituted a certain "modern lifestyle" for many people. A recently published review especially focussed on the contemporary phenomenon of "occasional competitive athletes" [4].

With a yearly incidence of about 1–3 fatal events in 100 000 young athletes (age <35 years) [1–3], exercise-related SCD might be relatively rare. However, the incidence of SCD undoubtedly increases in specific subgroups. Indeed in older and untrained, previously sedentary individuals the incidence may be as high as nearly 7 deaths in 100 000 individuals per year [4, 7]. In case of known cardiac disease the risk of exercise-related SCD is fundamental [8–11]. Nevertheless, the latest large surveys capturing public attention (e.g., HF-ACTION trial, 12) state that "Adapted physical exercise is a well established cornerstone of therapy in almost any cardiovascular disease, even severe chronic heart failure" [12].

However, in the case of an underlying cardiovascular disease, recommendations for sports-participation have to be individually tailored. In general, risk has to be weighed against benefit and has to be discussed with the individual patient. Currently, detailed European as well as North American recommendations serve as reliable guidelines through most of the known cardiovascular diseases [9, 10].

Classification of sports and physiologic responses to exercise

Not only "competitive"or "non-competitive" characteristics of a certain physical activity determine the risk of fatal events. Depending on the underlying cardiac condition, it is the physiological response to exercise itself we primarily need to be aware of.

During physical exercise, the cardiovascular system undergoes various adaptive changes. As such, cardiac output can be increased by a gain of cardiac stroke volume until about 70% of the maximum physical capa-

Table 2
Classification of sports (adapted and modified from Mitchell et al. [13]).

	Low dynamic	Moderate dynamic	High dynamic
Low static	Golf	Fencing	Badminton
	Riflery	Table Tennis	Running (marathon)
	Curling	Volleyball	Cross-country skiing (classic)
			Squash[1]
Moderate static	Car Racing[1, 2]	Figure Skating[1]	Basketball/ Netball[1]
	Diving[2]	Running (sprint)	Biathlon
	Equestration[1, 2]		Ice-/Fieldhockey[1]
	Motorcycling[1, 2]		Football[1]
	Gymnastics[1]		Cross-country Skiing (skating)
	Sailing		Running (mid/long)
	Martial Arts[1]		Swimming[2]
			Tennis
			Handball[1]
High static	Bobsledding/Luge[1, 2]	Body Building[1]	Boxing[1]
	Rock Climbing[1, 2]	Downhill Skiing[1, 2]	Canoeing/Kayaking[2]
	Weight Lifting[1]	Wrestling (Swiss)[1]	Cycling[1, 2]
	Windsurfing[1, 2]	Snow Boarding[1, 2]	Decathlon
	Waterskiing[1, 2]		Rowing
			Speed Skating[1]
			Triathlon[1, 2]

[1] Danger of bodily collision; [2] Increased risk if syncope occurs

city has been reached. After this "cut-off", cardiac output will mainly be increased by a rise of heart rate. However, with increasing heart rate, mechanical diastole continously shortens and the ventricular filling becomes compromised. Therefore, a high stroke volume is the most important precondition to assure a high cardiac output and thus a high oxygen uptake (peak or maximal VO_2). Additionally, maximum heart rate physiologically decreases with age, although regular training can slow up this process (approximate estimation of individual maximum heart rate: 220 – age).

Depending on the type of exercise (e.g., "dynamic/isotonic" versus "static/isometric") characteristic hemodynamic changes can be determined (table 1). Although different types of sports rarely show purely dynamic or static qualities but a combination of both modalities – it pays to understand the underlying changes in order to tailor specific recommendations depending on the pathophysiology of the underlying heart disease.

In predominantly *dynamic exercise* there is a great demand for oxygen to supply the metabolic needs of the contracting muscle. This is typically provided by a distinct increase in cardiac output as a result of increased heart rate and stroke volume as well as a decrease of peripheral vascular resistance. The decrease of peripheral resistance leads to a decline of diastolic blood pressure with a relatively stable mean arterial pressure. *Static exercise*, on the other hand, causes a marked rise in mean arterial pressure by significantly increased systolic and diastolic blood pressure, with a relatively small increase of cardiac output (mainly provided by a rise of the heart rate).

Accordingly, different sports can be classified according to their predominant adaptive changes during specific exertion. Furthermore, one has to consider specific danger of bodily collision or special concerns in case of syncope during performance of a certain activity (table 2).

