

Guidelines Cardiological guidelines for competitive sports eligibility

Consensus Document by

Italian Society of Sports Cardiology (SIC Sport), Italian Association of Out-of-Hospital Cardiologists (ANCE), Italian Association of Hospital Cardiologists (ANMCO), Italian Federation of Sports Medicine (FMSI)

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Preface	662	Sports activity in patients with an implantable cardioverter-defibrillator	676
PRACTICAL AND MEDICAL-LEGAL ASPECTS		References	676
Medical-legal aspects	662	CONGENITAL AND ACQUIRED VALVULAR HEART DISEASES	
Accreditation in Medicine and Sports Cardiology	664	Congenital heart diseases	682
SPORTS CLASSIFICATION BASED ON CARDIOVASCULAR INVOLVEMENT		Preface	682
Introduction	665	Patent ductus arteriosus	683
Classification	665	Atrial septal defects	683
ARRHYTHMIAS AND POTENTIALLY ARRHYTHMOGENIC CONDITIONS		Anomalous pulmonary venous return	684
Preface	667	Atrioventricular septal defects.....	684
Symptoms of possible arrhythmic origin	668	Ventricular septal defects.....	684
Syncope	668	Aortic coarctation.....	685
Palpitations	669	Aortic stenosis	685
Arrhythmias.....	670	Pulmonary stenosis.....	686
Bradycardias and conduction defects	670	Tetralogy of Fallot	686
Premature beats	671	Transposition of the great arteries	687
Supraventricular tachycardias	671	Acquired valvular heart diseases and mitral valve prolapse	687
Ventricular tachycardias	673	Mitral stenosis	687
Potentially arrhythmogenic conditions ...	674	Mitral regurgitation	687
Mitral valve prolapse.....	674	Aortic stenosis	688
The Brugada syndrome	674	Aortic regurgitation	688
Arrhythmogenic right ventricular cardiomyopathy	675	Valve prosthesis.....	688
Long QT syndrome	675	Mitral valve prolapse.....	688
Short PR interval (Lown-Ganong-Levine syndrome).....	675	References	689
Indications to catheter ablation in athletes	675	CARDIOMYOPATHIES, MYOCARDITIS AND PERICARDITIS	
Return to sports activity after transcatheter ablation.....	675	Cardiomyopathies.....	691
Sports activity in subjects with a pacemaker.....	675	Hypertrophic cardiomyopathy.....	691
		Diagnosis.....	691
		Risk assessment.....	692
		Indications	692

Dilated cardiomyopathy	692	Blood pressure measurement.....	696
Diagnosis	692	Etiology and target organ damage	697
Risk assessment	693	Sports eligibility	698
Indications	693	Antihypertensive treatment	
Arrhythmogenic right ventricular		and sports activity.....	699
cardiomyopathy	693	Physical activity for the prevention	
Diagnosis	693	and treatment of hypertension	699
Indications	694	References	699
Other cardiomyopathies	694	CORONARY ARTERY DISEASE.....	
Myocarditis.....	694	Introduction	700
Diagnosis	694	Sports eligibility in subjects with	
Indications	694	known coronary artery disease	700
Pericarditis.....	694	Sports eligibility in subjects with silent	
Diagnosis	695	coronary artery disease.....	700
Indications	695	Coronary artery disease and exercise	
References	695	in a gym setting	701
SYSTEMIC HYPERTENSION.....		References	701
Definition and classification.....	696	Appendix	702

Preface

Working out guidelines and protocols for healthcare professionals has always characterized the fruitful activity of any Scientific Society. It entails a long-lasting planning phase mainly devoted to choose the task force members which is not an easy task due to the coexistence of indolence, excessive tolerance or irritability among experts as well as to a sound antagonism among the different schools of thinking.

The cardiological protocols for sports eligibility, nowadays better known as “COCIS Protocols” (from the Italian Organizing Cardiological Committee on Sports Eligibility), are not an exception to this rule and this new edition, which follows the two previous ones published in 1989 and 1995, has had to cope with many difficulties. However, at the end of the working process, all those who participated in the protocol drafting felt as if they had made “something good” by preparing useful guidelines for daily clinical practice of their colleagues in cardiology and sports medicine. These guidelines present with novelty and new approaches as a result of the dramatic advances in scientific knowledge and are based on the extraordinary scientific and cultural experience of the last 20 years of the Italian Society of Sports Cardiology (SIC Sport) that promoted this initiative. The management of this initiative was rendered easier by the thorough interaction of the COCIS Committee members, the majority of whom, besides being members of the SIC Sport and the Italian Federation of Sports Medicine, were joining from other Scientific Societies such as the Italian Association of Out-of-Hospital Cardiologists, the Italian Association of Hospital Cardiologists, and the Italian Society of Cardiology, and were by statute compelled to participate in protocol drafting.

This new edition includes some basic and essential new information while still being concise and simple, characteristics that made it a “pocket” tool to which

easily refer to (that is the reason why references were omitted and are published only in the scientific journals). On one hand, the first part devoted to the medical-legal aspects was simplified, taking their knowledge for granted, and on the other a short paragraph was added dealing with accreditation of Centers and sports physicians in order to bring this issue to the attention of colleagues and institutions. The Committee members and the consulted experts who were outstanding representatives of the major Italian cardiological and sports medicine associations, deemed unanimously that an improvement in healthcare quality (health defense of athletes and people of any age practicing sport) represents a key step toward the definite establishment of Medicine and Sports Cardiology in our country.

PRACTICAL AND MEDICAL-LEGAL ASPECTS

Medical-legal aspects

Sports medicine practitioners are high-trained professionals engaged in prevention, treatment and rehabilitation programs aimed to provide care for individuals who engage in sports and thus they are legally responsible for their delivery of care, either in case of abuse or neglect, and are subjected to the duties and liabilities pertaining to all healthcare professionals of any specialty field.

With reference to competence (the duty of good practice), sports medicine practitioners and consultant cardiologists should apply the cultural and technical knowledge achieved by attending post-graduate schools, maintaining professional certification(s) with the today’s compulsory continuing medical education (CME).

With reference to prudence (the duty of forecasting dangerous or harmful situations related to their activity) and diligence (the duty of providing prompt care),

sports medicine practitioners and consultant cardiology are required to comply with such a behavior rigorously.

In order to ascertain sports eligibility, sports physicians must conform to several compulsory rules whose violation represents specific negligence. The observance of these regulations (established by authoritative laws, decrees, government and regional writs, and rules of Sports Federations) is therefore mandatory, in particular nowadays due to the continuing difference in the adopted rules among Regions and the spreading of the so-called “health federalism”.

Besides complying with the above-mentioned rules, sports physicians have the legal duty of putting into practice any required additional behavior and to resort to any diagnostic tool in order to make the correct diagnosis or to rule out the presence of any harmful condition to the athlete, being sure that the subject does not run unjustified and excessive risks. In case sports eligibility is uncertain and the available diagnostic options do not bring about a definite conclusion, the sports physician has the duty of rejecting sports eligibility.

When submitting an athlete to screening evaluations or diagnostic-therapeutic measures, sports physicians should provide competent medical service with respect for human dignity and confidentiality and their activity should be aimed primarily to safeguard the individual’s welfare and safety. The willingness of the athlete to practice sport as well as that of others (coaches, managers, sponsors, etc.) must not prevail upon the need of guaranteeing the subject’s welfare and in no way should it interfere with the definite judgment of the physician.

In uncertain cases, when standard non-invasive investigations do not allow for a definite diagnosis, some invasive procedures may be performed in accordance with the reference parameters used in clinical practice aiming to assess the risk/benefit ratio. This evaluation should always be carried out case by case: in this respect, it should be stressed that additional diagnostic examinations are performed not only to assess sports eligibility but also for diagnostic purposes in order to adopt preventive and therapeutic measures targeted at the individual’s health defense as well as at the defense of the right to work in case of professional athletes.

Nowadays more than in the past, before submitting any subject to risky diagnostic exams, it is mandatory to obtain a legally effective consensus, as it is required for any healthcare provision (Medical Deontological Code 1995, title III, chapter IV, articles 29 and 31).

A legally effective consensus means that it must be both well-informed and voluntary and is obtained after the subject has received unbiased and thorough medical information on the purpose and possible consequences of the proposed investigation or treatment. As for minors, control of health information is given to their parents who jointly exert their parental authority over the minor.

In practice, it is necessary a written record of the patient’s consent which certifies that he/she has understood the details and implications of what is proposed.

Written informed consent must unequivocally express the athlete’s will after he/she has received satisfactory answers to all his/her questions. Moreover:

- for the consent to be legally effective, subjects should have been provided with appropriate information;
- in case of refusal to consent on behalf of a mentally competent subject, no diagnostic option should be undertaken since medical treatment is not allowed in these circumstances;
- obtaining written informed consent does not protect doctors from legal liability in case of malpractice.

One example of informed consent may be as follows:

“I undersigned give my informed consent to the proposed treatment. I declare I have received full answers to any questions I had regarding the purpose and possible consequences of the proposed investigation”.

Date
(signature)”

With reference to cardiological sports eligibility, it should be recalled that it represents just one aspect, though essential, of the final eligibility judgment to be formulated by the sports medicine specialist. Such a judgment should rely upon the results of the investigations established by law and of any other additional exam he has the right to ask for. From a cardiological viewpoint, eligibility for competitive sports means that the physician gives his consent to:

- engage in a given sport (specific training) after accurate basic psychophysical training (general training);
- participate in athletic activities in accordance with the principles and rules set by sports regulations.

Among human activities, sports practice entails an acceptable and calculated risk. The most frequent risks consist in trauma injuries and cardiovascular accidents whose prevention can mainly be achieved by keeping under control the “human” and “technical” factors inherent to the practiced sport. The influence of the “human” factor may be restricted by an accurate selection of subjects who might engage in specific sports activities, and by serial controls of their fitness levels and training programs. As for the “technical” factor, it would be useful to know rules and regulations of each type of sport, assess activity areas, monitor environmental conditions, and maintain equipment.

Finally, the concern about medical-legal responsibility and/or the wrong or inadequate understanding of these matters may lead doctors to formulate a negative judgment disappointing the athlete’s expectations. Such an attitude, especially when not based on circumstantial evidence, may cause negative consequences either psychological or, in case of professional athletes, economic and career-related, so as to induce them to seek compensation for the damage suffered.

Another issue which needs to be addressed is confidentiality that has nowadays gained great importance due to the fact that sports physicians are requested to provide medical service in a context where mass-media and information "at any cost" are prevailing.

Frequently, due to economic interests, the intense pressure from the coaches, management, insurance companies, sponsors and even the motivated athletes themselves may render a decision of permanent or temporary non-eligibility a difficult task. This also occurs when the information released, even unintentionally, may damage the person concerned, the sports environment or the whole community.

Accreditation in Medicine and Sports Cardiology

The accreditation of all institutions of the National Health System was directed by decree-law 502/92 concerning the reform of this discipline within this field. Since then a number of legislative acts have succeeded aiming to put accreditation into practice, to define its requirements and institutionalize its procedure. Later on, Regions were empowered to define the accreditation systems for healthcare organizations as directed by the 1997 DPR which determines basic requirements and general criteria to which refer to for further development maintaining autonomous decision-making as well.

The accreditation procedure of healthcare organizations is ongoing in almost any local and regional area with the adoption of different application programs chosen from those receiving legislative approval. A further demonstration from the legislator of the willingness to implement this procedure quickly derives from the National Health Program 1998-2000 which clearly states the need for "a selection process of accreditation providers based on criteria of healthcare quality" that means to choose Centers and healthcare providers capable of supplying the best performance at the lowest cost.

In this context it is likely, if not predictable, that Institutions, Centers, Out-Patient Departments and single Sports Medicine healthcare organizations will be shortly involved in the accreditation process, according to different requirements among Regions. In this respect, taking for granted that institutions have complied with established standards (mainly concerning structure and safety), two main issues should be addressed:

- A. the identification of responses meeting standard requirements at the regional and national level in Sports Medicine;
- B. the development of additional criteria pertaining this medical specialty.

As for the first issue, the following prerequisites are essential:

1. personnel management: organization charts should be available and individual responsibilities defined;

2. work planning: the activity of each Center should be scheduled through recorded plans, the provided services should be listed, and the procedure for defining product standards should be started (e.g., type and level of an echocardiographic exam or of a maximal exercise test);

3. Centers should be equipped with an effective communication system with their users and management modalities of waiting lists as well as the priority criteria adopted should be made available to the public;

4. inventory of the biomedical equipment and periodic verification and maintenance plans should be applied with particular emphasis to emergency equipment (such as defibrillators and emergency medications);

5. personnel training and education activities should be planned and recorded. In the initial phase, for example, basic life support practical courses for medical care providers are recommended;

6. data management (record forms, eligibility, non-eligibility, appeals, etc.) should be standardized according to unequivocal criteria;

7. the application of definite procedures and "protocols" should be encouraged in case of "critical" activities and/or for those activities whose performance may show interindividual variability;

8. if requested, healthcare providers should undergo verification tests about users' satisfaction promoted by the proper healthcare institutions. It is thus reasonable that a "clinical audit" process be instituted by the Regional Appeal Committee;

9. each Center should identify a contact person for any accreditation process.

Obviously, specific additional criteria exist for Sports Medicine and they mainly deal with the definition of the different complexity levels of healthcare providers. Usually, it is possible to distinguish:

- a *first level*, which characterizes those institutions (out-of-hospital departments, offices of single specialists) granting sports eligibility or general assistance to people practicing sport;

- a *second level*, which characterizes those institutions carrying out consultant activity about complex cases and planning of training/rehabilitation programs;

- a *third level*, which characterizes those Centers with specific equipment and competence for the management, besides complex cases, of sports eligibility and assistance to disabled athletes, diabetics, master athletes, planning of rehabilitation programs, etc.

On the basis of the complexity level, the following should be defined accordingly:

- minimal personnel;

- minimal equipment: for example, Centers performing maximal exercise tests should be equipped with a defibrillator and second-level Centers should be able to perform echocardiography on-site;

- supply system and additional healthcare service provisioning. In other words, out-site healthcare providers and healthcare professionals should be clearly indicat-

ed, such as those performing biohumoral investigations, cardiological, neurological and ophthalmological evaluations, as well as their timing to fulfill requests and their degree of involvement in Sports Medicine;

- interconnections with healthcare providers of different levels and shared protocols;
- intervention plans for clinical emergency (mandatory for out-of- and near-hospital Centers);
- the so-called “clinical competence”, based on minimal levels of activity needed to guarantee maintenance of the acquired competence on behalf of healthcare providers. This activity should also be recorded in order to be made available for yearly certification.

If the aforementioned has to be considered as absolutely necessary in Sports Medicine, it has to be considered as even more mandatory in Sports Cardiology, a medical specialty that plays a pivotal role in providing care to subjects engaging in sports and that requires healthcare providers to be highly trained and undergo continuous updated professional education.

SPORTS CLASSIFICATION BASED ON CARDIOVASCULAR INVOLVEMENT

Introduction

Nowadays, an updated and exhaustive sports classification has become a fundamental tool in the daily practice of sports physicians and consultant cardiologists. Health professionals must know which organs are mostly involved in different sports, the physiologic and technical aspects related to each sport, as well as the proper - real or expected - “cardiovascular risk”. The need to classify sports activities for scientific and practical purposes has always been counteracted by the difficulty in identifying criteria which have to be clear enough as to providing a handy, simple and at the same time analytical classification, close to the physiologic adaptations to each sport.

From the sole physiologic viewpoint, sports activities have been formerly classified according to various criteria, based on one or more biological variables. In Italy, the best-known classification is that created by Antonio Dal Monte, based, on one side, on the energy source for muscle work and, on the other side, on the biomechanical features of each specific sport. Such a classification is still widely applied by sports physicians, exercise physiologists and trainers, though it has undergone a necessary update, in the effort to provide a better classification in the fields that have shown great athletic and technical improvement, and to include new disciplines.

Nevertheless, sports classification based on cardiovascular effort has met with similar difficulties. Such an approach, moreover, is of paramount importance when assessing eligibility for competitive sports (pre-participation screening), particularly in subjects with

cardiovascular disease or abnormalities, not requiring, *per se*, withdrawal from exercise and sports.

Cardiac involvement, indeed, can be constant over time, as it occurs in aerobic and endurance sports (marathon, cross-country skiing, cycling, etc.), or alternating, as it occurs in team games (alternate aerobic-anaerobic activities), without a true difference between the two in terms of cardiovascular risk. Intense and brief efforts, starting or finishing abruptly, can be a stronger arrhythmogenic trigger than those efforts, equally maximal, which start progressively and are interrupted gradually. Similarly, abrupt interruption after intense efforts may result in hemodynamic and rhythm disturbances more than the effort itself. Moreover, sports which entail a moderate increase in heart rate but a marked blood pressure (BP) elevation, may be deleterious in subjects with hypertension and aortic disease. In sports mainly characterized by a “neurogenic” involvement, cardiac work may be mild from the hemodynamic viewpoint but, on the other hand, it brings about a strong neuroendocrine stimulation, mainly sympathetic, although it may represent a real cardiac risk only in few cases.

When assessing cardiovascular risk in sports activities, the so-called intrinsic risk should never be underestimated, i.e. the risk related to the specific environment in which sports take place (scuba diving, climbing, motor sports, etc.). In this case, regardless of the underlying mechanisms and causes, the hypothetic occurrence of syncope or pre-syncope (see Arrhythmias section) may turn to be far more dangerous to the athlete and spectators (e.g. motor sports). Following such considerations, it is reasonable to suppose that cardiovascular risk might increase in sports with body impact when blows to the chest or strong reflex cardiac stimulation (head trauma, intense pain, etc.) can be expected, potentially resulting in arrhythmias, mostly bradyarrhythmias.

Finally, it can be useful to stress once again that the interaction between exercise performance and the cardiovascular system cannot always be solved in terms of simple and pathophysiologic mechanisms. Although it is possible to classify cardiovascular involvement and the inherent risk concerning each sport in grossly homogeneous categories, the overall risk depends, to some extent, on internal and external factors, such as health conditions, mental approach, environmental conditions, and so on.

Classification

In the 1995 edition of the COCIS Protocols, a new classification of sports based on cardiovascular involvement was introduced, which took into account the changes in few parameters easy to detect and monitor, such as heart rate and cardiac output, BP, peripheral resistance and degree of sympathetic activation due to emotional involvement.

A. Non-competitive sports with low to moderate cardiovascular involvement, characterized by constant hemodynamic requirements, submaximal heart rate and decreased peripheral resistance.

Hunting	Swimming
Touristic kayak	Skating
Touristic cycling	Footing or walking
Golf	Cross country skiing
Jogging	Trekking (soft)

B. Sports with “neurogenic” cardiovascular involvement, characterized by an increase in heart rate rather than in cardiac output, and caused by a strong emotional stress, especially in competitive sports.

B1. Moderate to high heart rate increase.

Motor racing
 Aviation sports
 Riding, polo
 Riding, dressing
 Motorcycle sports (speed)
 Motor boating
 Parachute
 Sport fishing, scuba diving
 Diving
 Sailing
 Modern pentathlon, only shooting and riding

B2. Minimum to moderate heart rate increase.

Bowls, bowling e curling
 Golf
 Sport fishing
 Shooting (fixed or moving target), bowshot and volley, etc.

C. Sports with “pressure” cardiovascular involvement, characterized by submaximal cardiac output, high to maximal heart rate and moderate to marked increase in peripheral resistances.

Climbing
 Sport climbing
 Track and field, sprint, throwing, jumps, decathlon (throwing and jumps), eptathlon (throwing and jumps)
 Bob, sled e skeleton
 Body building
 Cycling, speed and keirin
 Motorcycling, motocross
 Swimming, 50 m
 Swimming with flippers, 50 e 100 m, 4 × 50 m and 4 × 100 m relay
 Synchronized swimming
 Water skiing
 Skiing, slalom, down-hill, skiing alpinism, flying km, freestyle, snowboard, jumps, Nordic combined (only jump)
 Weight lifting
 Table tennis
 Windsurf

D. Sports with moderate to high cardiovascular involvement, characterized by a continuous and rapid increase, up to maximum, in heart rate and cardiac output.

Badminton
 Baseball
 Bowls, volley
 Soccer (futsal)
 River kayak, polo kayak
 Cricket
 Football
 Gymnastics and kallisthenics
 Ice hockey, field hockey, lawn hockey
 Underwater scuba hockey
 Judo, karate, taekwondo
 Wrestling
 Basketball, handball
 Waterpolo
 Volleyball and beach volley
 Ice and roller skating, figure and other types
 Modern pentathlon, only fencing
 Boxing
 Rugby
 Underwater scuba rugby
 Fencing
 Softball
 Squash
 Tamburello
 Tennis

E. Sports with high cardiovascular involvement, characterized by high to maximal heart rate and cardiac output.

Track and field, 400 m, 400 m hurdles, 800 m, 1500 m, 3000 m steeple-chase, 5000 m, 10 000 m, marathon, decathlon ed eptathlon (only running), 20 km e 50 km walk, half marathon, cross country running, ultra marathon
 Biathlon, skiing-shooting
 Olympic kayak
 Rowing
 Cycling, individual and team pursuit, point race, madison, 1 km time trial, individual time trial, mountain bike (cross country and downhill), cyclo-cross, BMX, spinning
 Nordic combined, only cross country skiing
 Swimming, 100 m, 200 m, 400 m, 800 m, 1500 m, long distance
 Swimming with flippers, 200 m, 400 m, 800 m and 1500 m
 Roller skating, speed, all distances
 Ice skating, speed (long and short track)
 Pentathlon, running and swimming
 Down-hill skiing, giant slalom, super G
 Cross country skiing, all distances
 Triathlon

Overall, the COCIS Committee considers this approach as being still valid. Moreover, it has become familiar to sports physicians. Nevertheless, some changes were made in order to fit new and old sports into the proper categories. It can be useful to remind, however, that such a classification has only the purpose of providing an indication, and it is dictated by practical needs, that are helping sports physicians and consultant cardiologists in evaluating cardiovascular risk reliably.