Specific recommendations on common heart disease

The 1985 Bethesda Conference (BC) of the American College of Cardiology (ACC) established consensus guidelines for eligibility/disqualification of competitive athletes with known cardiovascular diseases [9]. Lately, two more Bethesda Conferences (#26 and #36 in 1994 and 2005, respectively) have updated the former version [14, 15]. In 2005, the European Society of Cardiology (ESC) addressed its own recommendations in parallel [10]. Both panels always pronounced that these documents could not be viewed as obligatory guidelines but only as expert panel recommendations. Nevertheless, currently these two consensus recommendations are well established in clinical daily business.

In the following section, we highlight some selected cardiovascular pathologies to show their clinical consequences in the specific European [10] and North American guidelines (BC #36, 9). All statements refer on these two original papers and a comparison of the two panels [11]. As such, other specific references in literature are not mentioned in particular.

Although the guidelines generally are comparable, important discrepancies are outlined.

Coronary heart disease (CHD)

Apparently, no other cardiovascular disease demonstrates the ambiguity of physical exercise as classically as CHD. While regular physical activity reduces cardiovascular mortality and morbidity in primary and secondary prevention of the disease. CHD accounts for most exercise-related SCD, especially in individuals older than 35 years of age. Several mechanisms are responsible for these exercise-related events, such as sympathetic drive and release of catecholamines, platelet adhesion/activation (with risk of thrombotic complications), electrolyte disturbances and heart-related complications (such as sub-endocardial ischaemia and necrosis).

Athletes with known CHD should be systematically evaluated under the following criteria: *History* (to assess symptoms consistent with stable or unstable angina, presence of risk factors for CHD, as well as the type of sports in which the athlete participates, and family history of CHD/SCD), *resting ECG* and provocative testing: with *symptom-limited exercise testing* for evaluation of ischaemia-threshold, symptoms, ST segment changes, blood pressure and heart rate response, exercise capacity, and arrhythmias. *Echocardiography* (mainly to assess global LV function). *Coronary angiography* is mandatory in individuals with CHD willing to participate in competitive sports. *24 h Holter monitoring*, including a training session (to assess arrhythmias or silent ischaemic changes).

On the basis of the results of diagnostic testing the risk for fatal sports-associated events may be stratified:

A low probability for exercise-induced adverse cardiac events can be estimated if all of the following criteria are present:

- left ventricular ejection fraction of at least 50%
- normal exercise capacity according to age and gender (greater than 10 metabolic equivalents [METS], or greater than 35 ml O_2/kg/min if less than 50 years old; greater than 9 METS, or greater than 31 ml O_2/kg/min for 50 to 59 years old; greater than 8 METS, or greater than 28 ml O_2/kg/min, if 60 to 69 years old; and greater than 7 METS, or greater than 24 ml O_2/kg/min, if greater than or equal to 70 years old)
- absence of exercise-induced ischaemia on ECG/stress testing at lower steps

– absence of frequent, complex ventricular tachyarrhythmias at rest and during stress testing (including frequent premature ventricular contractions greater than 10% of beats/min), couplets, or ventricular tachycardia
– absence of significant coronary stenosis: i.e., <70% in major coronary arteries, or <50% of left main stem (ESC) respectively <50% in major vessels (BC #36).

A high probability for exercise-induced adverse cardiac events exists if one or more of the following criteria are present:
– left ventricular ejection fraction lower than 50%
– exercise-induced ischaemia (1 mm ST depression in two leads) on exercise testing at lower steps
– exercise-induced pathological dyspnoea (angina equivalent)
– syncope, or frequent, complex ventricular tachyarrhythmias at rest and/or during stress testing
– significant coronary stenosis of major coronary arteries (see above).

In both guidelines low/moderate dynamic and low static sports (ESC) respectively low dynamic and low/moderate static sports (BC #36) are recommended in case of a low propability of adverse events. However, intensely competitive situations should be avoided and at least an annual follow-up should be scheduled. In athletes with substantially increased risk a restriction to low-intensity sports is recommended.

After revascularisation (percutaneous coronary intervention or bypass surgery) patients are advised to wait at least four weeks until re-uptake of vigorous physical activity (after CABG, incisions should be completely healed) (BC #36) or after completing a supervised out-patient rehabilitation programme (ESC).

Furthermore, patients should be briefed about typical prodromal symptoms and the consequential behaviour when they occur (cease exercise, see a physician).

It should be noted that standard clinical exercise tests may not be appropriate for the evaluation of certain athletes with coronary heart disease as the classical graded exercise testing cannot replicate the cardiovascular stress produced by the sudden bursts of activity and the combination of high dynamic and static exercise.

Increasing amounts of coronary calcium burden should dictate a more cautious approach, particularly if the coronary calcium score is more than 100. It must be emphasised that even athletes identified as being at mildly increased risk and permitted to participate in low dynamic and static competitive sports cannot be assured that such participation will not increase the risk of cardiac events because any exercise transiently poses some increased exercise risk once CHD is established.

Congenital heart disease

As in most cardiovascular diseases, the benefit of sports on physical and mental health outclasses its potential harm. As such, only patients with congenital heart disease who are likely to deteriorate and are at high risk of life threatening arrhythmias should be restricted from sports participation. Nevertheless, literature regarding exercise in athletes with congenital heart disease is limited. Therefore, a restrictive attitude seems wise, in case of doubt. Obviously, dynamic exercise seems to be more suitable than static exercise.

Regular follow-up exams to identify those at risk are inevitable and should be scheduled every 6–12 months. Especially after complex surgery/scarring, patients with congenital heart disease are particularly prone to relevant arrhythmias (possible warning sign: widening of QRS duration >160 ms) and pulmonary hypertension during sports. Therefore, regular follow-up should stringently include assessment of arrhythmias and pulmonary hypertension/elevated pulmonary resistance during exercise.

Hypertrophic cardiomyopathy (HCM)

HCM is a relatively common genetic heart disease (prevalence 1:500 of the general population) and the most common cause of sports-associated SCD in young athletes. However, although the majority (75–95%) of HCM patients show abnormal ECG patterns the disease is characterised by heterogeneous presentation and natural history. Extrapolation of risk level from non-athletes to highly trained competitive athletes is tenuous. This relates to the unstable electrophysiologic substrate and propensity for potentially lethal ventricular tachyarrhythmias in HCM, interacting with the physiologic stresses inherent in athletic training and competition. Nevertheless, there is no reliable predictor to stratify the risk of suffering from a potentially lethal ventricular tachycardia (VT), therefore, guidelines recommened a demure attitude concerning sports participation excluding all athletes with definite diagnosis of HCM from competitive sports. As such, athletes with a probable or unequivocal clinical diagnosis of HCM should be excluded from most competitive sports, with the possible exception of those of low intensity (class IA sports).

Two important remarks need to be noted at this point: Firstly, although the clinical significance and natural history of genotype positive, but phenotype negative individuals remains unclear, at present, no compelling data are available with which to preclude these athletes from competitive sports, particularly in the absence of cardiac symptoms or a family history of sudden death. However, clinical follow-up examinations on a 12- to 18-month basis, in addition to serial two-dimensional echocardiography: 12-lead ECG, ambulatory Holter electrocardiogram (ECG), eventually cardiac magnetic resonance (CMR) imaging and exer-

cise stress testing should be provided. If all of these parameters continue to be normal, then eligibility to competitive sports can still be approved.

Secondly, the role of role of implantable cardioverterdefibrillators (ICDs) has to be highlighted: Although effective for sudden death prevention in observational studies, the unique physiologic milieu associated with competitive athletic activities (including intravascular volume and electrolyte disturbances, neurohormonal activity, and the potential for myocardial ischaemia) makes the absolute reliability of ICDs in such settings unpredictable. Furthermore, there is a possibility for device malfunction due to the risk of traumatic damage.

Thus, it needs to be clearly stated that the placement of an ICD in an HCM patient does not change the competitive sports recommendations (e.g., restriction from contact-sports and generally from most of competitive sports).

Acquired or congenital valve disease

In patients with acquired or congenital valve disease the specific pathophysiology of a valve dysfunction has to be considered. Generally, a follow-up of at least once a year is advisable depending on the severity of the valve disease – e.g., in moderate and severe valve disease.

In aortic regurgitation, the decrease of peripheral resistance and shortening of diastolic filling (as in dynamic/endurance exercise) leads to a decrease of regurgitant volume. On the other hand, particularly static exercise can predispose to an exacerbation of regurgitation due to the lessoned effect of decreased peripheral resistance during exercise. Only low static and/or low dynamic sports should be considered for athletes with relevant aortic regurgitation.

In patients with mitral regurgitation on the other hand, the increase in heart rate and blood pressure leads to an increase of regurgitant volume.

Athletes with aortic stenosis should undergo regular exercise testing with special regard to their blood pressure response during physical activity. Blunted blood pressure response has prognostic impact and can lead to symptomatic dizziness or even fainting/syncope.

In patients with prosthetic heart valves, the hemodynamic patterns during exercise can be abnormal despite unsuspicious measures at rest. As such, repetitive exercise testing is recommended. Generally, a sports restriction to low/moderate dynamic and static exercise is recommended.

Patients on anticoagulants should restrain from contact sports or sports with increased risk of bodily collision.