ARRHYTHMIAS AND POTENTIALLY ARRHYTHMOGENIC CONDITIONS

Preface

Cardiac arrhythmias in athletes may derive from either a physiologic phenomenon (i.e. sinus bradycardia) or a cardiac disease. In the latter case, arrhythmias may originate from different conditions such as genetic abnormalities of the ion channels (e.g. Brugada syndrome), congenital anomalies of the conduction system (e.g. Kent bundle), structural heart diseases, either congenital (e.g. atrial septal defects) or acquired (e.g. cardiomyopathies).

Arrhythmias may lead to no hemodynamic impairment or, conversely, they may result in varying degrees of pump failure, including heart failure and circulatory arrest. Furthermore, from a clinical viewpoint, they can be either silent or symptomatic, become a periodic and systematic alteration easily detectable by means of short ECG recording or continuous monitoring; in some cases, they can be paroxysmal or terminate spontaneously. In the latter case, they are hardly detectable at ECG recording.

Arrhythmia severity can range over a wide scale, from a benign form to various degrees of cardiac risk. When evaluating athletes with arrhythmias – either documented or suspected – sports physicians should first make a correct clinical examination, particularly to rule out any cardiac disease, as this is the most important prognostic factor¹⁻⁴. The clinical examination should include:

- a thorough family history, searching for any cases of sudden cardiac death (especially in youth and adulthood) and/or arrhythmogenic conditions shown to have a genetic and hereditary background;
- a close physiologic personal history, searching for any hints of substance abuse, i.e. smoking, alcohol, medications, drugs, doping. Risk factors for coronary artery disease should also be investigated (especially in Master athletes);
- a past clinical history, mainly concerning cardiovascular diseases;
- a recent clinical history, with the purpose of identifying symptoms such as palpitations, pre-syncope or syncope, weakness, chest pain, dyspnea, and unexplained worsening in physical performance. Sportsmen are likely to underestimate their symptoms, even when they are of some importance, hence the need for a careful questioning.

Athletes with arrhythmias should undergo the following three-level evaluation:

- *the first level* includes those exams which in Italy are mandatory to assess sports eligibility: medical history, clinical evaluation, standard ECG at rest and after physical exercise consisting of a 3-min step test;
- *the second level* includes additional tests, usually non-invasive, such as echocardiography (ECHO), maximal

exercise stress testing (EST) and 24-hour Holter monitoring (HM). The latter should cover an awake-sleep cycle and a session of appropriate exercise or specific training, provided that there are no clinical conditions or arrhythmias to put the patient's health at risk. According to clinical sense, laboratory tests may be recommended, including blood tests, thyroid hormones, serum electrolytes, rheumatic and immune tests for infective diseases;

- *the third level* includes non-invasive and invasive tests, carefully tailored to the documented or suspected arrhythmia, i.e. head-up tilt testing, pharmacological stressors (atropine, isoproterenol, flecainide, etc.), the search for the presence of ventricular late potentials by signal averaging, heart rate variability, T-wave alternans, transesophageal electrophysiologic study at rest or on exertion (TEPS), endocavitary electrophysiologic study (EPS). Some exams are still experimental, and their diagnostic power is still to be defined (such as T-wave alternans)⁵ and their results, therefore, are to be considered with caution. Previously mentioned tests can be performed along with any other examination required to rule out a structural cardiomyopathy.

To date, sports physicians have to correctly diagnose any arrhythmia and its expected outcome, and to define the proper treatment, including transcatheter ablation; moreover, they are called to assess sports eligibility for subjects who have already undergone transcatheter ablation or pacemaker or intracardiac device implantation. The purpose of this chapter, therefore, is to state criteria to determine sports eligibility in the following conditions:

- arrhythmias, either suspected or documented;
- conditions favoring arrhythmias;
- patients already submitted to transcatheter ablation or pacemaker or intracardiac device implantation.

The primary goal of this evaluation is eligibility to competitive sports based on risk assessment. That:

- arrhythmias may cause hemodynamic impairment, due to extremely fast or slow heart rates, either at rest or, most important, during or after exercise;
- arrhythmias may cause pre-syncope, syncope and/or cardiac arrest, and even exercise-related sudden cardiac death;
- sports activity may influence the pathologic and electrophysiologic substrate of arrhythmias, by worsening the clinical picture and/or the natural history of specific conditions.

Before stating the eligibility criteria, it should be pointed out that sometimes they may vary when taking into account:

- whether the athlete is just beginning practicing sport or has been practicing sports over the years;
- the athlete's age: requests for sports eligibility are made by children as well as older subjects;
- specific sports, the different cardiovascular involvement and the entity of "intrinsic risk" of each sport (i.e. driving vehicles, scuba diving, climbing, etc.). When

certifying sports eligibility one should recall that the workload of training sessions may be even higher than that of competitions.

Finally, it should be pointed out that, in some arrhythmias, data are scanty to enable to properly define their potential risk. Before looking in detail at each arrhythmia and syndrome of arrhythmic origin, it is to be underlined that experts did not always reach a general consensus on the suggested criteria. In some cases, the consensus was general (class I, according to current classification), while sometimes it met with conflicting evidence (class II, high prevalence of positive opinions, IIa, low prevalence of positive opinions, IIb). More rarely, a decision was suspended because of the lack of consensus or contrary general consensus (class III). Obviously, this was mainly due to the different degrees of scientific evidence found in the literature regarding specific issues, classified as level A (evidence based on incontestable data from large randomized clinical trials or meta-analysis), level B (evidence based on data from a limited number of trials), level C (no scientific evidence, but only the experts' consensus). In this document, consensus classes and levels of evidence are specified only for specific issues.

Symptoms of possible arrhythmic origin

Athletes with symptoms of suspected arrhythmic origin, such as syncope, palpitations at rest or on exertion, require a thorough evaluation by a cardiologist. Sports eligibility will be suspended until the cause has been discovered, especially in sports with intrinsic risk.

Syncope. Definition. Syncope is characterized by sudden and transient loss of consciousness and postural tone, with spontaneous and complete recovery within a short time interval. It is caused by an abrupt reduction of global brain blood flow⁶.

Pathophysiology. Syncope may be caused by different mechanisms. Main types are: neurocardiogenic syncope (vasovagal, sinus-carotid, environmental or situational), orthostatic syncope, those of primary cardiac origin (either arrhythmic or structural) and cerebrovascular syncope. In most athletes syncope is neurocardiogenic with a good prognosis⁷⁻²¹. However, it can be the hallmark of a misdiagnosed cardiac condition, even with a poor prognosis, or represent a potential marker of the risk of sudden cardiac death^{17,19}. Regardless of its origin, syncope may bring along the risk of injury, particularly in those athletes participating in sports with a high intrinsic risk.

Diagnosis. In the initial evaluation of the athlete with a history of syncope, sports physicians should pursue three main goals²⁰:

- differentiate syncope from other cardiac conditions

- likely to cause loss of consciousness (epilepsy, transient ischemic attack, drop attack, hypoglycemia, etc.);
- search for any clinical evidence suggesting the proper diagnosis;

- search for any underlying heart disease.

The initial evaluation must include the family and clinical history, BP measurement in ortho- and clinostatism, and standard ECG. The clinical history should assess thoroughly the presence of any cardiac disease, congenital or acquired, that may cause syncope. Recent clinical history is aimed at obtaining a clear description of the presumed or true syncope.

A non-syncopal event is suggested by confusion just after the event lasting > 5 min, sustained tonicoclonic movements (> 15 s) starting before the event, automatic movements, bitten tongue, cyanosis, epileptic aura (epilepsy); frequent events with somatic disturbances without a heart disease (psychiatric conditions); dizziness, dysarthria, diplopia (transient ischemic attack).

The diagnosis of vasovagal syncope is made with certainty in case of prodromal symptoms and precipitating factors, such as fear, pain, emotional stress, venopuncture, prolonged standing position. Conversely, factors leading to the suspicion of a vasovagal syncope are: events occurring just after exertion or after an unpleasant vision, smell or sound, and nausea or vomiting.

A situational syncope is ascertained when it occurs in combination with urination, defecation, cough, or swallowing.

The diagnosis of orthostatic syncope is made in the presence of orthostatic hypotension (a decrease in systolic BP ≥ 20 mmHg or systolic BP ≤ 90 mmHg) associated with syncope or pre-syncope.

On the other hand, the history hints suggesting a cardiac origin are syncope occurring in the supine position or during effort, or when preceded by palpitations, as well as a documented heart disease and/or a family history of sudden cardiac death.

Besides definitely pathologic features, the ECG interpretation should focus on those alterations non-specific *per se* which might be suggestive of arrhythmogenic conditions (i.e. negative T-wave in V_1 - V_2 , ST-segment elevation, long QT). Finally, given the prognostic importance of thoroughly ruling out any heart disease, the initial evaluation of athletes with syncope should also include ECHO. Carotid massage is recommended for athletes > 40 years. A correct approach to the subject with syncope is summarized in figure 1.

When the initial evaluation does not lead to a definite diagnosis, additional tests must be performed:

- EST: although showing a low diagnostic power^{10,14,16}, it is recommended in exercise-related syncope as well as in suspected coronary artery disease²⁰;

- HM: it is recommended in case of high pre-test probability of identifying an arrhythmic cause of syncope, in case of recurrent events, and when a heart disease is documented.

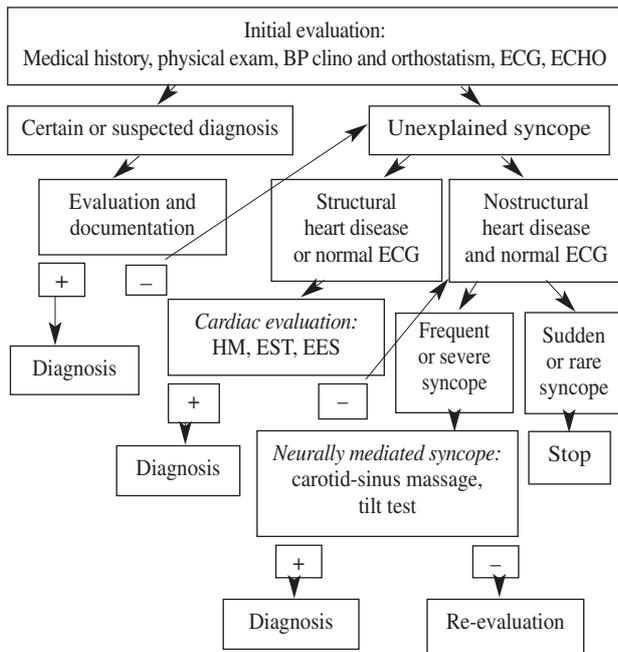


Figure 1. Diagnostic flow-chart for athletes with syncope. BP = blood pressure; ECG = electrocardiography; ECHO = echocardiography; EES = endocavitary electrophysiologic study; EST = exercise stress testing; HM = 24-hour Holter monitoring.

The need to perform additional cardiovascular tests, such as hemodynamic testing, magnetic resonance imaging, etc., will depend on the origin of the arrhythmia, known or suspected. When all exams are unable to disclose the cause of syncope and there is no evidence of organic heart disease, the following tests are recommended:

- head-up tilt testing, to show or confirm neurocardiogenic syncope. However, in athletes with respect to the general population, this test shows a low specificity, thus decreasing its diagnostic power^{7-14,22,23};
- TEPS/endocavitary EPS, when syncope is associated with palpitations, thus indicating a paroxysmal tachyarrhythmia.

In case of a heart disease, which would not be *per se* a contraindication to sports eligibility, or when there is a family history for premature sudden cardiac death, endocavitary EPS is recommended²⁰, even though not all experts agree upon this issue (consensus IIb, level of evidence B). In recurrent syncope of unknown origin, when an arrhythmia is suspected, a subcutaneous ECG loop recorder should be considered⁶.

Indications. Neurocardiogenic syncope in athletes seems to have a good prognosis^{20,21}. Therefore, once its mechanism has been definitely cleared, the athlete will be admitted to sports competitions. However, restrictive criteria must be followed in athletes practicing sports with high intrinsic risk, in which any loss of consciousness will bring about the actual possibility of severe adverse effects to the athlete and to people nearby like, for instance, spectators. In cardiac, arrhythmic or mechanic syncope, sports eligibility can be granted de-

pending on the type of arrhythmia, and/or on the potential associated clinical condition, as well as on the results of the endocavitary EPS, when available.

Palpitations. *Definition.* Palpitations are the self-perception of one's own heartbeat, usually not perceived^{24,25}. They may follow different causes and a thorough clinical history may lead to a first diagnosis. In particular, it is of great importance to differentiate palpitations caused by anxiety (as in pre-competitions) from extrasystolic palpitations and paroxysmal tachycardia. Anxiety-related palpitations are experienced as some type of anguish, and the heart rate is only slightly higher than normal. Extrasystolic palpitations are usually easy to detect, giving rise to a "missing heart-beat", "feeling of butterflies in the chest", separated by periods of normal heartbeat. Paroxysmal palpitations related to supraventricular or ventricular tachycardia (VT) begin and stop suddenly, generally accompanied by a high heart rate and a subjective feeling which is different from that experienced in anxiety-related and extrasystolic palpitations²⁶.

Pathophysiology. In subjects with structural heart disease, palpitations may be caused by major arrhythmic events, and must therefore be considered as a major symptom^{24,27}. However, even in subjects without heart disease, palpitations should not be underestimated, as they may be caused by arrhythmias leading to hemodynamic impairment, with adverse effects especially in sports with high intrinsic risk.

Diagnosis. Three main goals should be pursued when evaluating athletes with palpitations:

- to define the type of palpitations (from anxiety, extrasystoles, tachycardia);
- to rule out any underlying heart disease;
- in case of arrhythmic palpitations, try to obtain ECG recording when the athlete is experiencing palpitations²⁴⁻²⁷.

The initial evaluation must include a thorough history, familial, personal, pathologic, past and recent, an accurate physical examination, standard ECG and, when possible, an ECG during palpitations. In some cases, it is recommended to obtain a blood count, thyroid function markers, and electrolytes^{27,28}.

If the initial evaluation fails to detect an arrhythmic origin and signs of heart disease, the diagnostic process is complete. In case it brings about a suspected arrhythmic origin and/or a heart disease, additional tests are recommended. ECHO must always be considered; EST is recommended in exercise-related palpitations; HM is recommended in the presence of recurrent palpitations (on a daily basis, or so), as to have a high probability of being recorded. In case of sporadic paroxysmal palpitations, HM shows a low sensitivity, greatly limiting its prognostic power. When all previously mentioned tests fail to reach a definite diagnosis, the following should be considered:

- external event recorder or loop recorder, sometimes useful in recurrent, but rare, events (on a weekly or monthly basis)²⁹;
- electrophysiologic investigation, when palpitations are paroxysmal and/or associated with hemodynamic impairment. In subjects with no heart disease, with a high suspect of a supraventricular tachycardia, TEPS must be performed first^{26,30-34}. In patients with heart disease or suspected VT, endocavitary EPS must be performed.

Implantable loop recorders could turn of some use in the diagnosis of palpitations whose origin is still unknown at the end of the traditional evaluation^{20,29,35,36}. The need for additional tests, such as magnetic resonance imaging, hemodynamic study and/or coronary angiography, will depend on the clinical suspicion⁴.

Indications. In athletes with palpitations, sports eligibility depends on the type of arrhythmia (see specific section) and by the potential associated clinical condition. When neither important arrhythmias nor cardiac anomalies are found, no limitations to competitive sports are necessary.

Arrhythmias

Bradycardias and conduction defects. A low heart rate is frequently found in athletes, due to the autonomic adaptations to intense aerobic training and personal predisposition^{34,37-48}. Common examples are sinus bradycardia, sinus arrhythmia (especially in the young), sinoatrial and atrioventricular (AV) blocks caused by an extrinsic depression of the sinus and AV node, receding with the increase in the sympathetic tone (as it occurs during effort). However, one should recall that there are also pathologic bradycardias of structural origin deriving from intrinsic (sick sinus syndrome, or those involving the conduction system, etc.) or extrinsic causes (abnormal parasympathetic outflow) that might mimic a physiologic bradyarrhythmia⁴⁹⁻⁵¹.

Sinus bradycardia, sinus arrhythmia, and sinoatrial blocks. The evaluation criteria are based upon the type and intensity of training, the level of competition and the duration of sports practice^{39,41,44,46,48}:

- in deconditioned subjects a daytime heart rate ≤ 50 b/min at rest, not reaching 120 b/min during step test and/or associated with symptomatic bradycardia during recovery may be considered as non-physiologic. In this case it is recommended to obtain a second-level evaluation (EST and HM);
- in trained subjects practicing sports with high cardiovascular involvement, bradyarrhythmias, even marked, can be found, not necessarily implying a pathologic condition. However, a daytime heart rate ≤ 40 b/min at rest, associated with a heart rate during step test of

≤ 100 b/min, renders mandatory a second-level evaluation (EST and HM);

- third-level tests (pharmacological, TEPS/endocavitary EPS) can throw a light upon unclear cases^{49,52}.

In subjects with long QT syndrome (especially children and young patients), bradycardia may be a part of the clinical picture. Therefore, a long QT must always be ruled out in subjects with bradycardia.

Sports eligibility can be granted to:

- subjects free from heart disease and without any bradycardia-related symptoms;
- subjects in whom first-level tests have ruled out a sick sinus syndrome;
- subjects undergoing second-level tests, in whom EST shows a normal chronotropic response (reaching at least 85% of the age-predicted maximum heart rate) and HM fails to document ventricular pauses > 3 s. In elite athletes or in highly-trained subjects, especially when practicing aerobic sports, pauses > 3 s can be admitted, especially when occurring during night-time, provided that they are not associated with symptoms or arrhythmias likely related to bradycardia.

Atrioventricular blocks. Based on electrocardiographic and electrophysiologic features, AV blocks can be classified as follows:

- first-degree AV blocks with narrow QRS. A PR interval > 0.20 s at rest, which normalizes with hyperpnea and step test, does not represent a contraindication to sports^{34,37-40}. When atypical features are found, a second-level evaluation becomes mandatory (EST, HM, ECHO);
- second-degree AV blocks with narrow QRS (Luciani-Wenckebach or Mobitz type 1). When such an anomaly is found at standard rest ECG, second-level tests (EST, HM, ECHO) are mandatory. Sports eligibility can be granted to asymptomatic subjects without evidence of heart disease, in case of normalization of AV conduction with increasing heart rate and no ventricular pauses > 3 s at HM. In top-level athletes or in highly-trained sportsmen, in particular when practicing endurance sports, ventricular pauses > 3 s can be admitted, especially when occurring during sleep, provided that they do not match with bradycardia-related symptoms^{39,41,44,46-48}. Different types of nodal AV block are sometimes found in athletes (P-R periodicity with minimum increase, atypical periodicity and some types of AV block type 2:1, 3:1, etc.); their importance is similar to the Mobitz 1 type, and therefore the previous considerations also apply. Rarely, when a first-degree AV block with narrow QRS is observed during effort, endocavitary EPS is mandatory;
- advanced and complete AV blocks. When such conduction defects are found, second- and third-level tests are mandatory. Sporadic types, related to vagal hyperactivity (usually occurring during night-time and detected at HM) do not represent a contraindication to sports eligibility, although they require a tailored eval-

uation by specialists. Persistent types, not related to vagal hyperactivity, contraindicate sports, at least until a pacemaker has been implanted (see Pacemaker section)⁵¹.

Bradycardia-related supraventricular arrhythmias. Passive supraventricular heart rhythms, wandering pacemaker, isorhythmic AV dissociation, found at standard rest ECG and normalizing during spontaneous hyperpnea and/or exercise (step test) are a physiologic phenomenon that do not contraindicate *per se* sports activity^{39-41,47,48}.

Ventricular conduction defects. Right ventricular conduction defects with narrow QRS (< 0.12 s, also known as right bundle branch block, RBBB) in an otherwise normal heart do not contraindicate sports^{34,37,38,53}.

Right ventricular conduction defects with wide QRS (≥ 0.12 s), global left ventricular conduction defect (left bundle branch block, LBBB), superior left ventricular conduction defect (left anterior fascicular block, LAFB), intermittent and bifascicular right and left ventricular conduction defects (RBBB + LAFB) require a second-level evaluation (ECHO, EST, HM)⁵⁴⁻⁵⁹. Additional tests, either invasive or non-invasive, should be performed in case of a suspected coronary origin, with the aim at assessing the coronary tree.

In the absence of any findings suggesting a heart disease, eligibility can be granted to all subjects with isolated RBBB and LAFB, independently of the type of practiced sport. In case of LBBB and LAFB + RBBB with wide QRS, eligibility should be limited to sports of minimum to moderate cardiovascular involvement belonging to group B2 and some of group B1 (riding and sailing). Eligibility to more demanding sports is subordinated to third-level tests, mainly endocavitary EPS (consensus class IIb, level of evidence B)^{51,52,60}.

Atrioventricular blocks of any degree associated with ventricular activation defects. Third-level tests, endocavitary EPS in particular, are mandatory.

Premature beats. Supraventricular premature beats. Supraventricular premature beats (SVPB) are quite common in healthy athletes^{42,43,45,47}. Uncomplicated SVPB (isolated or sporadic, non-repetitive) at standard rest ECG, not increasing with effort (step test), in subjects with a normal heart at clinical examination do not contraindicate sports activity. In case of non-sporadic and repetitive SVPB, sports eligibility is subordinated to the results of second-level tests (ECHO, EST, HM).

Once the second-level evaluation has been performed and no symptoms or heart disease have been identified, sports eligibility might be granted if subjects do not develop persistent tachycardia and/or significant bradyarrhythmia (sinoatrial blocks, AV blocks) during or after effort.

Ventricular premature beats. Ventricular premature beats (VPB) are quite common in athletes^{42,43,45,47}. The main feature conditioning prognosis and, as a consequence, playing a role in sports eligibility, is the possible underlying heart disease^{1-4,61-70}. However, to date, randomized and large clinical trials dealing with this topic are scanty: trials performed in the general population, including athletes and sedentary subjects, have shown that, in the absence of a documented heart disease, VPB do not increase the risk of malignant ventricular arrhythmias⁷¹⁻⁷⁸.