Infective heart disease

The incidence of myocarditis seems underestimated for it is a challenging diagnosis with a debatable prognosis [16]. The most common are in terms of infective aetiology (viral), cardiotoxic agents such as cocaine or certain medications can also cause an inflammation in cardiac tissue. Apart from impaired LV-function the inflammation may create an electrically unstable substrate promoting the development of potentially fatal arrhythmias.

In pericarditis (infectious or non-infectious), the inflammation affects the pericardium, however, discrimination from myocarditis is often inaccurate as the subepicardial layers an the myocardium itself may also be involved. This crossover has an impact on the current guidelines:

Generally, no physical training should be performed in any case of acute infectious disease and fever. Once diagnosis of myocarditis is established a re-uptake of all sportive activity can be considered after 6 months of sports restriction, at the earliest, but only if the patient is asymptomatic and normal findings referring LV dimension/-function, blood exams, 12-lead ECG can be achieved and no significant arrhythmia occurs. In case of (isolated) pericarditis the guidelines recommend a reuptake after at least 6 months (ESC) or as soon as no residual pericardial effusion or other pathologic findings (athlete is asymptomatic, normal findings referring LV dimension/-function, blood exams, 12-lead ECG, no significant arrhythmia) can be monitored (BC #36).

Systemic hypertension

Due to the increase of mean arterial pressure in static sports, dynamic exercise is much more suitable for patients with hypertension, even if blood pressure is well controlled at rest.

Obviously, an accurate assessment of systemic hypertension is crucial (24 h ambulatory blood pressure [BP] measurement). All guidelines outline the importance of an *additional risk stratification.* Beside clinical baseline assessment (medical history, including the assessment of additional cardiovascular risk factors, family history, physical examination, ambulatory-/home BP-measurements, ECG, laboratory exams like serum glucose, serum lipids, etc.), accurate estimation of *target organ damage* (echocardiography, abdominal ultrasound, urine testing, etc.) should be performed. Last but not least, *associated clinical conditions* (stroke, heart failure, peripheral arterial disease, etc.) need to be considered.

Once risk assessment has been made and blood pressure is adequately controlled, specific recommendations can be made. As mentioned before, in general, static exercise is potentially harmful. Palatini et al. demonstrated exorbitant increases of intravascular blood pressure during static resistance training (>300/200 mm Hg) [17]. The European recommendations, referring on the severity of hypertension and the additional risk assessment, are more distinct than the Bethesda guidelines. In summary, all sports can be performed if a low added risk was estimated. No high static

or high dynamic sports should be recommended when the patient sustains a moderate added risk and all static sports should be avoided in case of a high added risk.

Last but not least and despite all adverse effects – regular training is known to lower blood pressure in the medium term. As such, regular exercise as a part of classical "life-style changes" is recommended in individuals at low and moderate risk before starting drug treatment.

Diuretics and beta-blockers are not recommended in competitive athletes with hypertension because they may impair exercise performance or cause electrolyte and fluid disturbances (although beta-blockers would have a high efficacy on exercise hypertension). Furthermore, in competitive sports, they may appear on the doping list (e.g., diuretics in sports with weight categories). Calcium channel blockers and ACE-inhibitors can be considered first-line drugs in athletes.

Although, no specific guidelines are established concerning treatment of explicit exercise hypertension, drug therapy should be considered in regularly physically active individuals.

Follow-up exams should be scheduled every 6–12 months.

Arrhythmias

Cardiac arrhythmias may occur without evidence of a morphological substrate. Mental and physical stress during exercise can produce relevant electrophysiological and hemodynamic changes due to physiological adaption during exercise (heart rate, av-conduction, etc.). Furthermore, electrolyte deviation during sports may trigger arrhythmias.

Usually, the Bethesda guidelines (#36) permit greater autonomy in interpretation and individualised guidelines compared with the ESC guidelines. As such, electrophysiologic study and radiofrequency ablation are often mandatory procedures for the ESC (but not in the Bethesda guidelines) in the assessment and management of athletes with arrhythmias. Generally, a close follow-up is recommended (at least yearly) by monitoring possible symptoms, LV-function, heart rate, 12-lead ECG and further rhythmological testing.

Some specific arryhythmias shall be mentioned in particular in this section, as they demonstrate significant differences between the two expert panels:

Arhythmogenic right ventricular cardiomyopathy (ARVC): ARVC, a genetic disorder with myocardial atrophy and subsequent replacement of connective and fatty tissue predominantly in the right ventricle is cited as a common cause of SCD in young athletes. Diagnosis is challenging and based on different criteria (family history, VT, typical ECG-changes and suggestive pattern in echocardiographic/MR-imaging). Athletes with probable or definite diagnosis should be excluded from all competitive sports, an exception for low intensity sports can be discussed (BC #36).