In athletes, when ECG at rest and/or during effort (step test) shows monomorphic, non-repetitive VPB, extremely low in number ($< 2-3$ during the whole recording), no further evaluation is required, provided that the family clinical history does not suggest any episode of premature sudden cardiac death or arrhythmogenic heart disease, the athlete be asymptomatic, physical examination be negative and the ECG, save for VPB, be otherwise absolutely normal. Cases with suspected heart disease, as well as cases with more than sporadic VPB should undergo a second-level evaluation (ECHO, EST e HM). The need for additional tests, namely third-level, will be individually based, according to the type of the suspected or documented cardiac anomaly. Sports eligibility can be granted:

- in the absence of a family clinical history of sudden cardiac death or arrhythmogenic heart disease;
- in the absence of any documented heart disease;
- in the absence of major symptoms (persistent palpitations, pre-syncope and syncope) related to arrhythmogenic abnormalities;
- in the presence of VPB < 30 /hour at HM, and in case of monomorphic, non-repetitive, non-premature, exercise-unrelated VPB.

In selected cases, sports eligibility can be granted even with VPB > 30 /hour, as well as with sporadic couplets, provided that a heart disease has previously been thoroughly ruled out. The large majority of experts have stated a positive opinion on both issues, even though without definite data (consensus class IIa, level of evidence C). Sports eligibility must be rejected in the following cases:

- a family history of sudden cardiac death or arrhythmogenic abnormalities;
- relevant heart disease, or likely to cause malignant arrhythmias;
- VPB whose electrophysiologic features are suggestive of risk, such as couplets with a high heart rate (R-R interval < 400 ms), repetitive and polymorphic, and premature VPB.

All ineligible cases may be re-evaluated after 4-6 months of detraining.

Supraventricular tachycardias. Supraventricular tachycardias can be paroxysmal, persistent and permanent. In all of these conditions, athletes should undergo a second-level evaluation (ECHO, EST, HM). The electro-

physiologic study (endocavitary EPS or TEPS) at rest or on effort should be performed in the paroxysmal forms with a suspected reentry tachycardia (nodal tachycardia, AV nodal reentry tachycardia)^{24,30-33,79}.

Supraventricular paroxysmal tachycardias in the absence of Wolff-Parkinson-White syndrome. In most cases, these tachycardias are provoked by nodal reentry; in the remaining cases, they are caused by a concealed accessory pathway, an atrial reentry, or by foci located in the atria or pulmonary veins^{26,80-84}. Those caused by re-entry are frequently inducible during electrophysiologic study (TEPS/endocavitary EPS) at rest and/or during effort. Sports eligibility can be granted when^{63,70,79,85-87}:

- avoiding sports with high intrinsic risk;
- underlying heart diseases have been ruled out;
- arrhythmias are sporadic and not related to exertion;
- arrhythmic events do not induce alarming symptoms (dizziness, pre-syncope, syncope) and are self-terminated;
- arrhythmias, either spontaneous or induced by endocavitary EPS or TEPS (at rest and/or during effort), do not reach a high heart rate;
- AV conduction accessory pathways are absent (otherwise, refer to Wolff-Parkinson-White syndrome-WPW).

In the remaining cases, sports eligibility is conditioned by the successful catheter ablation of the arrhythmic substrate.

Repetitive and persistent supraventricular tachycardias. This group includes inappropriate sinus tachycardia, reentrant tachycardia by anomalous slow decremental pathway "type Coumel" and atrial focal tachycardia by increased automaticity^{26,80-84}. Repetitive forms, and persistent forms in particular may cause a hypokinetic cardiomyopathy secondary to tachycardia⁸⁸⁻⁹⁶. They are susceptible to catheter ablation, which has good chances of resulting fully curative.

Not contraindicating sports activity are the types of tachycardia, usually repetitive, which occur without a heart disease, showing a heart rate, at rest and during effort, just a little above normal, and not causing hemodynamic impairment. In such cases, however, it is recommended to perform serial clinical and laboratory tests (at least once a year) of second level (ECHO, HM) confirming the stability of the arrhythmic and hemodynamic conditions. In the remaining cases, sports eligibility is subjected to catheter ablation.

Paroxysmal or persistent atrial fibrillation. Atrial fibrillation (AF) may occur in otherwise healthy hearts or as an additional feature in the setting of a heart disease⁹⁷⁻¹⁰⁶. In particular, AF may also represent the hallmark of life-threatening conditions such as the Brugada syndrome, myocarditis, arrhythmogenic right ventricular cardiomyopathy (ARVC), dilated cardiomyopathy, etc., which must therefore be ruled out.

In case of AF second-level tests must be performed (ECHO, EST, HM) and possibly TEPS/endocavitary EPS at rest and on exertion, the latter with the aim of identifying potential triggers for the arrhythmia, such as tachycardia from nodal reentrant or accessory pathways, and foci in the pulmonary veins liable of transcatheter ablation¹⁰⁷⁻¹¹². Sports eligibility can be granted provided that^{63,70}:

- subjects do not participate in sports with intrinsic risk;
- underlying heart diseases have been ruled out;
- possible precipitating factors have been searched for and eliminated (hyperthyroidism, alcohol, drugs and arrhythmogenic compound, etc.);
- no cause-effect relation is found between exercise and the ventricular arrhythmia;
- arrhythmic events are rare, do not cause remarkable symptoms (dizziness, pre-syncope, syncope, etc.), heart rate is not high, and they are self-terminating;
- sick sinus syndrome has been ruled out;
- antegrade AV conduction accessory pathways are not found (else, see WPW section);
- the subject is not on anticoagulant therapy.

Permanent atrial fibrillation. Usually this arrhythmia contraindicates eligibility to competitive sports. Eligibility can be granted, limited to group B2 sports and non-competitive athletic activities (group A) provided that^{63,70,113}:

- structural heart diseases have been ruled out;
- the ventricular arrhythmia does not cause significant symptoms;
- the heart rate, measured at EST and HM, does not exceed the age-predicted maximum heart rate, without significant bradycardia (heart rate < 40 b/min and/or pauses > 3 s) and/or bradycardia-related arrhythmias.

Atypical and typical atrial flutter (common and uncommon). Atrial flutter is very uncommon in healthy athletes^{43-45,114-116}. As it may cause high heart rates during effort, as a rule, it is not compatible with sports activity.

In all ineligible patients, transcatheter ablation, with a high rate of success in typical flutter and few complications, can allow the athlete without organic heart disease return to sports competitions.

Ventricular preexcitation. Wolff-Parkinson-White syndrome. At the origin of the ECG pattern of WPW is a congenital anomaly, namely an AV fast-conduction accessory pathway bypassing the normal conduction system. Usually, it is not associated with heart disease. WPW has a prevalence of 1-2% in the general population and sometimes disappears with age¹¹⁷⁻¹²². It may be complicated by different types of arrhythmia¹²³⁻¹³⁹, in particular:

- AV reentrant tachycardia, with antegrade conduction through the nodal pathway and retrograde through the accessory pathway (AV reentrant orthodromic tachycardia);

- the less common AV reentrant tachycardia, with antegrade conduction through the accessory pathway and retrograde conduction through the nodal pathway (AV reentrant antidromic tachycardia);
- AF which may be partially or entirely preexcited and may influence prognosis considerably, because of a dangerous tendency toward ventricular fibrillation.

At the pre-participation screening, subjects can either be asymptomatic or complain of symptoms from one or more arrhythmias. In WPW, symptoms may begin at anytime in life: the lack of symptoms, therefore, does not protect from arrhythmic risk.

In asymptomatic subjects without underlying heart disease, sports eligibility is conditioned by TEPS results at rest and during effort (or alternatively endocavitary EPS at rest and with isoproterenol)¹⁴⁰⁻¹⁵⁹. Children < 12 years without an underlying heart disease are an exception, as the risk of AF and/or sudden cardiac death is theoretical, thus tests for risk stratification, such as TEPS and endocavitary EPS, can be performed later in life^{123-128,132}.

Sports eligibility is rejected to subjects complaining of palpitations, or arrhythmia-related symptoms, and to asymptomatic subjects with:

- underlying heart disease;
- AF inducibility at TEPS/endocavitary EPS, with minimum R-R \leq 240 ms between preexcited beats at rest, and 200 ms during effort.

Sports eligibility is granted to asymptomatic subjects with:

- inducibility at TEPS/endocavitary EPS of preexcited AF, with minimum R-R > 240 ms at rest and > 200 ms during effort;
- AF and/or AV reentrant tachycardia not inducible at TEPS/endocavitary EPS at rest and during effort, and antegrade effective refractory period of accessory pathway > 240 ms at rest and > 200 ms during effort.

In case of borderline electrophysiologic parameters, atrial susceptibility should be evaluated (easy AF inducibility with non-aggressive protocols and/or induction of sustained AF, \geq 30 s) and the inducibility of AV reentrant tachycardia (testifying a retrograde conduction of the accessory pathway). In such cases, especially in athletes participating in sports with a high intrinsic risk (driving, scuba diving, climbing, etc.), sports eligibility should be allowed with caution.

When subjects are granted sports eligibility, serial TEPS should be performed in those with borderline electrophysiologic parameters, as well as in those who develop symptoms.

Ventricular preexcitation due to Mahaim fibers. The underlying substrate is a congenital slow accessory pathway, with decremental conduction. As a rule, such a pathway connects the right atrium (or the AV node) to the right ventricle¹⁶⁰⁻¹⁶³. Its precise incidence is unknown. Many asymptomatic cases are misdiagnosed as WPW, later properly diagnosed by means of the electrophysiologic study.

Subjects are rarely symptomatic, representing less than 1% of all cases with symptomatic ventricular pre-excitation. In such cases, the most common arrhythmia is the antidromic reentrant tachycardia, in which the right accessory pathway acts as orthodromic branch and the normal conduction system is the antidrome pathway in the circuit. Therefore, the resulting tachycardia has generally a morphology “LBBB type” and, as a rule, does not cause high heart rate, as the circuit is made of slow conducting fibers.

Usually, such a condition is found in healthy hearts, with a good prognosis. In fact, unlike WPW, a possible AF can never be conducted with high response rate to the ventricles, because the accessory pathway type Mahaim have a decremental conduction, similar to that of the AV node.

Sports eligibility can be granted, to asymptomatic subjects without inducible reentrant ventricular arrhythmias, once the electrophysiologic study has been performed. In symptomatic patients, the same criteria stated for supraventricular paroxysmal tachycardias without WPW should be adopted.

Ventricular tachycardias. *Nonsustained ventricular tachycardias.* Nonsustained ventricular tachycardias (NSVT) are characterized by \geq 3 beats, lasting < 30 s, without any hemodynamic impairment.

It is an uncommon arrhythmia in healthy subjects, thus generally requiring a second-level evaluation (ECHO, EST, HM)^{42,43,45,47,78}. Additional tests, namely of the third level (in particular endocavitary EPS as well as those needed to rule out a structural disease), will be individually based according to the type of the suspected or documented disease.

Sports eligibility can be granted¹⁶⁴⁻¹⁷¹:

- in the absence of a clinical family history of sudden cardiac death or arrhythmogenic disease;
- in the absence of arrhythmia-related symptoms;
- in the absence of a relevant heart disease, or any disease likely to cause malignant ventricular arrhythmias;
- in case of NSVT occurring sporadically over 24 hours;
- if the R-R interval is > 400 ms (heart rate < 150 b/min) during NSVT;
- in the absence of VPB considered at risk (see specific section).

Sports eligibility is rejected in the presence of:

- a family history of sudden cardiac death or hereditary arrhythmogenic diseases;
- an underlying heart disease;
- multiple episodes of NSVT over 24 hours and/or R-R interval < 400 ms during NSVT and/or the coexistence of couplets with R-R interval < 400 ms and of frequent VPB (> 30/hour);
- a straightforward cause-effect relation between the arrhythmia and exercise.

All criteria have met with the Experts' consensus, with a wide majority (class IIa), even if without an exhaustive scientific evidence (level of evidence C).

Slow ventricular tachycardias or ventricular accelerated rhythm. Ventricular accelerated rhythm is defined as a ventricular rhythm with a heart rate < 100 b/min. Especially when occurring during periods of sinus bradycardia, and without a heart disease, usually, it does not represent a contraindication to sports activity¹⁷². However, sports eligibility depends on the results of the second-level evaluation (ECHO, EST, HM).

Benign ventricular tachycardias (fascicular ventricular tachycardia and right ventricular outflow tachycardia). Benign VT include fascicular VT¹⁷³⁻¹⁷⁵ and automatic right ventricular outflow tachycardia, (RVOT)¹⁷⁶⁻¹⁷⁸. They are both characterized by the absence of an underlying heart disease and good hemodynamic tolerance, similar in this way to supraventricular tachycardias, also as far as prognosis is concerned.

Fascicular VT originates in the inferior and left part of the interventricular septum, with a paroxysmal pattern, causing a QRS morphology type RBBB + left anterior hemiblock. RVOT is due to an automatic focus placed in the outflow portion of the right ventricle (rarely in the left), can be repetitive or paroxysmal, showing a QRS morphology type LBBB with right deviation. As a consequence of its automaticity, this arrhythmia can be easily detected during continuous ECG recordings by monomorphic VPB either isolated or as couplets, triplets and sequences of varying duration, and non-sustained (NSVT). Usually, the R-R interval of complex VPB is not a short one. Repetitiveness is enhanced by exertion and some subjects even show a precise threshold, above which the arrhythmia becomes sustained.

Both types require a second-level evaluation (ECHO, EST, HM) to exclude a heart disease and document how the arrhythmia occurs, as well as its features.

Fascicular VT can be misdiagnosed as a paroxysmal supraventricular tachycardia with aberration. Endocavitary EPS should be performed in all unclear cases. RVOT can bring about serious doubts concerning differential diagnosis with VT originating from the outflow tract in ARVC. In case of doubts, endocavitary EPS is recommended: in RVOT, in fact, endocavitary EPS at rest fails to reproduce the tachycardia, while this can be obtained by isoproterenol. In ARVC, ventricular pacing is easily capable of inducing a sustained VT by reentry.

Criteria for sports eligibility in fascicular VT are those adopted for supraventricular paroxysmal tachycardias without WPW. RVOT is usually enhanced by exertion, and it is not compatible with highly demanding sports, save for after successful catheter ablation. Asymptomatic subjects with couplets, triplets and only few beats of NSVT, with a moderate ventricular rate at rest and during effort, may be an exception still limited to sports with minimum to moderate cardiovascular involvement of group B2 and non-competitive athletic activities (group A).

Malignant ventricular tachycardias. The finding, as well as the history documentation of sustained VT, polymorphic VT, torsades de pointes, and/or cardiac arrest in the form of ventricular fibrillation, usually contraindicates eligibility to competitive and recreational sports¹⁷⁹⁻¹⁸².

An exception is represented by ventricular arrhythmias occurring in the context of acute and wholly reversible disease, unlikely to relapse.

Potentially arrhythmogenic conditions

Mitral valve prolapse. A full chapter is dedicated to this topic. The relationship between mitral valve prolapse (MVP) and arrhythmias may be summarized as follows¹⁸³⁻¹⁸⁹:

- in many cases, the association between MVP and ventricular arrhythmias is due to the high prevalence of both conditions in the athletic population and they may coexist in the same subject;
- in some cases MVP is associated with a second arrhythmogenic condition, even a severe one, such as ARVC;
- in some cases ventricular arrhythmias seem to correlate with MVP and they might develop due to myocardial stretching operated by chordae tendinae from an impaired valvular system. Mostly the most severe arrhythmias are found in patients with severely degenerated heart valves.

To the purpose of sports eligibility, two features are of the utmost importance: the degree of valve dysfunction, and the type of ventricular arrhythmia. As for ventricular arrhythmias, it remains valid what has already been stated regarding each type. In particular, a conservative and protective approach should be adopted (non-eligibility) for ventricular repetitive arrhythmias, with a short R-R interval, especially when facilitated by exertion.

The Brugada syndrome. The Brugada syndrome is a genetic condition to be clearly defined yet and it is characterized by¹⁹⁰⁻²¹⁰:

- ST-segment elevation > 2 mm in leads V₁-V₂ (V₃) with tent- or saddle-shape morphology, sometimes associated with RBBB;
- risk of sudden cardiac death from malignant ventricular arrhythmias (sustained VT, ventricular fibrillation).

Patients at the highest risk are those with a family history of sudden cardiac death or syncope, while the predictive power of endocavitary EPS in asymptomatic subjects remains debated. Although no relation between exercise and ventricular arrhythmias exists (as a matter of fact, save for exceptions, sudden cardiac death occurs at rest or during sleep), subjects with symptomatic Brugada syndrome should not be granted sports eligibility.

Asymptomatic relatives of subjects with Brugada syndrome, even if with normal rest ECG, cannot be granted sports eligibility unless they undergo a phar-

macologic test (such as with flecainide), ruling out the syndrome. There is not yet a common agreement regarding the approach to asymptomatic subjects with Brugada-type ECG, with no relatives diagnosed as Brugada syndrome and/or with a history of sudden cardiac death.

Arrhythmogenic right ventricular cardiomyopathy.

This topic has gained its own paragraph in the chapter dedicated to cardiomyopathies. In the present chapter, we recall that ARVC, potentially causing sudden cardiac death, especially during effort, is not compatible with sports^{4,211-218}. Advanced forms are the most dangerous, being also the easiest to diagnose, as their clinical features are quickly detectable, when sought for thoroughly.

Being a potentially progressive condition, subjects at risk (relatives of diagnosed patients) and/or with barely suspected forms, without ventricular arrhythmias, can be temporarily granted sports eligibility, but they should be followed up with clinical and laboratory tests (ECHO, EST, HM) at least yearly.

Long QT syndrome. The QT interval is considered as long when its corrected value (QT in s/square root of the preceding R-R interval in s) exceeds 440 ms in males and 460 ms in females. QT should always be measured in lead D2²¹⁹⁻²²⁷.

In the presence of long QT, electrolytes are to be measured first (K⁺ and Ca⁺⁺), and subjects should be questioned regarding the possible intake of drugs likely to cause QT prolongation (e.g., antibiotics, antihistamines, etc.). In doubt cases, (QT at the upper limit) a second-level evaluation is mandatory (ECHO, EST, HM).

Congenital long QT syndrome contraindicates sports activity of any type, even without documented major ventricular arrhythmias.

Short PR interval (Lown-Ganong-Levine syndrome).

Asymptomatic subjects with short PR and narrow QRS can obtain sports eligibility. When a delta wave is suspected (a slurring in the initial part of the QRS complex) the electrophysiologic study is recommended to rule out ventricular preexcitation.

Patients complaining of paroxysmal palpitations should undergo an electrophysiologic study to find out the underlying mechanism.

Indications to catheter ablation in athletes

Catheter ablation of focal and reentrant tachyarrhythmias is nowadays a definite tool, yielding good results with a minimum risk of side effects^{111,228-246}.

Lethal complications are uncommon (< 1%), and they may occur when ablation is performed in the left cardiac section. Among non-lethal complications, AV block is the most life-threatening, even though it only

refers to catheter ablation of nodal reentrant tachycardia (< 1%) and antero-septal accessory pathways.

In athletes, indications to catheter ablation are slightly different from those in the general population. In athletes, in fact, the goal is not limited to a reduction in the risk of sudden cardiac death, or the elimination of invalidating symptoms, but goes further to re-admission to sports competitions when the arrhythmias are only theoretically dangerous.

The first indication in athletes is limited to symptomatic WPW from high-rate AF, symptomatic WPW from paroxysmal tachycardia and, secondarily, to asymptomatic WPW with high theoretical risk at TEPS/endocavitary EPS. The second indication is for subjects with symptomatic tachycardia. In the third case, the risk/benefit ratio of transcatheter ablation should be evaluated accurately. The risk is related to the different procedural techniques and the benefit is represented by a better quality of life and the possibility of returning to competitions. In short, catheter ablation has a favorable risk/benefit ratio in the following conditions:

- paroxysmal reentrant tachycardia (without WPW), except for those with rare and nonsustained episodes, and when heart rate during tachycardia is similar or lower than the age-predicted maximum heart rate;
- persistent and repetitive tachycardia, except for slow rate episodes;
- symptomatic WPW;
- asymptomatic WPW, electrophysiologically “at risk” or “borderline”;
- typical common and uncommon atrial flutter;
- symptomatic fascicular VT or RVOT.

Nowadays, catheter ablation of AF and of some types of atypical atrial flutter (especially from the left atrium) is to be considered an experimental application²⁴⁷⁻²⁵⁰, hence indicated only in few selected cases.

Return to sports activity after transcatheter ablation.

After successful transcatheter ablation, patients may be considered eligible to competitive sports if they undergo ECHO and HM and provided that:

- they do not suffer from any heart disease causing non-eligibility *per se*;
- ablation being performed at least 3 months previously;
- the ECG does not show any signs of ventricular preexcitation in case of WPW;
- they are asymptomatic, without any clinical recurrence of tachycardia.

In selected uncertain cases, follow-up TEPS/endocavitary EPS is recommended to monitor the real outcome of the technique. In the remaining cases, it is not necessary²⁵¹.

Sports activity in subjects with a pacemaker

Eligibility to competitive sports for cardiac subjects with a pacemaker depends on the type of the underlying

ing heart disease and on the presence/absence of associated ventricular arrhythmias^{28,63,70}.

Patients with pacemaker and no signs of heart disease will be granted eligibility to sports with minimum cardiovascular involvement (group B2) and non-competitive athletic activities (group A), provided that EST and HM have documented an appropriate increase in paced heart rate during exercise, and no significant associated arrhythmias.

Subjects with pacemaker will be discouraged from participating in contact sports, because of the possible damage occurring to electrocatheters and pacing units. Furthermore, the possible risk of electromagnetic interferences should be closely evaluated.

Sports activity in patients with an implantable cardioverter-defibrillator

Most patients with a cardioverter-defibrillator suffer from a severe structural heart disease, contraindicating *per se* competitive sports. Subjects with a cardioverter-defibrillator and a normal or "near-normal" cardiac function, as well as those without any evidence of structural heart disease, can be granted eligibility only to sports of minimum to moderate cardiovascular involvement (group B2) and non-competitive athletic activities (group A)^{28,63,70}. However, no exception can be made for patients with an exercise-related increase in sympathetic activity that may worsen malignant ventricular arrhythmias (e.g., congenital long QT syndrome, ARVC, catecholaminergic VT, etc.)^{4,182,211-225}.

Sports eligibility might only be granted after 6 months post-implantation or following the most recent arrhythmic episode requiring defibrillator intervention. In fact, the efficacy of the defibrillator to stop malignant ventricular arrhythmias during exercise and sports activity in particular, has not been cleared yet. Furthermore, heart rate threshold values for device activation should be carefully evaluated at EST and HM in order to minimize the risk of inappropriate shocks related to physiologic exercise-induced sinus tachycardia.

Patients with a cardioverter-defibrillator must be discouraged from practicing contact sports that may damage the device or the electrocatheter. Furthermore, the possible risk of electromagnetic interferences should also be assessed.

References

1. Maron BJ, Roberts WC, McAllister HA Jr, et al. Sudden death in young athletes. *Circulation* 1980; 62: 218-23.
2. Furlanello F, Bettini R, Cozzi F, et al. Ventricular arrhythmias and sudden death in athletes. *Ann NY Acad Sci* 1984; 427: 253-79.
3. Maron BJ, Epstein SE, Roberts WC. Causes of sudden death in competitive athletes. *J Am Coll Cardiol* 1986; 7: 204-14.

4. Thiene G, Nava A, Rossi L, Pennelli N. Right ventricular cardiomyopathy and sudden death in young people. *N Engl J Med* 1988; 318: 129-33.
5. El-Sherif N, Turitto G, Pedalino RP, et al. T-wave alternans, a powerful index for malignant ventricular arrhythmias. Fact or fiction? In: Raviele A, ed. *Cardiac arrhythmias* 2001. Milano: Springer Verlag Italia, 2002: 237-47.
6. Calkins H, Seifert M, Morady F. Clinical presentations and long-term follow-up of athletes with exercise-induced vasodepressor syncope. *Am Heart J* 1995; 129: 1159-64.
7. Pedersen WP, Janosik DL, Goldenberg IF, et al. Post-exercise asystolic arrest in a young man without organic heart disease: utility of head-up tilt testing in guiding therapy. *Am Heart J* 1989; 118: 410-3.
8. Rechavia E, Strasberg B, Agmon J. Head-up tilt table evaluation in a trained athlete with recurrent vaso-vagal syncope. *Chest* 1989; 95: 689-91.
9. Grubb BP, Temesey-Armos PN, Samoil D, et al. Tilt table testing in the evaluation and management of athletes with recurrent exercise-induced syncope. *Med Sci Sports Exerc* 1993; 25: 24-8.
10. Sneddon JF, Scalin G, Ward DE, et al. Exercise induced vasodepressor syncope. *Br Heart J* 1994; 71: 554-7.
11. Osswald S, Brooks R, O'Nunain SS, et al. Asystole after exercise in healthy persons. *Ann Intern Med* 1994; 120: 1008-11.
12. Sakaguchi S, Shultz JJ, Remole SC, Adler SW, Lurie KG, Bendit DG. Syncope associated with exercise, a manifestation of neurally mediated syncope. *Am J Cardiol* 1995; 75: 476-81.
13. Kosinski D, Grubb BP, Kip K, Hahn H. Exercise-induced neurocardiogenic syncope. *Am Heart J* 1996; 132: 451-2.
14. Abe H, Nakashima Y, Kohshi K, Kuroiwa A. Exercise-induced neurally mediated syncope. *Jpn Heart J* 1997; 38: 535-9.
15. Tortorella G, Tomasi C, Manari A, et al. Syncope in middle-aged endurance athletes. *G Ital Cardiol* 1998; 28: 517-9.
16. Bottoni N, Lolli G, Tomasi C, et al. Epidemiologia della sincope nella popolazione sportiva. *Int J Sports Cardiol* 1999; 8: 117-9.
17. Olshansky B. Evaluation of syncope in the athlete. *G Ital Cardiol* 1999; 29: 344-51.
18. Kosinski D, Grubb BP, Karas BJ, Frederick S. Exercise-induced neurocardiogenic syncope: clinical data, pathophysiological aspects, and potential role of tilt table testing. *Eurpace* 2000; 2: 77-82.
19. Alboni P, Brignole M, Menozzi C, et al. Diagnostic value of history in patients with syncope with or without heart disease. *J Am Coll Cardiol* 2001; 37: 1921-8.
20. Task Force Report of European Society of Cardiology. Guidelines on management (diagnosis and treatment) of syncope. *Eur Heart J* 2001; 22: 1256-306.
21. Brignole M, Menozzi C, Manari A, Gaggioli O. Classificazione e diagnosi della sincope neuromediata nello sportivo. *Int J Sports Cardiol* 1995; 4: 35-43.
22. Ferrario G, Peci P, Nicoli T, Sgherzi B, Giani P. Long distance runner's heart: is it a condition predisposing to malignant vasovagal syndrome? *Eur J Card Pacing Electrophysiol* 1993; 4: 264-8.
23. Ferrario G, Nicoli T, Peci P, Giani P. Cardiac adaptation to training: is it the key to understand the high incidence of positive tilting test in athletes? *Pacing Clin Electrophysiol* 1996; 19: 710.
24. Zimetbaum P, Josephson ME. Evaluation of patients with palpitations. *N Engl J Med* 1998; 338: 1369-73.
25. Brugada P, Gursoy S, Brugada J, Andriess E. Investigation of palpitations. *Lancet* 1993; 341: 1254-8.
26. Delise P. Le tachiaritmie sopraventricolari. Dall'elettrocar-

- diogramma all'ablazione transcateretere. Roma: Edizioni CESI, 1997.
27. Weber BE, Kapoor WH. Evaluations and outcomes of patients with palpitations. *Am J Med* 1996; 100: 138-48.
 28. Zipes DP, Miles WM, Klein LS. Assessment of patient with cardiac arrhythmia. In: Zipes DP, Jalife J, eds. *Cardiac electrophysiology: from cell to bedside*. Philadelphia, PA: WB Saunders, 1995: 1009-12.
 29. Crawford MH, Bernstein SJ, Deedwania PC, et al. ACC/AHA guidelines for ambulatory electrocardiography: executive summary and recommendations. A report of the American College of Cardiology/American Heart Association task force on practice guidelines (committee to revise the guidelines for ambulatory electrocardiography). *Circulation* 1999; 100: 886-93.
 30. Gallagher JJ, Smith WM, Kasell J, et al. Use of the esophageal lead in the diagnosis of mechanisms of reciprocating supraventricular tachycardia. *Pacing Clin Electrophysiol* 1980; 3: 440-8.
 31. Gallagher JJ, Smith WM, Kasell J, et al. Esophageal pacing: a diagnostic and therapeutic tool. *Circulation* 1982; 65: 336-44.
 32. Bonso A, Delise P, D'Este D, et al. Utilità dello studio elettrofisiologico transesofageo nei soggetti apparentemente non cardiopatici con episodi di cardiopalmo parossistico da causa indeterminata. *G Ital Cardiol* 1990; 20 (Suppl I): 135.
 33. Delise P, Bonso A, Corò L, et al. Rilievi elettrofisiologici endocavitari e transesofagei nella fibrillazione atriale idiopatica. *G Ital Cardiol* 1991; 21: 1093-100.
 34. Venerando A, Rulli V. Frequency, morphology and meaning of the ECG anomalies found in Olympic marathon runners and walkers. *J Sports Med Phys Fitness* 1964; 50: 135-41.
 35. Krahn AD, Klein GJ, Yee R, Skanes AC. Randomized assessment of syncope trial: conventional diagnostic testing versus a prolonged monitoring strategy. *Circulation* 2001; 104: 46-56.
 36. Waktare JE, Camm AJ. Holter and event recordings for arrhythmia detection. In: Zareba W, Maison-Blanche P, Locati EH, eds. *Noninvasive electrocardiology in clinical practice*. Armonk, NY: Futura Publishing Company, 2001: 3-30.
 37. Beckner GL, Winsor T. Cardiovascular adaptations to prolonged physical efforts. *Circulation* 1954; 9: 835-42.
 38. Raskoff WS, Goldman S, Cohn K. The athletic heart. *JAMA* 1976; 236: 158-70.
 39. Zeppilli P, Fenici R, Sassara M, et al. Wenckebach second degree A-V block in top ranking athletes: an old problem revisited. *Am Heart J* 1980; 100: 821-8.
 40. Talan D, Bauernfeind R, Ashley W, et al. Twenty-four hour continuous ECG recordings in long-distance runners. *Chest* 1982; 82: 19-23.
 41. Venerando A. Adattamenti cardiovascolari ai differenti tipi di sport. In: Venerando A, Zeppilli P, eds. *Cardiologia dello sport*. 29th edition. Milano: Masson Italia, 1982.
 42. Buja GF, Santini G, Miraglia G, Nava A. Twenty-four hour Holter monitoring in the conquest of K2. *Int J Sports Cardiol* 1985; 2: 43-50.
 43. Caselli G, Pelliccia A, Spataro A, et al. L'ECG dinamico (ECGD) nella valutazione diagnostica e prognostica delle aritmie ipocinetiche negli atleti. In: *Atti del II Congresso Nazionale SIC Sport*. Bologna: Aulo Gaggi Editore, 1985: 255-66.
 44. Zeppilli P, Zuliani U, Biffi A, et al. Indagini cardiologiche e metaboliche nel corso di una 24 ore di sci da fondo. *G Ital Cardiol* 1985; 15: 493-52.
 45. Maron BJ, Bodison S, Wesley YE, Tucker E, Green KJ. Results of screening a large group of intercollegiate competitive athletes for cardiovascular disease. *J Am Coll Cardiol* 1987; 10: 1214-22.
 46. Bettini R, Furlanello F, Vecchiet L, et al. Ritmo cardiaco nell'atleta: studio con Holter in calciatori "top level" ed ex calciatori professionisti. *G Ital Cardiol* 1990; 20: 810-8.
 47. Bjornstad H, Storstein L, Dyre Meen H, Hals O. Electrocardiographic findings according to level of fitness and sport activity. *Cardiology* 1993; 83: 268-79.
 48. Bjornstad H, Storstein L, Dyre Meen H, Hals O. Ambulatory electrocardiographic findings in top athletes, athletic students and control subjects. *Cardiology* 1994; 84: 42-50.
 49. Jose AD. Effect of combined sympathetic and parasympathetic blockade on heart rate and cardiac function in man. *Am J Cardiol* 1966; 18: 476-81.
 50. Schwartz PJ, Locati EH, Napolitano C, et al. The long QT syndrome. In: Zipes DP, Jalife J, eds. *Cardiac electrophysiology: from cell to bedside*. Philadelphia, PA: WB Saunders, 1995: 788-811.
 51. Gregoratos G, Cheitlin MD, Conill A, et al. ACC/AHA guidelines for implantation of cardiac pacemakers and antiarrhythmia devices: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Pacemaker Implantation). *J Am Coll Cardiol* 1998; 5: 1175-209.
 52. Zipes DP, DiMarco JP, Gillette PC, et al. Guidelines for clinical intracardiac electrophysiological and catheter ablation procedures. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Clinical Intracardiac Electrophysiology and Catheter Ablation Procedures), developed in collaboration with the North American Society of Pacing and Electrophysiology. *J Am Coll Cardiol* 1995; 26: 555-73.
 53. Delise P, Piccolo E, D'Este D, et al. Electrogenesis of the S1S2S3 electrocardiographic pattern. A study in humans based on body surface potential and right ventricular endocardial mapping. *J Electrocardiol* 1990; 23: 23-31.
 54. Piccolo E, Raviele A, Delise P, et al. The role of left ventricular conduction in the electrogenesis of left ventricular hypertrophy. An electrophysiologic study in man. *Circulation* 1979; 59: 1044-51.
 55. Piccolo E, Delise P, Raviele A, et al. The anterior displacement of the QRS loop as a right ventricular conduction disturbance. Electrophysiologic and vectorcardiographic study in man. *J Electrocardiol* 1980; 13: 267-71.
 56. Piccolo E, Raviele A, Delise P. I ritardi della attivazione ventricolare. In: Piccolo E, ed. *Elettrocardiografia e vettorcardiografia*. Padova: Piccin, 1981: 131-204.
 57. Dhingra R, Palileo E, Strasberg B, et al. Significance of the HV interval in 517 patients with chronic bifascicular block. *Circulation* 1981; 64: 1265-70.
 58. McNulty JH, Rahimtoola SH, Murphy E, et al. Natural history of "high risk" bundle branch block: final report of a prospective study. *N Engl J Med* 1982; 307: 137-43.
 59. Gronda M, Magnani A, Occhetta E, et al. Electrophysiologic study of atrioventricular block and ventricular conduction defects. *G Ital Cardiol* 1984; 14: 768-73.
 60. Brignole M, Menozzi C, Moya A, et al. Mechanism of syncope in patients with bundle branch block and negative electrophysiologic study. *Circulation* 2001; 104: 2045-50.
 61. Corrado D, Thiene G, Nava A, Rossi L, Pennelli N. Sudden death in young competitive athletes: clinico-pathologic correlations in 22 cases. *Am J Med* 1990; 89: 588-96.
 62. 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: 568-75.
 63. 26th Bethesda Conference: Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. January 6-7 1994. *J Am Coll Cardiol* 1994; 24: 845-99.

64. Maron BJ, Pelliccia A, Spirito P. Cardiac disease in young trained athletes. Insights into methods for distinguishing athlete's heart from structural heart disease, with particular emphasis on hypertrophic cardiomyopathy. *Circulation* 1995; 91: 1596-601.
65. Libberthson RR. Sudden death from cardiac causes in children and young adults. *N Engl J Med* 1996; 334: 1039-44.
66. Maron BJ, Shirani J, Pollac LC, et al. Sudden death in young competitive athletes. Clinical, demographic and pathological profiles. *JAMA* 1996; 276: 199-205.
67. Corrado D, Basso C, Schiavon M, Thiene G. Screening for hypertrophic cardiomyopathy in young athletes. *N Engl J Med* 1998; 339: 364-9.
68. Maron BJ. Cardiovascular risks to young persons on the athletic field. *Ann Intern Med* 1998; 129: 379-86.
69. Basso C, Maron BJ, Corrado D, Thiene G. 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: 1493-501.
70. Estes NA III, Link MS, Cannon D, et al. Report of the NASPE policy conference on arrhythmias and the athlete. *J Cardiovasc Electrophysiol* 2001; 12: 1208-19.
71. Sami M, Chaitman B, Fisher L, et al. Significance of exercise-induced ventricular arrhythmia in stable coronary artery disease: a coronary artery surgery project. *Am J Cardiol* 1984; 54: 1182-8.
72. Zipes DP, Cobb LA, Garson A Jr, et al. Cardiovascular abnormalities in the athlete: recommendations regarding eligibility for competition. Task Force VI: Arrhythmias. *J Am Coll Cardiol* 1985; 6: 1225-32.
73. Peduzzi P, Hultgren H, Thomsen J, et al. Prognostic value of baseline exercise tests. *Prog Cardiovasc Dis* 1986; 28: 285-92.
74. Busby MJ, Shefrin EA, Fleg JL. Prevalence and long-term significance of exercise-induced frequent or repetitive ventricular ectopic beats in apparently healthy volunteers. *J Am Coll Cardiol* 1989; 14: 1659-65.
75. Naccarella F, Accorsi A, Palmieri M, et al. Aritmie ventricolari in apparente assenza di patologia cardiovascolare in atleti ed in soggetti praticanti attività sportiva. Aspetti diagnostici e prognostici. *Int J Sports Cardiol* 1994, Atti del VI Congresso Nazionale SIC Sport, Roma 24-26 novembre 1993.
76. Rigo F, Chinellato P, Ragazzo M, Zanella C, Caprioglio F, Delise P. Iter diagnostico e significato prognostico dei battiti prematuri ventricolari nel cuore sano. In: Delise P, ed. Normalità e patologia in Cardiologia dello Sport. Roma: CESI, 1997: 137-43.
77. Schweikert RA, Paskow FJ, Snader C, et al. Association of exercise-induced ventricular ectopic activity with thallium myocardial perfusion and angiographic coronary artery disease in stable, low-risk populations. *Am J Cardiol* 1999; 83: 530-4.
78. Biffi A, Pelliccia A, Verdile L, et al. Long-term clinical significance of frequent and complex ventricular tachyarrhythmias in trained athletes. *J Am Coll Cardiol* 2002; 40: 446-52.
79. Delise P, D'Este D, Bonso A, et al. Utilità dello studio elettrofisiologico transesofageo durante test ergometrico nella valutazione delle tachicardie parossistiche sopraventricolari insorgenti sotto sforzo. *G Ital Cardiol* 1989; 19: 1094-104.
80. Wu D, Denes P, Amat-Y-Leon F, et al. Clinical, electrocardiographic and electrophysiologic observations in patients with paroxysmal supraventricular tachycardia. *Am J Cardiol* 1978; 41: 1045-51.
81. Denes P, Wu D, Dhingra RC, et al. Demonstration of dual AV nodal pathways with paroxysmal supraventricular tachycardias. *Circulation* 1974; 48: 549-58.
82. Sung RJ, Waxman H, Saksena S, Juma Z. Sequence of retrograde atrial activation in patients with dual atrioventricular nodal pathways. *Circulation* 1981; 64: 1059-67.
83. Coumel P. Junctional reciprocating tachycardias. The permanent and paroxysmal forms of AV nodal reciprocating tachycardias. *J Electrocardiol* 1975; 8: 79-86.
84. Haissaguerre M, Jais P, Shah D, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. *N Engl J Med* 1998; 339: 659-66.
85. Goldreyer BN, Kastor JA, Kershbaum KL. The hemodynamic effects of induced supraventricular tachycardia in man. *Circulation* 1976; 54: 783-9.
86. Zilbermann A, Rogel S. Hemodynamic evaluation of common cardiac arrhythmias. *Int J Cardiol* 1990; 27: 341-9.
87. Leitch WJ, Klein GJ, Yee R, et al. Syncope associated with supraventricular tachycardia. *Circulation* 1992; 85: 1064-71.
88. Claiborne TS. Auricular tachycardia with atrioventricular block of 12 years duration in a 16-years-old girl. *Am Heart J* 1950; 39: 444-50.
89. Shachow N, Spellman S, Rubin I. Persistent supraventricular tachycardia: case report with review of literature. *Circulation* 1954; 10: 232-6.
90. Coleman HN, Taylor RR, Pool PE, et al. Congestive heart failure following chronic tachycardia. *Am Heart J* 1971; 81: 790-8.
91. Keane JF, Plauth W, Nadas AS. Chronic ectopic tachycardia of infancy and childhood. *Am Heart J* 1972; 84: 748-57.
92. Engel TR, Bush CA, Shaal SF. Tachycardia-aggravated heart disease. *Ann Intern Med* 1974; 80: 790-8.
93. Gillette PC, Wampler DG, Garso A Jr, et al. Treatment of atrial automatic tachycardia by ablation procedures. *J Am Coll Cardiol* 1985; 6: 405-9.
94. McLaren CJ, Gersh BJ, Sugrue DD, et al. Tachycardia induced myocardial dysfunction: a reversible phenomenon? *Br Heart J* 1985; 53: 323-7.
95. Paker DL, Bardy GH, Worley SJ, et al. Tachycardia-induced cardiomyopathy: a reversible form of left ventricular dysfunction. *Am J Cardiol* 1986; 57: 563-70.
96. Sganzerla P, Fabbiochi F, Grazi S, et al. Electrophysiologic and haemodynamic correlates in supraventricular tachycardia. *Eur J Heart* 1990; 10: 32-40.
97. Gajewski J, Singer RB. Mortality in an insured population with atrial fibrillation. *JAMA* 1981; 245: 1540-4.
98. Kannel WB, Abbott RD, Savage DD, McNamara PM. Epidemiological features of chronic atrial fibrillation. The Framingham Study. *N Engl J Med* 1982; 306: 1018-22.
99. Godtfredsen J. Atrial fibrillation. Etiology, course, and prognosis. A follow-up study of 1212 cases. In: Kulbertus HE, Olsson SB, Schlepper M, eds. Atrial fibrillation. Mölndal: AB-Hässle, 1982: 148-57.
100. Kopecky SL, Gersh BJ, Phil MD, et al. The natural history of idiopathic "lone" atrial fibrillation: a population-based study over three decades. *N Engl J Med* 1987; 317: 669-76.
101. Scardi S, Ceschia G, Pandullo C, Benettoni A, Pivotti F. La storia naturale dei pazienti con fibrillazione atriale isolata. *G Ital Cardiol* 1987; 17: 492-7.
102. Delise P, Bonso A, Allibardi P, et al. Valore clinico e prognostico della valutazione della vulnerabilità atriale con lo studio elettrofisiologico endocavitario e transesofageo. *G Ital Cardiol* 1990; 20: 533-2.
103. Alboni P, Scarfò S, Paparella N, et al. Hemodynamics of induced atrial fibrillation before and after oral sotalol. *New Trends in Arrhythmias* 1992; 8: 489-98.
104. Benjamin EJ, Wolf PA, D'Agostino RB, Silbershatz H, Kannel WB, Levy D. Impact of atrial fibrillation on the risk of death: The Framingham Heart Study. *Circulation* 1998; 98: 946-52.
105. Delise P. La fibrillazione atriale innescata da tachicardia sopraventricolare: una forma apparentemente idiopatica trat-