Ion channelopathies: The recommendations include long-QT syndrome (LQTS), Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT). The short-QT syndrome, an entity which is getting increasing attention currently, is not mentioned in either document.

Clinical diagnosis of LQTS can be challenging as QT interval corrected for heart rate (QTc) might be borderline or even within normal limits in a large proportion of genetically proven LQTS patients [18]. Conversely, occurrence of QTc interval above the widely used upper limits of 0.44 s in male subjects or 0.46 s in female subjects is not an uncommon finding in young trained athletes. Threshold values for long QTc are lower in the European compared to the American guidelines (0.44 s versus 0.47 s in males and 0.46 s versus 0.48 s in females). When QTc interval exceeds these limits genetic testing is recommended (particularly in the European guidelines) to increase the likelihood of definitive diagnosis. Once diagnosis is confirmed, the European guidelines exclude athletes from all competitive sports while the Bethesda guidelines allow low intensity sports (particularly in LQT 3). Although the risk of sudden death is probably not zero in genotype-positive, phenotype-negative individuals, the Bethesda document states that, on the basis of the available scientific evidence, it is not justifiable to generally preclude such individuals from competitive sports. The European expert panel, however, discourages asymptomatic genotype-positive, phenotype-negative individuals with proven mutation and normal QTc interval on 12-lead ECG from participation in all competitive sports. Nevertheless, most information indicates that serious arrhythmias are uncommon in individuals with QTc interval <500 ms. A unique recommendation applies to such individuals with LQT1 mutation, who should refrain particularly from competitive swimming, because of the strong association between this sport and cardiac events.

Brugada syndrome and CPVT

Both documents restrict athletes with Brugada syndrome from participation. A clear association between exercise and sudden death in the Brugada syndrome has not been established. However, data is limited and the potential impact of hyperthermia (as it may happen during sports) on fatal arrhythmias in patients with Brugada syndrome lead to restrictions in sport: Disqualification from all competitive sports is recommended by the ESC document, with a potential exception in BC #36 only for low-dynamic and low-static sport. Differences between the BC #36 and ESC documents are raised with regard to gene carriers of Brugada syndrome and CPVT. According to BC #36, gene carriers without the phenotype (in the absence of symptoms and ventricular tachyarrhythmia inducible at electrophysiologic study) should not be precluded from participation in competitive sports. In contrast, the ESC document states that

all gene carriers (without the phenotype) should be restricted from competitive sport.

WPW syndrome

Although the North American guidelines do not recommend electrophysiologic study as mandatory in asymptomatic athletes (with the exception of asymptomatic athletes if engaged in moderate or high-level competitive sports), testing is required in symptomatic patients. The ESC guidelines, in contrast, consider complete risk assessment (including electrophysiologic study) in all patients (whether they show symptoms or not). Both guidelines state that athletes with increased risk on the basis of electrophysiologic study and those who are symptomatic with atrial flutter/fibrillation or syncope should undergo radiofrequency ablation of the accessory pathway to retain athletic eligibility. Guidelines devide regarding the "post-interventional" period after which competitive sports can be resumed (4 weeks (BC #36) versus 3 months in Europe).

Devices

Patients with pace-makers (PM) can participate in sports according to the underlying disease but should be restricted from sports with danger of bodily collision (as marked in table 2) because of possible damage of the device or electrodes. An important remark has to be made regarding patients with implanted ICDs: Current guidelines consistently recommend restriction from competitive sport activities in athletes with ICDs, with the possible exception of some low-intensity sports without associated risk of trauma to the device. The cut-off heart rate for the device needs to be set according to the physiological sinus tachycardia during exercise to prevent inadequate shocks.

It has to be stated that this paper is not supposed to be a complete overview. It only deals with a selection of cardiovascular pathologies and their relation to exercise-related sudden cardiac death to demonstrate the two landmark expert panel recommendations in this field. We recommend to consult the detailed recommendations for further information.

Conclusion

Regular physical training is not only recommendable but mandatory in individuals with known cardiovascular disease. Although, they cannot be seen as ubiquitous and obligatory recommendations, the European [10] and North American [9] consensus panel recommendations serve as important and well-established guidelines in this field.

Although generally similar, the two landmark consensus documents (ESC and Bethesda Conference #36) harbour several important differences related to the risk assessment of sudden cardiac death during competitive sports and the criteria applied for disqualification of athletes with cardiovascular disease. Until a unique consensus could be found these discrepancies have to be respected.

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