- tabile con la radiofrequenza. *G Ital Cardiol* 1998; 28: 308-11.
106. Scardi S, Mazzone C, Pandullo C, et al. Lone atrial fibrillation: prognostic differences between paroxysmal and chronic forms after 10 years of follow-up. *Am Heart J* 1999; 137: 686-91.
 107. Hart RG, Pearce LA, Rothbart RM, et al. Stroke with intermittent atrial fibrillation: incidence and predictors during aspirin therapy. *J Am Coll Cardiol* 2000; 35: 183-7.
 108. Sung RJ, Castellanos A, Mallon SM, et al. Mechanisms of spontaneous alternation between reciprocating tachycardia and atrial flutter-fibrillation in the Wolff-Parkinson-White syndrome. *Circulation* 1977; 56: 409-16.
 109. Windham CR, Amat-y-Leon F, Wu D, et al. Effects of cycle length on atrial vulnerability. *Circulation* 1977; 55: 260-7.
 110. Prystowsky EN. Tachycardia-induced tachycardia: a mechanism of initiation of atrial fibrillation. In: Di Marco JP, Prystowsky EN, eds. *Atrial arrhythmias: state of the art*. Armonk, NY: Futura Publishing Company, 1995: 81-95.
 111. Delise P, Gianfranchi L, Paparella N, et al. Clinical usefulness of slow pathway ablation in patients with both paroxysmal atrioventricular nodal reentrant tachycardia and atrial fibrillation. *Am J Cardiol* 1997; 79: 1421-3.
 112. Jais P, Haissaguerre M, Shah DC, et al. A focal source of atrial fibrillation treated by discrete radiofrequency ablation. *Circulation* 1997; 95: 572-6.
 113. Atwood JD, Myeres J, Sullivan M, et al. Maximal exercise testing and gas exchange in patients with chronic atrial fibrillation. *J Am Coll Cardiol* 1988; 11: 508-13.
 114. Fisher B, Haissaguerre M, Garrigues Poquet F, et al. Radiofrequency catheter ablation of common atrial flutter in 80 patients. *J Am Coll Cardiol* 1995; 25: 1365-73.
 115. Lee H, Tai CT, Yu WC, et al. Effects of radiofrequency catheter ablation on quality of life in patients with atrial flutter. *Am J Cardiol* 1999; 84: 278-83.
 116. Anselme F, Saudi MD, Poty MD, et al. Radiofrequency catheter ablation of common atrial flutter. Significance of palpitations and quality-of-life evaluation in patients with proven isthmus block. *Circulation* 1999; 99: 534-40.
 117. Guize L, Soria R, Chaouat JC, Chretien JM, Houe D, Le Heuzey JY. Prevalence et evolution du syndrome de Wolff-Parkinson-White dans une population de 138 048 sujets. *Ann Med Interne* 1985; 136: 474-8.
 118. Giardina AC, Ehlers KH, Engle MA. Wolff-Parkinson-White syndrome in infants and children. *Br Heart J* 1972; 34: 839-46.
 119. Gillette PC, Garson A Jr, McNamara DG. Wolff-Parkinson-White syndrome in children: natural history and clinical spectrum. (abstr) *Circulation* 1980; 62 (Suppl III): III-271.
 120. Perry J, Garson A. Supraventricular tachycardia due to Wolff-Parkinson-White syndrome in children: early disappearance and late occurrence. *J Am Coll Cardiol* 1990; 16: 1215-20.
 121. Losekoot TG, Lubbers LJ. The Wolff-Parkinson-White syndrome in childhood. *Int J Cardiol* 1990; 27: 293-309.
 122. Klein J, Yee R, Sharma A. Longitudinal electrophysiologic assessment of asymptomatic patients with the Wolff-Parkinson-White electrocardiographic pattern. *N Engl J Med* 1989; 320: 1229-33.
 123. Deal B, Keane J, Gillette P, Garson A. Wolff-Parkinson-White syndrome and supraventricular tachycardia during infancy: management and follow-up. *J Am Coll Cardiol* 1985; 5: 130-5.
 124. Becquart J, Vaksman G, Becquart U, et al. Long-term follow up of Wolff-Parkinson-White syndrome discovered in infancy. *Eur Heart J* 1989; 9: 13.
 125. Soria R, Guize L, Chretien JM, et al. L'histoire naturelle de 270 cas de syndrome de Wolff-Parkinson-White dans un'enquete de population generale. *Arch Mal Coeur Vaiss* 1989; 82: 331-6.
 126. Sorbo MD, Buja GF, Miorelli M, et al. Wolff-Parkinson-White syndrome: epidemiological and clinical findings in a large, regional population of young males. *New Trends in Arrhythmias* 1993; 9: 1113-6.
 127. Chiba N, Nakai K, Hotta K, et al. Incidence and clinical significance of Wolff-Parkinson-White pattern in 6 to 18 year-old school children in Iwate Prefecture, Japan. *Eur Heart J* 1996; 17: 404.
 128. Munger TM, Packer DL, Hammill SC, et al. A population study of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953-1989. *Circulation* 1993; 87: 866-73.
 129. Wellens HJ, Durrer D. Wolff-Parkinson-White syndrome and atrial fibrillation. *Am J Cardiol* 1974; 34: 777-83.
 130. Campbell RW, Smith R, Gallagher JJ, Pritchett EL, Wallace AG. Atrial fibrillation in the preexcitation syndrome. *Am J Cardiol* 1977; 40: 514-21.
 131. Bauernfeind RA, Wyndham CR, Swiryn SP, et al. Paroxysmal atrial fibrillation in the WPW syndrome. *Am J Cardiol* 1981; 47: 562-71.
 132. Bromberg BI, Lindsay BD, Cain ME, Cox JL. Impact of clinical history and electrophysiologic characterization of accessory pathways on management strategies to reduce sudden death among children with Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1996; 27: 690-5.
 133. Smith RF. The Wolff-Parkinson-White syndrome as an aviation risk. *Circulation* 1964; 29: 672-9.
 134. Berkman NL, Lamb LE. The Wolff-Parkinson-White electrocardiogram. A follow up study of five to twenty-eight years. *N Engl J Med* 1968; 278: 492-4.
 135. Flensted-Jensen E. Wolff-Parkinson-White syndrome: a long-term follow up of 47 cases. *Acta Med Scand* 1969; 186: 65-74.
 136. Brembilla-Perrot B, Aliot E, Louis P, et al. Devenir de 195 patients atteints de syndrome de Wolff-Parkinson-White. *Arch Mal Coeur Vaiss* 1987; 80: 271-7.
 137. Klein JG, Bashore TM, Sellers RD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White Syndrome. *N Engl J Med* 1979; 301: 1080-9.
 138. Montoya PT. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *Circulation* 1988; 78 (II): 23.
 139. Basso C, Corrado D, Rossi L, Thiene G. Ventricular preexcitation in children and young adults. Atrial myocarditis as a possible trigger of sudden death. *Circulation* 2001; 103: 269-75.
 140. Morady F, Sledge C, Shen E, Sung RJ, Gonzales R, Scheinman MM. Electrophysiologic testing in the management of patients with the Wolff-Parkinson-White syndrome and atrial fibrillation. *Am J Cardiol* 1983; 51: 1623-9.
 141. Critelli G, Gallagher JJ, Peticone F, Coltorti F, Monda V, Condorelli M. Evaluation of noninvasive tests for identifying patients with preexcitation syndrome at risk of rapid ventricular response. *Am Heart J* 1984; 108: 905-12.
 142. Delise P, D'Este D, Raviele A, et al. Electrophysiologic evaluation of atrial vulnerability in patients with ventricular preexcitation. *New Trends in Arrhythmias* 1985; 1: 311-5.
 143. Milstein S, Sharma AD, Klein GJ. Electrophysiologic profile of asymptomatic Wolff-Parkinson-White pattern. *Am J Cardiol* 1986; 57: 1097-103.
 144. Vergara G, Furlanello F, Disertori M, et al. La valutazione elettrofisiologica degli atleti con Wolff-Parkinson-White: induzione di tachiaritmia sopraventricolare di base e sotto sforzo con elettrostimolazione atriale transesofagea. *G Ital Cardiol* 1986; 16: 625-30.
 145. Delise P, D'Este D, Raviele A, et al. Electrophysiologic

- evaluation of asymptomatic athletes with ventricular pre-excitation. *Int J Sports Cardiol* 1986; 3: 46-50.
146. Delise P, D'Este D, Raviele A, et al. Electrophysiologic profile of the WPW pattern in asymptomatic athletes versus symptomatic patients. Evidence for a different risk to develop life-threatening atrial fibrillation. In: *Proceedings of the Second International Conference on Sports Cardiology*. Sorrento, 2-5 aprile 1987: 117-120.
 147. Sharma AD, Yee R, Guiraudon G, Klewin GJ. Sensitivity and specificity of invasive and noninvasive testing for risk of sudden death in Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1987; 10: 373-81.
 148. Milstein S, Sharma AD, Guiraudon GM, Klein GJ. An algorithm for the electrocardiographic localization of accessory pathways in the Wolff-Parkinson-White syndrome. *Pacing Clin Electrophysiol* 1987; 10: 555-63.
 149. Delise P, D'Este D, Bonso A, et al. Diverso rischio di fibrillazione atriale ad alta frequenza nel WPW sintomatico e asintomatico. Valutazione elettrofisiologica. *G Ital Cardiol* 1987; 17: 127-35.
 150. Vergara G, Furlanello F, Disertori M, et al. Induction of supraventricular tachyarrhythmias at rest and during exercise with transesophageal atrial pacing in the electrophysiological evaluation of asymptomatic athletes with Wolff-Parkinson-White syndrome. *Eur Heart J* 1988; 9: 1119-25.
 151. Packer DL, Pressley JC, German LD, Prystowsky EN. Accuracy of invasive testing for direct identification of sudden death risk in the Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1988; 11: 78A-82A.
 152. Delise P, D'Este D, Bonso A, et al. Clinical usefulness of electrophysiologic evaluation of atrial vulnerability. *New Trends in Arrhythmias* 1988; 4: 559-63.
 153. Delise P, Bonso A, D'Este D, et al. Prognostic value of the electrophysiologic evaluation of atrial vulnerability in the WPW syndrome. *Eur Heart J* 1989; 10: 397.
 154. Vergara G, Delise P. Dibattito sulla sindrome di Wolff-Parkinson-White. *G Ital Cardiol* 1989; 19: 234-44.
 155. Gaita F, Giustetto C, Riccardi R, Mangiardi L, Brusca A. Induzione di tachiaritmie atriali in pazienti con Wolff-Parkinson-White con e senza fibrillazione atriale spontanea. *G Ital Cardiol* 1990; 20: 109-13.
 156. Beckman KJ, Gallastegui JL, Bauman JL, Hariman RJ. The predictive value of electrophysiologic studies in untreated patients with Wolff-Parkinson-White syndrome. *J Am Coll Cardiol* 1990; 15: 640-7.
 157. Delise P. La sindrome di Wolff-Parkinson-White a rischio. *Cardiologia* 1991; 36 (Suppl 8): 81-5.
 158. Bertaglia M, Delise P, D'Este D, et al. Valore prognostico dello studio elettrofisiologico endocavitario e transesofageo nella sindrome di WPW. *G Ital Cardiol* 1993; 23 (Suppl 1): 195.
 159. Brembilla-Perrot B, Ghawi R. Electrophysiological characteristics of asymptomatic Wolff-Parkinson-White syndrome. *Eur Heart J* 1993; 14: 511-5.
 160. Klein GJ, Guiraudon GM, Kerr CR, et al. "Nodoventricular" accessory pathway: evidence for a distinct accessory atrioventricular pathway with atrioventricular node-like properties. *J Am Coll Cardiol* 1988; 11: 1035-40.
 161. Fujiki A, Yoshida S, Tani M, Sasayama S. Electrophysiologic differences between nodoventricular (Mahaim) fiber and atrioventricular accessory fiber with decremental properties. *Am Heart J* 1989; 118: 1057-62.
 162. Gallagher JJ, Smith WM, Kasell JH, Benson DW Jr, Sterba R, Grant AO. Role of Mahaim fibers in cardiac arrhythmias in man. *Circulation* 1981; 64: 176-89.
 163. Haïssaguerre M, Warin JF, Le Metayer P, et al. Catheter ablation of Mahaim fibers with preservation of atrioventricular nodal conduction. *Circulation* 1990; 82: 418-27.
 164. Kinder C, Tamburro P, Kopp D, et al. The clinical significance of nonsustained ventricular tachycardia: current perspective. *Pacing Clin Electrophysiol* 1994; 17: 637-64.
 165. Bigger JT, Fleiss JL, Kleiger R, et al. The relationship among ventricular arrhythmias, left ventricular dysfunction and mortality in the 2 years after myocardial infarction. *Circulation* 1984; 69: 250-8.
 166. Olshausen KV, Stienen U, Schwartz F, et al. Long-term prognostic significance of ventricular arrhythmias in idiopathic dilated cardiomyopathy. *Am J Cardiol* 1988; 61: 146-51.
 167. Teerlink JR, Jalaluddin M, Anderson S, et al. Ambulatory ventricular arrhythmias in patients with heart failure do not specifically predict an increased risk of sudden death. PROMISE (Prospective Randomized Milrinone Survival Evaluation) Investigators. *Circulation* 2000; 101: 40-6.
 168. Wilber DJ, Olshansky B, Moran JF, Scanlon PJ. Electrophysiologic testing and nonsustained ventricular tachycardia. Use and limitations in patients with coronary artery disease and impaired ventricular function. *Circulation* 1990; 82: 350-8.
 169. Eisenberg SJ, Scheinman MM, Dullet NK, et al. Sudden cardiac death and polymorphous ventricular tachycardia in patients with normal QT intervals and normal systolic cardiac function. *Am J Cardiol* 1995; 75: 687-92.
 170. Buxton AE, Lee KL, DiCarlo L, et al. Electrophysiologic testing to identify patients with coronary artery disease who are at risk for sudden death. Multicenter Unsustained Tachycardia Trial Investigators. *N Engl J Med* 2000; 342: 1937-45.
 171. Maron BJ, Shen W-K, 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; 342: 365-73.
 172. Delise P, D'Este D, Monico E, et al. Ritmo idioventricolare accelerato con dissociazione AV isoritmica. Studio elettrofisiologico. *G Ital Cardiol* 1980; 10: 1588-92.
 173. Belhassen B, Rotmensh H, Laniado S. Response of recurrent sustained ventricular tachycardia to verapamil. *Br Heart J* 1981; 46: 679-81.
 174. Lin F, Finley D, Rahimtoola J, Wu D. Idiopathic paroxysmal ventricular tachycardia with a QRS pattern of right bundle branch block and left axis deviation: a unique clinical entity with specific properties. *Am J Cardiol* 1983; 52: 95-8.
 175. Delise P, Raviele A, Di Pede F, et al. Tachicardia ventricolare idiopatica sensibile al verapamil. Presentazione di tre casi e revisione della letteratura. *G Ital Cardiol* 1985; 15: 54-9.
 176. Morady F, Kadish AH, Dicarlo L, et al. Long-term results of catheter ablation of idiopathic right ventricular tachycardia. *Circulation* 1990; 82: 2093-9.
 177. Mosvowitz C, Schwartzman D, Callans DJ, et al. Idiopathic right ventricular outflow tract tachycardia: narrowing the anatomical location for successful ablation. *Am Heart J* 1996; 131: 930-6.
 178. Lerman BB, Stein KM, Markowitz SM. Idiopathic right ventricular outflow tract tachycardia: a clinical approach. *Pacing Clin Electrophysiol* 1996; 19: 2120-37.
 179. Myerburg RJ, Kessler KM, Ester D, et al. Long-term survival after prehospital cardiac arrest: analysis of outcome during an 8 year study. *Circulation* 1984; 70: 538-46.
 180. Saxon L, Uretz EF, Denes P. Significance of the clinical presentation in ventricular tachycardia/fibrillation. *Am Heart J* 1989; 118: 695-9.
 181. Brugada P, Talajic M, Smeets J, Mulleneers R, Wellens HJ. The value of the clinical history to assess prognosis of patients with ventricular tachycardia or ventricular fibrillation after myocardial infarction. *Eur Heart J* 1989; 10: 747-52.
 182. Leenhardt A, Lucet V, Denjoy I, et al. Catecholaminergic polymorphic ventricular tachycardia in children. A 7-year follow-up of 21 patients. *Circulation* 1995; 91: 1512-9.

183. Campbell RW, Godman MG, Fiddler GI, et al. Ventricular arrhythmias in syndrome of balloon deformity of mitral valve. Definition of possible high risk group. *Br Heart J* 1976; 38: 1053-7.
184. Boudoulas H, Schaal SF, Stang KM, et al. Mitral valve prolapse: cardiac arrest with long-term survival. *Int J Cardiol* 1990; 26: 37-44.
185. Vohra J, Sathé S, Warren R, Tatoulis J, Hunt D. Malignant ventricular arrhythmias in patients with mitral valve prolapse and mild mitral regurgitation. *Pacing Clin Electrophysiol* 1993; 16: 387-93.
186. Babuty D, Cosnay P, Breuillac JC, et al. Ventricular arrhythmia factors in mitral valve prolapse. *Pacing Clin Electrophysiol* 1994; 17: 1090-9.
187. Kulan K, Komsuoglu B, Tuncer C, Kulan C. Significance of QT dispersion on ventricular arrhythmias in mitral valve prolapse. *Int J Cardiol* 1996; 54: 251-7.
188. Corrado D, Basso C, Nava A, et al. Sudden death in young people with apparently isolated mitral valve prolapse. *G Ital Cardiol* 1997; 27: 101-16.
189. Freed LA, Levy D, Levine RA, et al. Prevalence and clinical outcome of mitral-valve prolapse. *N Engl J Med* 1999; 341: 1-7.
190. Martini B, Nava A, Thiene G, et al. Ventricular fibrillation without apparent heart disease: description of six cases. *Am Heart J* 1989; 118: 1203-9.
191. Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrocardiographic syndrome. *J Am Coll Cardiol* 1992; 20: 1391-6.
192. Proclemer A, Facchin D, Feruglio A, Nucifora R. Fibrillazione ventricolare recidivante, blocco di branca destra, persistente sopraslivellamento del tratto ST in V₁-V₃: una nuova sindrome aritmica? *G Ital Cardiol* 1993; 23: 1211-8.
193. Bjerregaard P, Gussak I, Kotar SL, et al. Recurrent syncope in a patient with a prominent J wave. *Am Heart J* 1994; 127: 1426-30.
194. Miyazaki H, Mitamura H, Miyoshi S, et al. Autonomic and antiarrhythmic drug modulation of ST segment elevation in patients with Brugada syndrome. *J Am Coll Cardiol* 1996; 27: 1061-70.
195. Nademanee K, Veerakul G, Nimmanit S, et al. Arrhythmogenic marker for the sudden unexplained death syndrome in Thai Men. *Circulation* 1997; 96: 2595-600.
196. Brugada J, Brugada R, Brugada P. Right bundle-branch block and ST-segment elevation in leads V₁ through V₃. A marker for sudden death in patients without demonstrable structural heart disease. *Circulation* 1998; 97: 457-60.
197. Roden DM, Wilde AM. Drug-induced J point elevation: a marker for genetic risk of sudden death or ECG curiosity? *J Cardiovasc Electrophysiol* 1999; 10: 219-21.
198. Fujiki A, Usui M, Nagasawa H, Mizumaki K, Hayashi H, Inoue H. ST segment elevation in the right precordial leads induced with class IC antiarrhythmic drugs: insight into the mechanisms of Brugada syndrome. *J Cardiovasc Electrophysiol* 1999; 10: 214-8.
199. Fujimori K, Soda M, Byun T, et al. Incidence of asymptomatic Brugada syndrome among middle to high-aged subjects: an exhaustive investigation of local residents in Japan. *Circulation* 2000; 102 (Suppl II): II-676.
200. Miyasaka Y, Yamada K, Sugiura T, et al. Prevalence and mortality of right bundle-branch block and right precordial ST-segment elevation (Brugada type ECG) in a general population. *Circulation* 2000; 102 (Suppl II): II-676.
201. Weiss R, Nguyen T, Shusterman V, et al. Long term follow-up in a large family with RBBB and ST-segment elevation not linked to SCN5A suggests a less malignant phenotype. *Circulation* 2000; 102 (Suppl II): II-676.
202. Bianco M, Bria S, Gianfelici A, Sanna N, Palmieri V, Zeppilli P. Does early repolarization in the athlete have analogies with the Brugada syndrome. *Eur Heart J* 2001; 22: 504-10.
203. Corrado D, Zoppo F, Buja G, et al. Programmed ventricular stimulation and clinical outcome in symptomatic and asymptomatic patients with Brugada syndrome. *Pacing Clin Electrophysiol* 2001; 24: 558.
204. Corrado D, Basso C, Buja G, Nava A, Rossi L, Thiene G. Right bundle branch block, right precordial ST-segment elevation and sudden death in young people. *Circulation* 2001; 103: 710-7.
205. Brugada P, Geelen P, Brugada R, Mont L, Brugada J. Prognostic value of electrophysiologic investigations in Brugada syndrome. *J Cardiovasc Electrophysiol* 2001; 12: 1004-7.
206. Priori SG, Napolitano C, Gasparini M, et al. Natural history of Brugada syndrome: insights for risk stratification and management. *Circulation* 2002; 105: 1342-7.
207. Gasparini M, Priori S, Mantica M, et al. Programmed electrical stimulation in Brugada syndrome: how reproducible are the results? *J Cardiovasc Electrophysiol* 2002; 13: 880-7.
208. Kanda M, Shimizu W, Matsuo K, et al. Electrophysiologic characteristics and implications of induced ventricular fibrillation in symptomatic patients with Brugada syndrome. *J Am Coll Cardiol* 2002; 39: 1799-805.
209. Antzelevitch C, Brugada P, Brugada J, et al. Brugada syndrome: 1992-2002. A historical perspective. *J Am Coll Cardiol* 2003; 41: 1665-71.
210. Brugada P, Brugada R, Mont L, Rivero M, Geelen P, Brugada J. Natural history of Brugada syndrome: the prognostic value of programmed electrical stimulation of the heart. *J Cardiovasc Electrophysiol* 2003; 14: 455-7.
211. Marcus FI, Fontaine G, Guiraudon G, et al. Right ventricular dysplasia: a report of 24 cases. *Circulation* 1982; 65: 384-98.
212. Laurent M, Descaves C, Biron Y, et al. Familial form of arrhythmogenic right ventricular dysplasia. *Am Heart J* 1987; 113: 827-9.
213. Blomstrom-Lundqvist C, Sabel KG, Olsson SB. A long-term follow-up of 15 patients with arrhythmogenic right ventricular dysplasia. *Eur Heart J* 1987; 58: 477-88.
214. Nava A, Thiene G, Canciani B, et al. Familial occurrence of right ventricular dysplasia: a study involving nine families. *J Am Coll Cardiol* 1988; 12: 1222-8.
215. McKenna WJ, Thiene G, Nava A, et al. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. Task Force of the Working Group Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology. *Br Heart J* 1994; 71: 215-8.
216. Corrado D, Basso C, Thiene G, et al. Spectrum of clinicopathologic manifestations of arrhythmogenic right ventricular cardiomyopathy/dysplasia: a multicenter study. *J Am Coll Cardiol* 1997; 30: 1512-20.
217. Nava A, Bauce B, Basso C, et al. Clinical profile and long-term follow-up of 37 families with arrhythmogenic right ventricular cardiomyopathy. *J Am Coll Cardiol* 2000; 36: 2226-33.
218. Bauce B, Nava A, Rampazzo A, et al. Familial effort polymorphic ventricular arrhythmias in arrhythmogenic right ventricular cardiomyopathy map to chromosome 1q42-43. *Am J Cardiol* 2000; 95: 573-9.
219. Jervell A, Lange-Nielsen F. Congenital deaf mutism, functional heart disease with prolongation of the QT interval and sudden death. *Am Heart J* 1957; 54: 59-68.
220. Romano C, Gemme G, Pongiglione R. Aritmie cardiache rare dell'età pediatrica. II. Accessi sincopali per fibrillazione ventricolare parossistica. *Clin Pediatr (Bologna)* 1963; 45: 656-83.

221. Ward OC. A new familial cardiac syndrome in children. *J Ir Med Assoc* 1964; 54: 103-6.
222. Schwartz PJ. Idiopathic long QT syndrome: progress and questions. *Am Heart J* 1985; 109: 399-411.
223. Moss AG. Prolonged QT-interval syndrome. *JAMA* 1986; 256: 2985-7.
224. Rosen MR. Long QT syndrome patients with gene mutations. *Circulation* 1995; 92: 3373-5.
225. Wang Q, Shen J, Atkinson D, et al. SCN5A mutations associated with an inherited cardiac arrhythmia, long QT syndrome. *Cell* 1995; 80: 805-11.
226. Sanguinetti MC, Jiang C, Curran ME, Keating MT. A mechanistic link between an inherited and an acquired cardiac arrhythmia: HERG encodes the IKr potassium channel. *Cell* 1995; 81: 299-307.
227. Duggal P, Vesely MR, Wattanasirichaigoon D, Villafane J, Kaushik V, Beggs AH. Mutation of the gene for IsK associated with both Jervell and Lange-Nielsen and Romano-Ward forms of long-QT syndromes. *Circulation* 1998; 97: 142-6.
228. Borggrefe M, Budde KP, Podczeczek A, Breithardt G. High frequency alternating current ablation of an accessory pathway in humans. *J Am Coll Cardiol* 1987; 10: 576-82.
229. Kuck KH, Kunze KP, Schluter M, et al. Ablation of a left-sided free-wall accessory pathway by percutaneous catheter application of radiofrequency current in a patient with the Wolff-Parkinson-White syndrome. *Pacing Clin Electrophysiol* 1989; 12: 1681.
230. Delise P, Bonso A, Raviele A, et al. Tachicardia reciprocante idionodale insensibile alla profilassi farmacologica. Descrizione di un caso trattato con successo con la radiofrequenza. *G Ital Cardiol* 1990; 20: 1168-73.
231. Delise P, Bonso A, Raviele A, et al. Successful catheter ablation by radiofrequency energy of posteroseptal accessory pathway in a patient with drug refractory Wolff-Parkinson-White syndrome. *Eur Heart J* 1991; 12: 1321-5.
232. Lee M, Morady F, Kadish A, et al. Catheter modification of the atrioventricular junction with radiofrequency energy for control of atrioventricular nodal reentry tachycardia. *Circulation* 1991; 83: 827-35.
233. Jackman WM, Wang X, Wang X, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. *N Engl J Med* 1991; 324: 1605-12.
234. Jackman W, Beckman K, McClelland J, et al. Treatment of supraventricular tachycardia due to atrioventricular nodal reentry by radiofrequency catheter ablation of slow-pathway conduction. *N Engl J Med* 1992; 327: 313-8.
235. Lesh MD, Van Hare GF, Schamp DJ, et al. Curative percutaneous catheter ablation using radiofrequency energy for accessory pathways in all locations: results in 100 consecutive patients. *J Am Coll Cardiol* 1992; 19: 1303-10.
236. De Ponti R, Casari A, Salerno JA, et al. Ablazione transcatetere mediante radiofrequenza delle vie anomale atrioventricolari sinistre: ruolo dell'approccio transettale. *G Ital Cardiol* 1992; 22: 1255-61.
237. Scheinman MM. Catheter ablation for cardiac arrhythmias, personnel, and facilities. North American Society of Pacing and Electrophysiology Ad Hoc Committee on Catheter Ablation. *Pacing Clin Electrophysiol* 1992; 15: 715-21.
238. Haissaguerre M, Gaita F, Fischer B, et al. Elimination of AV node reentrant tachycardia using slow potentials to guide application of radiofrequency energy. *Circulation* 1992; 85: 2162-70.
239. Delise P, Bonso A, Corò L, Piccolo E. L'ablazione con radiofrequenza della via anomala. Una possibile terapia risolutiva per l'atleta con Wolff-Parkinson-White. *Int J Sports Cardiol* 1993; 2: 19-25.
240. Mitrani R, Klein L, Hackett K, Zipes DP, Miles WM. Radiofrequency ablation for atrioventricular node reentrant tachycardia: comparison between fast (anterior) and slow (posterior) pathway ablation. *J Am Coll Cardiol* 1993; 21: 432-41.
241. Cosio FG, Lopez-Gil M, Goicolea A, et al. Radiofrequency ablation of the inferior vena cava-tricuspid valve isthmus in common atrial flutter. *Am J Cardiol* 1993; 71: 705-10.
242. Kay NG, Chong F, Epstein AE, et al. Radiofrequency ablation for treatment of primary atrial tachycardias. *J Am Coll Cardiol* 1993; 21: 901-8.
243. Goldberger J, Kall J, Ehlert F, et al. Effectiveness of radiofrequency catheter ablation for treatment of atrial tachycardia. *Am J Cardiol* 1993; 72: 787-92.
244. Hindricks G. The Multicentre European Radiofrequency Survey (MERFS): complications of radiofrequency catheter ablation of arrhythmias. The Multicentre European Radiofrequency Survey (MERFS) Investigators of the Working Group on Arrhythmias of the European Society of Cardiology. *Eur Heart J* 1993; 14: 1644-53.
245. Scheinman M. NASPE survey on catheter ablation. *Pacing Clin Electrophysiol* 1995; 18: 1474-8.
246. Delise P, Bonso A, Corò L, et al. How to predict and avoid complete AV block complicating radiofrequency ablation of AV nodal slow pathway. In: Raviele A, ed. *Cardiac arrhythmias 1999*. Milano: Springer-Verlag Italia, 2000: 176-84.
247. Pappone C, Oreto G, Lamberti F, et al. Catheter ablation of paroxysmal atrial fibrillation using a 3D mapping system. *Circulation* 1999; 100: 1203-8.
248. Haissaguerre M, Jais P, Shah D, et al. Electrophysiological endpoint for catheter ablation of atrial fibrillation initiated from multiple pulmonary venous foci. *Circulation* 2000; 101: 1409-17.
249. Pappone C, Rosanio S, Oreto G, et al. Circumferential radiofrequency ablation of pulmonary vein ostia. A new anatomic approach for curing atrial fibrillation. *Circulation* 2000; 102: 2619-28.
250. Pappone C, Oreto G, Rosanio S, et al. Atrial electroanatomic remodeling after circumferential radiofrequency pulmonary vein ablation. Efficacy of an anatomic approach in a large cohort of patients with atrial fibrillation. *Circulation* 2001; 104: 2539-44.
251. Delise P, Bonso A, Mantovan R, et al. Ruolo della metodica transesofagea nella valutazione dei pazienti sottoposti ad ablazione transcatetere. In: Disertori M, Marconi P, eds. *La stimolazione cardiaca transesofagea e l'elettrocardiografia ad alta risoluzione*. 1993: 215-25.

CONGENITAL AND ACQUIRED VALVULAR HEART DISEASES

Congenital heart diseases

Preface. Sports activity may be beneficial at all ages: it enhances physical fitness and strength, contributes to keeping body weight within the normal range, and improves the flexibility and function of the osteo-articular and muscle systems. It may also lead to remarkable improvements in psychological conditions, i.e. in the quality of life.

These aspects are of paramount importance in the young age, when sport plays a role in psychological and physical growth and justify the growing requests to allow sports activity to children and teenagers with

heart disease. This issue has become ever stronger thanks to the recent diagnostic and therapeutic advances, especially in surgery and interventional transcatheter techniques, granting a return to active life to a growing number of young patients, who would be formerly kept off the athletic fields.

The Italian law, however, contemplates a mandatory pre-participation visit to assess eligibility to competitive or recreational sports. Such an eligibility implies a specific responsibility of sports physicians who should advise on each necessary clinical and instrumental examination to evaluate the disease severity, the residual functional capacity, and whether a specific heart disease may be compatible with a given sport.

However, this issue is rather complex: the congenital heart disease population is not homogeneous, not only as for the type of cardiac anomaly, but also because subjects with the same congenital disorder can be either on “natural history” or might have undergone surgical correction. In addition, the availability of different surgical procedural approaches yields a wide range of results. This strongly calls for a close cooperation between sports physicians and pediatric cardiologists, especially in the management of the most complicated cases.

Although it is impossible to establish general guidelines to be applied to each single case, some diseases contraindicate *per se* competitive sports due to their severity and/or complexity. This group of diseases includes:

- Ebstein’s anomaly;
- tricuspid valve atresia;
- right ventricular outflow obstruction, with either a normal septum or an interventricular septal defect (without a complete recovery of the right ventricular function);
- Eisenmenger syndrome;
- idiopathic pulmonary hypertension;
- congenitally corrected transposition of the great vessels;
- transposition of the great vessels, corrected by the Mustard or Senning procedure;
- associated defects of the left ventricular outflow;
- anomalous origin of the coronary arteries;
- univentricular heart;
- Marfan syndrome;
- Ehlers-Danlos syndrome.

This list should also include all cardiac anomalies whose surgical correction requires the insertion of prosthetic ducts and/or valves, except for what stated in each single chapter.

Patent ductus arteriosus. Hemodynamically non-significant patent ductus arteriosus (PDA) (Botallo’s) without hemodynamic impairment is characterized by a mild continuous murmur, best heard right below the left clavicle, with normal radiological and echocardiographic diameters, and normal ECG.

In addition to the typical heart murmur, hemodynamically significant PDA is characterized by signs of left-sided, or combined, enlargement on ECG, X-ray and ECHO and an increased pulmonary perfusion, either with or without pulmonary hypertension. In this case early correction is required.

ECHO-Doppler is useful to make the proper diagnostic and prognostic evaluation (indirect assessment of the pulmonary BP and of the pulmonary flow to systemic flow ratio: Qp/Qs)¹. Usually, cardiac catheterization is not necessary, unless the entity of hemodynamic involvement or the presence of other cardiac diseases (coronary or artero-venous fistulae, etc.) has to be cleared up.

Indications. PDA without hemodynamic impairment does not represent a contraindication to sports activity of any type, except for scuba diving (potential risk of paradoxical embolization).

The types of PDA corrected by traditional surgery or transcatheter procedures², at least 6 months post-intervention, must be re-evaluated with ECG, ECHO color Doppler and EST.

If the clinical and laboratory findings demonstrate a real normalization of ventricular involvement, no pulmonary hypertension, and a normal performance, eligibility can be granted to all sports activities.

Atrial septal defects. *Ostium secundum or sinus venosus.* Atrial septal defects (ASD), type ostium secundum or venous sinus, are one of the most common congenital cardiac anomalies in young athletes. They may be suspected after a basal ejection murmur, associated with a wide split, fixed or near fixed S2 (the enhanced pulmonary component of S2 may indicate pulmonary hypertension). Nowadays, ECG, chest X-ray and, most of all, ECHO-Doppler and ECHO color Doppler can be used for diagnostic validation and for the assessment of ASD hemodynamics.

Hemodynamically non-significant ASD is characterized by a “scarce” clinical and laboratory picture, normal right ventricular dimensions and normal function of the interventricular septum at ECHO.

In hemodynamically significant ASD, correction is mandatory as early as possible, and can be performed surgically or, whenever allowed by the dimensions and position of the defect, by means of interventional transcatheter techniques (device implantation).

Due to the frequent association between ASD and arrhythmias, especially supraventricular^{3,4}, EST and HM are recommended comprehensive of a training session.

Indications. Subjects with hemodynamically non-significant ASD can participate in sports of any type, except for scuba diving (risk of paradoxical embolization)⁵⁻⁸. Apnea diving, on the contrary, is allowed.

In ASD corrected by surgery or interventional techniques⁹, a second evaluation by ECG, ECHO color

Doppler, EST, HM is required 6 months post-operatively. Sports eligibility cannot be granted in case of:

- residual pulmonary hypertension;
- paroxysmal supraventricular tachyarrhythmias, persistent and permanent, or symptomatic sinoatrial dysfunction³ (see Arrhythmias section);
- minimal residual shunt (contraindicating scuba diving)*.

Ostium primum. The diagnosis of ASD type ostium primum is made on the basis of typical clinical or laboratory (ECG, ECHO Doppler, chest X-ray) signs of right ventricular volume overload and increased pulmonary perfusion, either associated with leftward QRS complex axis and/or signs of mitral regurgitation.

The diagnostic and prognostic evaluation requires great attention, and an accurate instrumental testing is always necessary before stating the admission to sports. Moreover, except for minor forms quite uncommon, this cardiac anomaly has to be corrected surgically^{12,13}.

Indications. In the rare cases of isolated and restricted ASD, without cardiac function anomalies, once performed ECG, ECHO color Doppler, EST and HM, eligibility can be granted to sports of minimum to moderate cardiovascular involvement, such as those of group B2, to some of group B1 (horse riding, sailing) and to non-competitive athletic activities (group A).

Six months post-operatively, the subject should be re-evaluated by means of ECG, ECHO color Doppler, EST and HM comprehensive of a training session.

Following the complete regression of hemodynamic dysfunction and residual blood flow and conduction defects are no longer present, eligibility can be considered to all sports after a close individualized examination.

Anomalous pulmonary venous return. Anomalous pulmonary venous return (PVR) can involve some or all pulmonary veins.

Partial anomalous PVR can be isolated or associated with ASD whose clinical picture is similar. In case of hemodynamically non-significant anomalous PVR (usually limited to one vessel), eligibility can be granted to all sports.

Complete anomalous PVR or hemodynamically significant anomalous PVR requires early surgical correction. Subjects with corrected anomalous PVR can be granted eligibility to all sports, provided the following:

- no residual pulmonary hypertension;
- normal exercise tolerance;
- no significant brady and/or tachyarrhythmias resulting at EST and HM comprehensive of a training session.

Atrioventricular septal defects. Atrioventricular septal defects (AVSD) are characterized by a missing or reduced AV septum, namely of that wall made of a anterior fibrous portion and a posterior muscle-like part, separating, in the normal heart, the right atrium from the left ventricular outflow. The classification of different types of AVSD is based on the number of AV valvular orifices, and on the relation between the AV valvular leaflets and the septal structures of the atria and ventricles^{13,14}. The different types are as follows:

- incomplete;
- intermediate;
- complete.

Incomplete AVSD are characterized by two separate AV valves with a cleft in the anterior left "mitral" leaflet, by an ASD type ostium primum of variable size and situated in the lower part of interatrial septum, and by an abnormal interventricular septum, without interventricular communication.

Intermediate AVSD, differently from the incomplete type, are characterized by one or more interventricular defects of small size and located within the chordae tendineae anchored on the crest of the interventricular septum.

Complete AVSD are characterized by a single AV valve, usually made of five leaflets, along with ASD type ostium primum and a large interventricular defect.

Indications. Sports eligibility can be considered only after surgical correction and is limited to selected cases with optimal outcome. Eligibility can be granted to competitive sports with minimum to moderate cardiovascular involvement, such as those of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A, provided that the post-surgical evaluation shows:

- absence of atrial septal and/or interventricular defects (or, if any, they should be negligible);
- no pulmonary hypertension;
- no left ventricular dilation or hypokinesia;
- no or negligible mitral valve regurgitation;
- normal exercise tolerance;
- no significant brady and/or tachyarrhythmias and conduction abnormalities at EST and HM comprehensive of a training session.

Ventricular septal defects. Usually two groups of ventricular septal defects (VSD) can be identified:

- hemodynamically non-significant VSD type Roger: they are easy to detect because of the typical harsh holosystolic murmur, radiating toward the precordium (the aphorism "much ado for nothing" is valid). To the purpose of clinical and functional evaluation, besides ECHO color Doppler which can document even the smallest VSD, especially muscular, EST is sufficient;
- hemodynamically significant VSD: once evaluated by ECHO color Doppler or cardiac catheterization, they must be corrected surgically.

* The occurrence of complete RBBB post-operatively is not to be considered as a contraindication to sports eligibility¹⁰. When eligibility is requested for scuba diving with equipment, contrast transesophageal ECHO color Doppler should be performed once the correction has been performed¹¹.

Indications. Hemodynamically non-significant VSD type Roger (not associated with other cardiac anomalies) do not contraindicate any sports, except for scuba diving (potential risk of paradoxical embolization). Especially in children, such a defect may close spontaneously¹⁵⁻¹⁷.

Corrected VSD, 6 months post-intervention, can be re-evaluated by means of ECG, ECHO color Doppler, EST, and HM comprehensive of a training session. Sports eligibility cannot be granted in case of:

- hemodynamically significant residual VSD;
- persistent pulmonary hypertension;
- ECHO showing abnormal left ventricular dimensions and function. This may occur when the VSD has been closed with ample patches and/or the intervention has been performed by ventriculotomy (right or left);
- no significant brady and/or tachyarrhythmias at EST and/or HM comprehensive of a training session.

In cases with optimal outcome, when the diagnostic tests show the full *restitutio ad integrum* and no arrhythmias, eligibility can be granted to all sports.

Aortic coarctation. Aortic coarctation (ACo) is characterized by a narrowing in the aortic arch before or after the ductal region (duct of Botallo), resulting in an obstruction to the blood flow. It causes proximal hypertension (head and upper limbs) and hypotension and hypoperfusion in the distal regions (abdomen, kidneys, lower limbs). This paragraph will deal with the “simple” or isolated form without associated defects, which, however, are common and should be ruled out accurately (bicuspid aortic valve, VSD, etc.). In particular, bicuspid aortic valve is considered as a risk factor possibly leading to aneurysmal dilation of the ascending aorta. The suspect of ACo must be raised whenever a young patient presents with:

- high BP values, mainly systolic;
- weak or absent femoral pulse;
- systolic murmur, best heard in the interscapular region of the back.

The diagnosis of ACo should be confirmed by the demonstration of a pressure gradient between the two regions, and/or by the direct visualization of the anatomic defect. Nowadays, this goal can be achieved non-invasively by means of ECHO color Doppler^{18,19}. Magnetic resonance imaging is of considerable value in assessing aortic morphology and function²⁰. Cardiac catheterization should be restricted to those patients requiring surgical correction, or to those who may undergo correction by means of transcatheter angioplasty^{21,22}.

An important aspect is represented by BP variations during maximal exercise. When exceeding the normal values according to age, even if the ACo is apparently mild, BP may contraindicate sports eligibility.

Indications. The hemodynamically non-significant forms of ACo are characterized by a mean pressure gradient < 10-15 mmHg at ECHO color Doppler (without

diastolic flow), a normal or mildly elevated brachial BP, weak femoral pulse of femoral pulse, without collateral arteries and significant left ventricular hypertrophy (ECG and ECHO). These subjects can be granted eligibility to sports of minimum to moderate involvement (group B, and non-competitive athletic activities of group A). Group C sports (with a “pressure” cardiovascular involvement) are excluded. As for sports of groups D and E, owing to their greater cardiovascular involvement, the evaluation will be closely tailored on individual cases.

Hemodynamically significant forms of ACo are characterized by a mean pressure gradient > 15 mmHg, hypertension at rest and on effort, diffuse collateral arteries, etc. They contraindicate any types of sports activity and require the correction of the defect, either by transcatheter angioplasty and/or stent implantation, or by traditional surgical repair^{21,22}.

Six months after correction, subjects may be re-evaluated according to the same criteria stated above. Because of the possibility of residual hypertension, it is recommended to perform a 24-hour BP monitoring²³⁻²⁵. Patients showing a pseudo- or complete normalization of the clinical and laboratory findings are admitted to participate in any sports, except for those belonging to group C (“pressure” cardiovascular involvement).

Because of the possibility, although rare, of aortic rupture as a consequence of chest trauma, the COCIS Committee, even without a scientific evidence, states that subjects with hemodynamically non-significant or successfully corrected ACo can be granted sports eligibility if they do not show other aortic anomalies (bicuspid aortic valve, dilation of the ascending aorta). Eligibility can also be granted for those contact sports (such as soccer and basketball) at low risk of violent blows to the chest.

Aortic stenosis. Usually congenital aortic stenosis (AS) is caused by an anomalous or missing valve leaflet. Bicuspid aortic valve is the most common specific anomaly²⁶⁻²⁸.

In young subjects the diagnosis is made on the basis of an ejection “click” associated with a systolic murmur in the aortic area. ECHO color Doppler allows to confirm the diagnosis and to evaluate non-invasively the degree of blood flow obstruction^{29,30} as well as the degree of aortic regurgitation. The latter is a frequent finding and sometimes it is the sole hemodynamic alteration in the young with bicuspid aortic valve. AS with a mean pressure gradient < 20 mmHg at rest are considered as hemodynamically non-significant.

Indications. Subjects with minimal AS or uncomplicated bicuspid aortic valve can participate in sports of any type, provided that the following criteria are met:

- no left ventricular hypertrophy (ECG, ECHO) and normal systo-diastolic ventricular function; normal dimensions of the aortic bulb and thoracic aorta. This as-

pect is of paramount importance in case of bicuspid aortic valve and should be investigated with ECHO and possibly with magnetic resonance imaging³¹⁻³³;

- normal EST (normal increase in systolic BP, no ST-T wave alterations);
- no significant arrhythmias at rest and during effort (EST and HM comprehensive of a training session).

Subjects with hemodynamically significant AS (mean pressure gradient > 20 mmHg) cannot participate in competitive sports and should be referred to intervention, when indicated.

Once valvuloplasty has been performed³⁴, with a good outcome in selected cases (residual mean pressure gradient < 20 mmHg, non-significant aortic regurgitation, no ECG abnormalities and/or arrhythmias at EST and HM comprehensive of a training session), eligibility can be granted to competitive sports of minimum to moderate cardiovascular involvement, such as those of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A, with a mandatory cardiovascular evaluation every 6 months. The same criteria should be applied to patients with normally functioning homografts or bioprostheses.

An increasing number of young subjects undergo the Ross procedure (replacement of the aortic valve with the patient's pulmonary valve, re-implantation of the coronary arteries, positioning of a homograft in the pulmonary site). This procedure may result in a number of residual defects, usually negligible^{34,35}. In these cases, eligibility can be granted to sports of group B2, some of group B1 (horse riding, sailing) and non-competitive athletic activities of group A, provided that:

- left ventricular dimensions and function are normal;
- the mean pressure gradient between the right ventricle and the pulmonary artery is < 20 mmHg;
- there is no significant aortic regurgitation;
- EST and HM show no ECG abnormalities and/or arrhythmias, comprehensive of a training session.

Also this condition requires a complete cardiovascular evaluation after 6 months.

As for patients with a mechanical valve, see specific section.

For discrete subaortic stenoses, the same criteria applied to valvular stenosis should be adopted, with little adjustments. For operated subaortic stenoses, eligibility can be granted to sports of any type, provided that the post-operative functional examination shows:

- a residual mean gradient < 10 mmHg without significant aortic regurgitation;
- normal left ventricular dimensions and function (ECHO);
- normal EST (normal increase in systolic BP, no ST-T wave abnormalities);
- no significant arrhythmias at rest and at EST and HM, comprehensive of a training session.

On the other hand, supra-aortic stenosis should always be evaluated accurately, because of the reported possibility of coronary artery anomalies.

Pulmonary stenosis. Pulmonary valve stenosis (PS) can be suspected based on a systolic ejection murmur best heard in the pulmonary area, possibly with an ejection click, ECG markers of right ventricular hypertrophy and/or radiologic evidence of a dilated pulmonary artery. Differently from AS, subjects with PS are often asymptomatic, even with a severe stenosis. The degree of the obstruction can be easily evaluated by means of clinical criteria, ECG and ECHO color Doppler³⁶.

PS with a peak maximum gradient < 30 mmHg are considered as hemodynamically non-significant; PS with a gradient > 30 mmHg are considered as hemodynamically significant (also classified as moderate, gradient between 30 and 50 mmHg, and severe, gradient > 50 mmHg).

Indications. In hemodynamically non-significant PS eligibility can be granted to sports of any type, provided that EST shows a normal performance.

In moderate PS, eligibility can be granted to competitive sports of minimum to moderate cardiovascular involvement, such as those of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A. In severe PS eligibility to competitive sports can be granted only after successful repair, either by balloon valvuloplasty³⁷ or surgical valvulotomy.

Six months after correction, eligibility to competitive sports can be granted, provided that:

- the intervention was not performed by ventriculotomy;
- the residual peak gradient is < 30 mmHg;
- mild pulmonary regurgitation, if any;
- normal right ventricular function (ejection fraction > 50%).

Tetralogy of Fallot. Tetralogy of Fallot is characterized by a non-restrictive VSD overriding the aorta and by PS. It is the most frequent congenital heart disease causing cyanosis. Patients with tetralogy of Fallot or different types of cyanotic conditions cannot participate in sports unless they have undergone complete surgical correction. Nowadays, interventions are performed earlier in life, and the outcome has become improved considerably.

Up to date, most experts thought reasonable not to grant eligibility to competitive sports to patients who have undergone surgical repair since the reported risk of arrhythmic sudden cardiac death could not always be defined reliably on clinical and laboratory post-operative data³⁸. Moreover, the risk was even higher in patients operated upon with conventional procedure and/or later in life at the beginning of the cardiac surgery era. The well established tendency to an early and complete surgical correction allows sports physicians to examine eligibility to operated patients, whose outcome is nowadays far better than in the past. Therefore, eligibility can be considered to competitive sports of minimum to moderate cardiovascular involvement,

such as those of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A, only in selected cases in which the intervention was not performed by ventriculotomy, and if the post-intervention evaluation shows the following:

- no significant residual pulmonary peak gradient (> 30 mmHg), and trivial or mild pulmonary regurgitation, if any;
- no right ventricular hypertrophy and/or dilation, normal ejection fraction ($> 50\%$ at ECHO; in case of doubt, cardiac catheterization is indicated);
- normal exercise tolerance^{39,40}, with a normal increase in systolic BP, and no pathologic alterations in ventricular repolarization;
- no significant arrhythmias, either at rest or during EST and HM comprehensive of a training session⁴¹⁻⁴³.

Due to the complex features and the different aspects and outcome of this cardiac anomaly, it should be always carried out a tailored evaluation and patients should be referred to expert health professionals. Sports eligibility should be re-assessed every 6 months by a thorough cardiovascular evaluation.

Transposition of the great arteries. The transposition of the great arteries is a complex congenital heart defect and its surgical correction has radically changed over the past few years. Formerly, repair was performed by Mustard or Senning procedures which consisted of the inversion of the venous return within the atria (so-called atrial switch) causing the right ventricle to work in the hemodynamic “left” or systemic position⁴⁴⁻⁴⁶. Moreover, intra-atrial surgery contributed to develop supraventricular arrhythmias; thus subjects who underwent such procedures should be denied eligibility to competitive sports.

Nowadays, the transposition of the great arteries is corrected within 15 days of birth by means of the “arterial switch” (the great vessels are switched and the coronary arteries are translocated to the neo-aorta). However, this procedure, while allowing the correct anatomic and functional recovery, might facilitate aortic valve regurgitation and might cause stenosis of the pulmonary trunk and/or branches⁴⁷.

Based on these data and similarly to what already stated about tetralogy of Fallot, eligibility can be considered to competitive sports of minimum to moderate cardiovascular involvement, such as those of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A, in selected cases with a long-term optimal outcome. Because of the complex features of this cardiac anomaly (residual valvular defects, need to verify the correct function of the translocated coronary arteries, etc.) and its great individual variability, it is recommended that the evaluation be accurately planned in each single case, referring the patient to expert health professionals. Sports eligibility should be re-assessed every 6 months by a thorough cardiovascular evaluation.

Acquired valvular heart diseases and mitral valve prolapse

Mitral stenosis. Mitral stenosis (MS) is nearly always of rheumatic etiology. The obstruction to left ventricular inflow results in an increase of the left atrial and pulmonary capillary pressures at rest, and worsens on exertion because of the higher heart rate (and its inherent reduction in diastolic filling time and cardiac output)^{48,49}. An independent risk factor is represented by peripheral embolization.

The hemodynamic severity of MS can be easily assessed non-invasively at ECG and in particular at ECHO color Doppler, which allows a reliable evaluation of the mitral valve cross-sectional area, transvalvular pressure gradient and pulmonary artery pressure⁵⁰. In doubt cases, transesophageal ECHO can be performed in order to better evaluate the anatomic features of the mitral valve.

For instance, MS can be considered as mild when the estimated valve cross-sectional area is > 2 cm², as moderate when it ranges between 1.1 and 1.9 cm², otherwise it is severe.

Indications. Sports of any type are contraindicated in moderate to severe forms, and always in cases with permanent AF.

In mild forms, as well as in selected cases of moderate MS with sinus rhythm, eligibility can be considered to sports of minimum to moderate cardiovascular involvement (group B2, and non-competitive athletic activities of group A) provided that EST has shown normal exercise tolerance, and no significant ventricular arrhythmias are observed at HM comprehensive of a training session.

In subjects with MS corrected by either commissurotomy or valvuloplasty, 6 months post-intervention, eligibility can be granted to sports of minimum to moderate cardiovascular involvement (group B2, and non-competitive athletic activities of group A), provided that there is no pulmonary hypertension with a cross-sectional area ≥ 2 cm², and no significant regurgitation⁵¹.

Mitral regurgitation. Unlike MS (which can be associated in its rheumatic form), mitral regurgitation (MR) may have a multiple etiology: rheumatic (ever more rare), congenital by a cleft in the anterior leaflet (ostium primum and AVSD), degenerative (MVP, presently the most frequent cause), infective (endocarditis), etc.

When assessing MR severity to the purpose of sports eligibility, the etiology is of the utmost importance:

- in the secondary forms (for example, in the Marfan syndrome) the possible eligibility depends upon the underlying disease severity⁵²;
- in the primary forms (rheumatic etiology, leaflet prolapse) eligibility should be considered based on the de-

gree of hemodynamic involvement⁵³⁻⁵⁵, i.e. left atrial and ventricular dimensions (ECG and ECHO), left ventricular function at rest and during effort (by stress ECHO Doppler and/or radionuclide ventriculography) and possible ventricular arrhythmias (EST and HM comprehensive of a training session).

MR is considered as mild when detected only at auscultation, validated by ECHO color Doppler (mild to moderate regurgitation), with a normal ECG and normal atrial and ventricular dimensions at ECHO. MR is considered as moderate when associated with a slight left ventricular enlargement, with preserved ventricular function at rest and during effort (normal increase in ejection fraction during dynamic effort). MR is considered as severe in the remaining cases.

Indications. Patients with mild MR can participate in sports of minimum to moderate cardiovascular involvement (group B, and non-competitive athletic activities of group A). In selected cases, eligibility can be considered to sports of moderate to high cardiovascular involvement (group D), with a close monitoring over time (6-month eligibility).

Patients with moderate MR cannot participate in competitive sports, except for those activities of minimum to moderate cardiovascular involvement of group B2, some of group B1 (sailing, horse riding and polo), and of group A.

Patients with severe MR cannot participate in competitive sports of any type.

In subjects undergoing surgical valve repair⁵⁶ by means of valvuloplasty, sports eligibility can be re-considered by evaluating the possible progression of the underlying disease, the hemodynamic outcome, left ventricular dimensions and function, the presence/absence of significant ventricular arrhythmias at EST and HM comprehensive of a training session. Similarly to the other cardiac anomalies described above, patients should be referred to expert health professionals.

As for indications in prosthetic mitral valve replacement, see the specific paragraph.

Aortic stenosis. For acquired AS see what already stated about congenital forms. Except for the rheumatic forms, AS in adulthood is caused by a degenerative process and calcification of a congenital anomalous valve.

Aortic regurgitation. Similarly to MR, aortic regurgitation (AR) may show different etiologies: congenital (bicuspid aortic valve), rheumatic, from infective endocarditis, secondary to Marfan syndrome, etc.

General considerations stated on MR also apply to AR. However, on exertion, the hemodynamically significant forms, usually symptomatic, may cause partial coronary insufficiency, though AR hemodynamics may improve during effort since the shortened diastole and decreased peripheral resistance (dynamic exercise) may decrease the amount of regurgitating blood⁵⁷.

AR can be considered as mild if no alterations are found in left ventricular dimensions and function at rest and during effort (assessed at stress-ECHO or radionuclide ventriculography), and there are no systemic signs of AR (increased pulse pressure, pulsus celeris, etc.). AR can be considered as moderate when such peripheral signs are found, but left ventricular dimensions are only slightly increased, and ventricular function at rest and during effort is normal. AR is considered as severe in the remaining cases.

Indications. Patients with mild AR will be allowed to participate in sports of minimum to moderate cardiovascular involvement, such as those of group B, and non-competitive athletic activities of group A. In selected cases eligibility can also be granted to moderately or highly demanding sports, once an accurate assessment of ventricular function on exertion has been obtained. However, a close monitoring of left ventricular dimensions and function over time is recommended (6-month eligibility).

In patients with moderate AR, eligibility cannot be granted, except for sports of minimum to moderate cardiovascular involvement of group B2, and some of group B1 (horse riding, sailing).

Participation in competitive sports of any type will always be denied in patients with severe AR.

Valve prosthesis. Overall, sports eligibility is rejected to patients with mechanical valves on chronic anticoagulant therapy⁵⁸. However, in selected patients eligibility can be granted to sports of minimum to moderate cardiovascular involvement of group B2, and non-competitive athletic activities of group A provided that findings are consistent with a normal function of the prosthetic valve, no ventricular impairment and no significant ventricular arrhythmias are observed at EST and HM comprehensive of a training session.

Mitral valve prolapse. MVP is characterized by one or both mitral leaflets protruding into the mitral annulus toward the left atrium during ventricular systole. In most cases, it is caused by myxomatous degeneration of the valve or of the subvalvular system⁵⁹.

The most relevant clinical and diagnostic features are a mid-end-systolic click of varying intensity and/or a murmur by end-systolic or holosystolic regurgitation at auscultation, and specific valve abnormalities at ECHO color Doppler⁶⁰. The latter exam is of pivotal use when assessing the degree of MVP, its etiology and the possible association with additional anomalies (prolapse of other valves, aortic dilation, etc.).

Suggestive, though not specific, findings of MVP are the physical constitution (Marfan-like)^{61,62}, family history, atypical chest pain, orthostatic hypotension, and palpitations. In fact, MVP is associated, to some degree, with brady and/or tachyarrhythmias (see specific section).

In order to correctly assess athletes with MVP it is necessary to obtain an accurate personal and family history, and to perform ECHO color Doppler, EST and HM comprehensive of a training session.

The degree of MVP may vary considerably, from severe to extremely mild forms, without any clinical and/or hemodynamic signs (no or minimal and sporadic MR). Subjects will be considered at risk and therefore not allowed to participate in competitive sports in case of:

- unexplained syncope, family history of premature sudden cardiac death, long-QT syndrome;
- moderate to severe MR;
- recurrent supraventricular tachyarrhythmias or complex ventricular arrhythmias at rest and/or during effort (see specific section)⁶³⁻⁶⁷.

In patients with an overt myxomatous mitral valve and mild MR, eligibility may be considered to competitive sports of minimum to moderate cardiovascular involvement of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A. A complete cardiological evaluation, at least yearly, is necessary, because MVP anatomy and function may degenerate over time due to worsening of mucoid degeneration and possible rupture of the chordae or, more rarely, due to infective endocarditis⁶⁸⁻⁷⁰.

The criteria stated above are to be considered as valid also when MVP is associated with tricuspid valve regurgitation. Conversely, major attention should be paid when MVP is associated with aortic prolapse of one or more cusps and/or with aortic root dilation, independently of the presence/absence of Marfan syndrome.

References

1. Musewe NN, Smallhorn JF, Benson LN, Burrows PE, Freedom RM. Validation of Doppler-derived pulmonary arterial pressure in patients with ductus arteriosus with different hemodynamic states. *Circulation* 1987; 76: 1081-91.
2. Ballerini L, Mullins CE, Cifarelli A, et al. Chiusura non chirurgica del dotto arterioso pervio in età pediatrica con dispositivo di Rashkind a doppio ombrello. *G Ital Cardiol* 1990; 20: 805-9.
3. Vetter VL, Horowitz LN. Electrophysiologic residua and sequelae of surgery for congenital heart defects. *Am J Cardiol* 1982; 50: 588-604.
4. Bink-Boelkens MT, Velvis H, van der Heide JJ, Eygelaar A, Hardjowijono RA. Dysrhythmias after atrial surgery in children. *Am Heart J* 1983; 106: 125-30.
5. Cross SJ, Evans SA, Thomson LF, Lee HS, Jennings KP, Shields TG. Safety of subaqua diving with a patent foramen ovale. *BMJ* 1992; 304: 481-2.
6. Moccheggiani R, Berrettini U. Forame ovale pervio. In: Berrettini U, ed. *Fisiopatologia cardiovascolare e polmonare in ambiente iperbarico*. Milano: Edizioni MICOM, 1999: 57.
7. De Castro S, Cartoni D, Fiorelli M, et al. Morphological and functional characteristics of patent foramen ovale and their embolic implications. *Stroke* 2000; 31: 2407-13.
8. Cantais E, Louge P, Suppini A, Foster PP, Palmier B. Right-to-left shunt and risk of decompression illness with cochleovestibular and cerebral symptoms in divers: case control study in 101 consecutive dive accidents. *Crit Care Med* 2003; 31: 84-8.
9. Cecconi M, Pangrazi A, Manfrin M, et al. Il trattamento interventistico nel paziente con difetto interatriale. *Sports Card* 2001; 3: 174-8.
10. Bharati S, Lev M. Sequelae of atriotomy and ventriculotomy on the endocardium, conduction system and coronary arteries. *Am J Cardiol* 1982; 50: 580-7.
11. Lynch JJ, Schuchard GH, Gross CM, Wann LS. Prevalence of right-to-left atrial shunting in healthy population. Detection by Valsalva maneuver contrast echocardiography. *Am J Cardiol* 1984; 53: 1478-80.
12. King RM, Puga FJ, Danielson GK, et al. Prognostic factors and surgical treatment of partial atrioventricular canal. *Circulation* 1986; 74: I42-I46.
13. Feldt RH, Edwards WD, Porter CJ, Dearani JA, Seward JB, Puga FJ. Atrioventricular septal defects. In: Allen HD, Gutgesell HP, Clark EB, Driscoll DJ, eds. *Moss and Adams' heart disease in infants, children and adolescents*. 6th edition. Philadelphia, PA: Lippincott Williams & Wilkins, 2001: 618-35.
14. Seward JB, Tajik AJ, Hagler DJ. Two-dimensional echocardiographic features of atrioventricular canal defect. In: Lundstrom NR, ed. *Pediatric echocardiography: cross sectional, M-mode and Doppler*. New York, NY: Elsevier/North Holland, 1980: 197-206.
15. Moller JH, Patton C, Varco RL, Lillehei CW. Late results (30 to 35 years) after operative closure of isolated ventricular septal defect from 1954 to 1960. *Am J Cardiol* 1991; 68: 1491-7.
16. Kidd L, Driscoll DJ, Gersony WM, et al. Second natural history study of congenital heart defects: results of treatment of patients with ventricular septal defects. *Circulation* 1993; 87: I38-I51.
17. Onat T, Ahunbay G, Batmaz G, Celebi A. The natural course of isolated ventricular septal defect during adolescence. *Pediatr Cardiol* 1998; 19: 230-4.
18. Simpson IA, Sahn DJ, Valdes Cruz LM, Chung KJ, Sherman FS, Swensson RE. Color Doppler flow mapping in patients with coarctation of the aorta: new observations and improved evaluation with color flow diameter and proximal acceleration as predictor of severity. *Circulation* 1988; 77: 736-44.
19. Carvalho JS, Redington AN, Shinebourne EA, Rigby ML, Gibson D. Continuous wave Doppler echocardiography and coarctation of the aorta: gradients and flow patterns in the assessment of severity. *Br Heart J* 1990; 64: 133-7.
20. Crepaz R, Pitscheider W, Oberhollenzer R, et al. Follow-up a lungo termine in pazienti operati di coartazione aortica. Valutazione con eco-Doppler e RMN della funzione ventricolare sinistra e del gradiente transistmico. *G Ital Cardiol* 1993; 23: 767-76.
21. Carminati M. Dilatation of congenital aortic stenosis. *Cardiologia* 1995; 40: 409-11.
22. Zabal C, Attie F, Rosas M, Buendia-Hernandez A, Garcia-Montes JA. The adult patient with native coarctation of the aorta: balloon angioplasty or primary stenting? *Heart* 2003; 89: 77-83.
23. Markel H, Rocchini AP, Beekman RH, et al. Exercise induced hypertension after repair of coarctation of the aorta: arm versus leg exercise. *J Am Coll Cardiol* 1986; 8: 165-71.
24. Mori F, Favilli S, Zuppiroli A, et al. Idoneità sportiva in pazienti operati di coartazione aortica: valutazione mediante ecocardiografia Doppler da sforzo. *G Ital Cardiol* 1993; 23: 225-37.
25. Hauser M. Exercise blood pressure in congenital heart dis-

- ease and in patients after coarctation repair. *Heart* 2003; 89: 125-6.
26. Ward C. Clinical significance of the bicuspid aortic valve. *Heart* 2000; 83: 81-5.
 27. Fedak P, Verma S, David TD, et al. Clinical and pathophysiological implications of a bicuspid aortic valve. *Circulation* 2002; 106: 900-4.
 28. Harrison MR, Gurley JC, Smith MD, Grayburn PA, DeMaria AN. A practical application of Doppler echocardiography for the assessment of severity of aortic stenosis. *Am Heart J* 1988; 115: 622-8.
 29. Baldinelli A, Cecconi M, Perna GP. La bicuspidia aortica: aspetti clinici anatomo-funzionali. Esperienza ecocardiografica. *Sports Card* 2002; 3: 19-22.
 30. Lindsay J Jr. Coarctation of the aorta, bicuspid aortic valve and abnormal ascending aortic wall. *Am J Cardiol* 1988; 61: 182-4.
 31. Giusti S, Cocco P, Thiene G. Valvola aortica bicuspid: una cardiopatia congenita "minore" a rischio di gravi complicanze. *G Ital Cardiol* 1991; 21: 189-201.
 32. Nistri S, Sorbo MD, Basso C, Thiene G. Bicuspid aortic valve: abnormal aortic elastic properties. *J Heart Valve Dis* 2002; 11: 369-73.
 33. Alegret JM, Duran I, Palazon O, et al. Prevalence of and predictors of bicuspid aortic valves in patients with dilated aortic roots. *Am J Cardiol* 2003; 91: 619-22.
 34. Sullivan ID, Wren C, Bain H, et al. Balloon dilatation of the aortic valve for congenital aortic stenosis in childhood. *Br Heart J* 1989; 61: 186-91.
 35. Fullerton DA, Fredericksen JW, Sundaresan RS, Horvath KA. The Ross procedure in adults: intermediate-term results. *Ann Thorac Surg* 2003; 76: 471-6.
 36. Oliveira Lima C, Sahn DJ, Valdes-Cruz LM, et al. Noninvasive prediction of transvalvular pressure gradient in patients with pulmonary stenosis by quantitative two-dimensional echo-Doppler studies. *Circulation* 1983; 67: 866-71.
 37. Kan JS, White RI Jr, Mitchell SE, Gardner TJ. Percutaneous balloon valvuloplasty: a new method for treating congenital pulmonary valve stenosis. *N Engl J Med* 1982; 307: 540-2.
 38. Garson A, Gillette PC, Gutgesell HP, McNamara DG. Stress-induced ventricular arrhythmias after repair of Tetralogy of Fallot. *Am J Cardiol* 1980; 46: 1006-12.
 39. Tokumura M, Yoshiba S, Kojima Y, Nanri S. Impaired cardiorespiratory response to brief sudden strenuous exercise in the postoperative tetralogy of Fallot patients: a ten-second pedaling test. *Pediatr Cardiol* 2002; 23: 496-501.
 40. Sarubbi B, Pacileo G, Pisacane C, et al. Exercise capacity in young patients after total repair of tetralogy of Fallot. *Pediatr Cardiol* 2000; 21: 211-5.
 41. Daliotto L, Rizzoli G, Menti L, et al. Accuracy of electrocardiographic and echocardiographic indices in predicting life threatening ventricular arrhythmias in patients operated for tetralogy of Fallot. *Heart* 1999; 81: 650-5.
 42. Vignati G, Mauri L, Figini A, Pome G, Pellegrini A. Immediate and late arrhythmia in patients operated on for tetralogy of Fallot. *Pediatr Med Chir* 1998; 20: 3-6.
 43. Gatzoulis MA, Balaji S, Webber SA, et al. Risk factors for arrhythmia and sudden cardiac death late after repair of tetralogy of Fallot: a multicentre study. *Lancet* 2000; 356: 975-81.
 44. Graham TP Jr. Haemodynamic residua and sequelae following intraatrial repair of transposition of the great arteries: a review. *Pediatr Cardiol* 1982; 2: 203-13.
 45. Helbing WA, Hansen B, Ottenkamp J, et al. Long-term results of atrial correction for transposition of the great arteries: comparison of Mustard and Senning operations. *J Thorac Cardiovasc Surg* 1994; 108: 363-72.
 46. Wilson NJ, Clarkson PM, Barratt-Boyes BG, et al. Long-term outcome after the Mustard repair for simple transposition of the great arteries: 28-year follow-up. *J Am Coll Cardiol* 1998; 32: 758-65.
 47. Reybrouck T, Eyskens B, Mertens L, Defoor J, Daenen W, Gewillig M. Cardiorespiratory exercise function after the arterial switch operation for transposition of the great arteries. *Eur Heart J* 2001; 22: 1052-9.
 48. Dahan M, Paillole C, Martin D, Gourgon R. Determinants of stroke volume response to exercise in patients with mitral stenosis: a Doppler echocardiographic study. *J Am Coll Cardiol* 1993; 21: 384-9.
 49. Cohen-Solal A, Aupetit JF, Dahan M, Baleynaud S, Slama M, Gourgon R. Peak oxygen uptake during exercise in mitral stenosis with sinus rhythm or atrial fibrillation: lack of correlation with valve area. *Eur Heart J* 1994; 15: 37-44.
 50. Voelker W, Berner A, Regele B, Schmid M. Effect of exercise on valvular resistance in patients with mitral stenosis. *J Am Coll Cardiol* 1993; 22: 777-82.
 51. Reid CL, Chandraratna PA, Kawanishi DT, Kotlewski A, Rahimtoola SH. Influence of mitral valve morphology on double-balloon catheter balloon valvuloplasty in patients with mitral stenosis. Analysis of factors predicting immediate and 3-month results. *Circulation* 1989; 80: 515-24.
 52. Roberts WC, Honig HS. The spectrum of cardiovascular disease in the Marfan syndrome: a clinico-morphologic study of 18 necropsy patients and comparison to 151 previously reported necropsy patients. *Am Heart J* 1982; 104: 115-35.
 53. Chen C, Thomas JD, Anconina J, et al. Impact of impinging wall jet on color Doppler quantification of mitral regurgitation. *Circulation* 1991; 84: 712-20.
 54. Thomas L, Foster E, Hoffman JIE, Schiller NB. The mitral regurgitation index: an echocardiographic guide to severity. *J Am Coll Cardiol* 1999; 33: 2016-22.
 55. Pu M, Thomas JD, Vadervoort PM, Steward WJ, Cosgrove DM, Griffin BP. Comparison of quantitative and semiquantitative methods for assessing mitral regurgitation by transesophageal echocardiography. *Am J Cardiol* 2001; 87: 66-70.
 56. Ling LH, Enriquez-Sarano M, Steward JB, et al. Early surgery in patients with mitral regurgitation due to flail leaflets: a long-term outcome study. *Circulation* 1997; 96: 1819-25.
 57. Wisenbaugh T, Spann JF, Carabello BA. Differences in myocardial performance and load between patients with similar amounts of chronic aortic versus chronic mitral regurgitation. *J Am Coll Cardiol* 1984; 3: 916-23.
 58. Solinas P, Notaristefano A, Ascani F. Cardiac surgery and sport: mechanical valve prosthesis and sport. In: D'Alessandro LC, ed. *Heart surgery* 1989. Roma: CESI, 1989: 599.
 59. Barlow JB. Idiopathic (degenerative) and rheumatic mitral valve prolapse: historical aspects and overview. *J Heart Valve Dis* 1992; 1: 163-74.
 60. Feigenbaum H. Echocardiography in the management of mitral valve prolapse. *Aust NZ J Med* 1992; 22: 550-5.
 61. Flack JM, Kvasnicka JH, Gardin JM, Gidding SS, Manolio TA, Jacobs DR Jr. Anthropometric and physiologic correlates of mitral valve prolapse in a biethnic cohort of young adults: the CARDIA study. *Am Heart J* 1999; 138: 486-92.
 62. Arfken CL, Schulman P, McLaren MJ, Lachman AS. Mitral valve prolapse and body habitus in children. *Pediatr Cardiol* 1993; 14: 33-6.
 63. Kramer HM, Kligfield P, Devoreux RB, Savage DD, Kramer-Fox R. Arrhythmias in mitral valve prolapse. Effect of selection bias. *Arch Intern Med* 1984; 144: 2360-4.
 64. La Vecchia L, Ometto R, Centofante P, et al. Arrhythmic profile, ventricular function, and histomorphometric findings in patients with idiopathic ventricular tachycardia and mitral valve prolapse: clinical and prognostic evaluation. *Clin Cardiol* 1998; 21: 731-5.

65. Vohra J, Sathe S, Warren R, Tatoulis J, Hunt D. Malignant ventricular arrhythmias in patients with mitral valve prolapse and mild mitral regurgitation. *Pacing Clin Electrophysiol* 1993; 16: 387-93.
66. Corrado D, Basso C, Nava A, Rossi L, Thiene G. Sudden death in young people with apparently isolated mitral valve prolapse. *G Ital Cardiol* 1997; 27: 1097-105.
67. Dolla 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: 921-31.
68. Duren DR, Becker AE, Dunning AJ. Long-term follow-up of idiopathic mitral valve prolapse in 300 patients: a prospective study. *J Am Coll Cardiol* 1988; 11: 42-7.
69. Zuppiroli A, Mori F, Favilli S. et al. Natural histories of mitral valve prolapse. Influence of patient selection on cardiovascular event rates. *Ital Heart J* 2001; 2: 107-14.
70. MacMahon SW, Roberts JK, Kramer Fox R, Zucker DM, Roberts RB, Devereux RB. Mitral valve prolapse and infective endocarditis. *Am Heart J* 1987; 113: 1291-8.

CARDIOMYOPATHIES, MYOCARDITIS AND PERICARDITIS

Cardiomyopathies

In 1980, the World Health Organization and the International Society and Federation of Cardiology defined cardiomyopathies as those myocardial diseases of unknown etiology, by classifying them in three groups according to the predominant morphologic finding: dilated, hypertrophic, and restrictive. The rationale to such an approach was the specificity of pathophysiologic features, of the clinical course and of drug treatment. A fourth group, defined as unclassified cardiomyopathies, gathered those types otherwise not included in the three main categories. Then other specific myocardial diseases were taken into consideration, such as those of known etiology or associated with systemic diseases¹ till the inclusion, in 1996, of the arrhythmogenic right ventricular cardiomyopathy (ARVC) (Table I)².

Table I. Classification of cardiomyopathies.

Hypertrophic cardiomyopathy
Dilated cardiomyopathy
Idiopathic, familial/genetic, viral/autoimmune, alcoholic/toxic
Restrictive cardiomyopathy
Idiopathic myocardial fibrosis, endomyocardial fibrosis, Löffler's disease, amyloidosis
Arrhythmogenic right ventricular cardiomyopathy
Unclassified
Fibroelastosis, myocardial non-compaction, systolic dysfunction with minimal dilation, mitochondrial myopathies, etc.
Specific
Ischemic, valvular, hypertensive, inflammatory (see myocarditis), tachycardia-induced, metabolic, peripartum, etc.

In the sports medicine setting, some forms are of great interest, such as hypertrophic (HCM) or dilated (DCM) cardiomyopathy and ARVC, because of their prevalence, and because they might cause sudden cardiac death in young athletes. Moreover, these heart diseases may mimic some typical features of the "athlete's heart", leading to complex differential diagnosis issues.

Hypertrophic cardiomyopathy

HCM is an idiopathic myocardial disease of genetic etiology and is characterized by a hypertrophic, not enlarged, left ventricle, without any further cardiac or systemic cause responsible for the hypertrophy³. The prevalence of HCM is higher than formerly hypothesized (estimated prevalence in the western population, 0.2%)^{4,5} presenting with a wide range of morphologic and clinical findings, ranging from sudden cardiac death during sports and exercise, to benign forms with a paucisymptomatic clinical course, with no events until old age.

Diagnosis. Useful findings are:

- family history of HCM and/or premature sudden cardiac death. In the past years, many genetic defects have been disclosed regarding the genes encoding sarcomere polypeptides, thus allowing great advances in the understanding of HCM etiopathogenesis and pathophysiology, and raising expectations toward genetic analysis, with the hope that it could lead to a definite diagnosis regardless of the morphologic and clinical features⁶. However, such an analysis is available only in selected clinical Centers, it is expensive and time-consuming and, thus far, cannot be considered as a routine diagnostic tool;
- clinical symptoms and signs: symptoms are of minimal relevance since athletes are often (or, at least, so they say!) asymptomatic⁷. Only in few cases the clinical history shows syncope, palpitations, chest pain, and effort dyspnea. Great attention should be paid to syncope, especially when occurring on exertion⁸;
- clinical findings can be negative or sometimes show a fourth cardiac sound, an ejection murmur best heard between the apex and the left sternal border, and on the base (in the rare obstructive forms), and sometimes an apical systolic murmur from MR;
- ECG: it is almost always abnormal, showing one or more alterations, such as high-voltage QRS, left-axis deviation sometimes marked pathologic Q-waves, abnormal ventricular repolarization, and signs of left atrial enlargement^{9,10}. It should be outlined that these alterations may be observed also in highly-trained athletes with no cardiac morphologic anomalies: therefore, by themselves, they do not allow the diagnosis of HCM;
- ECHO: it can confirm or exclude HCM in most cases. The main diagnostic feature is left ventricular hy-

hypertrophy, whose degree and pattern are extremely variable (even in the affected members of the same family), even though the maximum wall thickness usually exceeds 20 mm. Hypertrophy has a typical asymmetric and non-homogeneous pattern and mostly involves the interventricular septum. In few cases, hypertrophy can be mild (maximum wall thickness 13-15 mm), similar to that observed in male top endurance athletes (cyclists, rowers, triathletes, etc.). Additional anomalies, such as mitral valve structural anomalies, an anomalous left ventricle, left atrial enlargement and impaired left ventricular filling (inverted E/A ratio of the mitral flow, slowing relaxation) may also be found which help in the differential diagnosis of the physiologic hypertrophy of the athlete's heart (Table II)^{3,11,12}.

In all uncertain cases, when ECHO does not provide conclusive results, it can be useful to re-evaluate the athlete after a period of total deconditioning (at least 3 months): the finding of a clear reduction in the ventricular wall thickness within normal range (< 13 mm) is one of the criteria to make the diagnosis of the athlete's heart¹³. Children and teenagers deserve great attention, because in the majority of cases hypertrophy develops during pubertal growth¹⁴; therefore, serial ECHO evaluation are recommended in children with affected relatives, until their full growth.

Invasive tests, such as radionuclide ventriculography, coronary angiography and biopsy are usually not necessary to the diagnosis of HCM, and should be limited to selected cases, after a thorough evaluation by expert health professionals.

Risk assessment. Although sudden cardiac death (often during sport or exercise) can be the first (and unique) event in the natural history of HCM, the actual risk of HCM is hard to assess since prospective clinical trials are not available¹⁵. However, additional useful findings leading to a better risk assessment can be obtained at:

- EST in order to evaluate carefully the BP pattern on exertion, ventricular arrhythmias or symptoms¹⁶, if any, and the total workload (or VO₂ max, when performing a cardiopulmonary test);
- HM in order to define the type and complexity of arrhythmias, if any^{17,18}.

Thus, even in the absence of data from specific trials, it is reasonable to define at low risk of sudden cardiac death or of cardiovascular events those patients with HCM who show¹⁹:

- no symptoms, especially syncope or prolonged, recurrent or effort palpitations;
- no family history of sudden cardiac death;
- "mild" hypertrophy (maximum ventricular wall thickness < 18 mm), no atrial enlargement (< 45 mm) and normal Doppler diastolic filling pattern;
- normal BP increase at EST;
- no significant atrial and ventricular arrhythmias (HM) (see specific section);
- age > 35 years.

Indications. Subjects with a definite diagnosis of HCM should not participate in competitive sports of any type. A possible exception might be represented by subjects classified at low risk, after a close and tailored evaluation, based on the criteria stated above, carried out by expert health professionals. In this case, eligibility can be granted to sports of minimum to moderate cardiovascular involvement of group B2, and some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A, with a strict and serial evaluation.

Dilated cardiomyopathy

DCM is a myocardial disease characterized by impaired systolic function and (usually) left ventricular enlargement. DCM may be genetic and hereditary, or secondary to infectious, inflammatory (see myocarditis) or metabolic forms, or may result from exposure to toxic compounds²⁰. Sports physicians generally encounter the initial forms of DCM, asymptomatic or paucisymptomatic, with minimal functional impairment.

Diagnosis. Useful diagnostic findings are:

- family history of DCM and/or premature sudden cardiac death²¹;
- clinical history, showing fatigue, effort dyspnea, palpitations or syncope in a variable number of cases;
- physical examination, which may disclose a third or

Table II. Differential diagnosis between hypertrophic cardiomyopathy (HCM) and the athlete's heart.

	HCM	Athlete's heart
Hypertrophic pattern	Asymmetric, non-homogeneous	Symmetric, homogeneous
Maximum wall thickness (mm)	> 16	< 16
Left ventricular dimensions	Normal or decreased	Normal or increased
Doppler diastolic filling	Altered or normal	Normal
Hypertrophy reversibility after deconditioning	Absent or incomplete	Complete
HCM family history	Often present	Absent

fourth sound and sometimes a systolic apical murmur due to MR;

- ECG abnormalities concerning the QRS complex (high or low amplitude, conduction disturbances, etc.), ventricular repolarization, atrial enlargement, as well as atrial and/or ventricular arrhythmias²²;
- ECHO, which can confirm left ventricular systolic dysfunction usually associated with left ventricular enlargement. Sometimes, the degree of left ventricular dysfunction is mild, with a variable enlargement, similar to that found in top endurance athletes, such as cyclists, cross-country skiers, rowers, long-distance runners, etc. (Table III)²³.

In uncertain cases, ECHO or radionuclide ventriculography can be useful to evaluate left ventricular systolic function on exertion. If ejection fraction does not increase or even falls during effort, this may be suggestive of DCM or of a “non-physiologic” left ventricular enlargement²⁴.

Risk assessment. In patients with DCM it is not easy to establish the influence of sports activity in increasing the risk of hemodynamic deterioration or sudden cardiac death. Additional further data for a better risk assessment can be obtained at:

- EST, to evaluate accurately BP pattern on exertion, ventricular arrhythmias or symptoms, if any, and total workload (or VO_2 max, when performing a cardiopulmonary test);
- HM, to define the type and complexity of ventricular arrhythmias, if any.

Thus, even in the absence of data from specific trials, it is reasonable to define at low risk of sudden cardiac death or cardiovascular events those patients with DCM who show:

- no symptoms (particularly syncope);
- no family history of sudden cardiac death;
- normal increase in BP on exertion;
- only a mild left ventricular dysfunction, however recovering on exertion;
- no significant supraventricular or ventricular arrhythmias at HM²⁵ (see specific section).

Indications. Subjects with a definite diagnosis of DCM should not participate in competitive sports of any type.

A possible exception might be represented by subjects classified at low risk, after a close and tailored evaluation, based on the criteria stated above, carried out by expert health professionals. In this case, eligibility can be granted to sports of minimum to moderate cardiovascular involvement of group B2, some of group B1 (horse riding, sailing), and non-competitive athletic activities of group A, with a strict and serial evaluation.

Arrhythmogenic right ventricular cardiomyopathy

ARVC is a primary myocardial disease of the right ventricle and is characterized by myocardial cell loss of varying degree, replaced by adipose or fibrous tissue, and by ventricular arrhythmias, sometimes life-threatening²⁶. In Italy, this cardiac disease plays a relevant role in Sports Medicine, since it is one of the most common causes of sudden cardiac death in young athletes^{27,28}.

Diagnosis. Useful diagnostic findings are:

- family history of ARVC and/or premature sudden cardiac death^{26,29,30};
- clinical history of syncope or palpitations, usually exercise-related. It may be found in the minority of patients and clinical findings are often unremarkable³¹;
- ECG: it often shows one or more abnormalities, such as negative T-waves in the right precordial leads ($V_1 - V_3$) in subjects > 12 years and without RBBB, QRS duration > 0.11 s in the right precordial leads, epsilon waves and/or ventricular late potentials, VPB, even complex, with a LBBB morphology²⁶;
- ECHO: it shows sometimes right ventricular morphologic and functional anomalies, such as global enlargement and systolic dysfunction or, more commonly, regional morphologic abnormalities, such as free wall thinning and sacculation with impaired regional motion^{26,31};
- magnetic resonance imaging and magnetic resonance angiography: besides the above-mentioned right ventricular abnormalities, it can provide additional information on the presence and distribution of adipose tissue^{32,33}.

The diagnosis of ARVC is not always easy, especially in the initial phases of the disease or in the forms with segmental right ventricular anomalies. The diag-

Table III. Differential diagnosis between dilated cardiomyopathy (DCM) and the athlete’s heart.

	DCM	Athlete’s heart
Left ventricular systolic function	Depressed (EF < 50%)	Normal (EF > 50%)
Regional wall motion abnormalities	Present	Absent
Ventricular geometry	Altered	Normal
DCM family history	Often present	Absent

EF = ejection fraction.

nosis should be made on multiparametric criteria and carried out by expert health professionals.

Indications. The risk of sudden cardiac death in subjects with ARVC, in particular when practicing sports, is difficult to evaluate because of the lack of reliable data from prospective trials.

However, it is well known that sudden cardiac death may be the first clinical episode of this disease³⁴. For this reason, subjects with a definite diagnosis of ARVC should not participate in competitive sports of any type.

As for subjects with ventricular arrhythmias with LBBB morphology, but without any echocardiographic or magnetic resonance evidence of morphologic and functional right ventricular abnormalities suggestive of ARVC, see the specific section.

Other cardiomyopathies

In Italy, as well as in most Western countries, less common types of cardiomyopathy are endomyocardial fibroelastosis with or without hypereosinophilia, and systemic heart diseases such as sarcoidosis and amyloidosis^{2,35}. These pathologies can be diagnosed based on symptoms and signs (venous congestion, edema, dyspnea), and echocardiographic findings (cardiac hypertrophy, increased myocardial echo-reflectivity, impaired compliance).

These cardiomyopathies contraindicate participation in competitive sports of any type.

The nosographic classification of conduction system disturbances of degenerative etiology is still debated, although growing evidence supports the causal role of genetic anomalies (hence the importance of studying patients' relatives)³⁶. These disturbances usually cause marked sinus bradyarrhythmias, sometimes associated with supraventricular tachyarrhythmias (brady-tachy syndrome), advanced nodal AV block, and delayed ventricular activation involving one or more branches of the His bundle (see Arrhythmias section).

Myocarditis

Myocarditis is defined histologically as an inflammatory process involving the myocardium with an inflammatory infiltrate and myocyte necrosis or damage of non-ischemic origin^{2,37}.

The incidence of myocarditis in athletes is probably higher than formerly estimated due to the variability in the clinical picture and to the fact that non-invasive diagnostic criteria are non-specific. In Sports Medicine, the infective forms, mostly of viral etiology, are the most relevant³⁸.

Diagnosis. Useful diagnostic findings are:

- in acute, overt myocarditis, the typical symptoms are

chest pain, palpitations, and effort dyspnea, usually accompanied by an elevation in inflammation markers. However, the clinical course may often be paucisymptomatic (fatigue, transient elevation of the body temperature, tachycardia, sporadic palpitations) and therefore may be easily underestimated by the athlete³⁹;

- physical examination: in the acute forms, cardiac auscultation can reveal a third or fourth sound, and sometimes an apical murmur due to MR;

- ECG: it can demonstrate various morphologic anomalies, such as low-voltage QRS in the precordial leads, ventricular repolarization abnormalities, atrial and/or ventricular arrhythmias, delayed AV conduction and ventricular activation³⁹. The frequent occurrence of arrhythmias makes HM mandatory;

- ECHO: in the overt forms, it can demonstrate global left ventricular systolic dysfunction with an associated left ventricular enlargement. The degree of dysfunction and enlargement may be mild and/or regional (more often involving apical) and may recover upon resolution of the myocarditis. A useful contribution can be provided by magnetic resonance imaging (when performed by expert personnel!).

The availability of histologic criteria (Dallas criteria) increases the diagnostic accuracy³⁷⁻⁴¹. However, endomyocardial biopsy should be performed only when the clinical diagnosis is of high probability and the diagnostic confirmation is necessary for therapeutic and medical-legal reasons.

Indications. Subjects with a definite diagnosis of myocarditis cannot participate in competitive sports of any type until complete recovery and in all cases not before 6 months from the onset of the disease. After this time interval, in the absence of any biohumoral and clinical signs, patients can be re-evaluated in order to assess the morphologic and functional outcome and/or arrhythmias, if any.

Eligibility to competitive sports can be considered if the clinical evaluation and non-invasive tests show normal contractility and no significant arrhythmias (see specific section). However, athletes should undergo serial re-evaluation carried out by expert health professionals.

Pericarditis

Pericarditis is defined as an inflammatory pericardial process usually involving the subepicardial myocardial layers.

The incidence of this disease in young athletes is quite high and more often is of viral etiology. The clinical course is usually short and clinical recovery is reached in 1 or 2 weeks; however the complete biological healing may take a longer time. Furthermore, relapses are not rare (recurrent pericarditis), especially within 6-12 months of the first episode⁴².

In some forms of pericarditis, considerably more common in the past, such as tubercular pericarditis, the biological evolution of the inflammatory process may cause fibrosclerosis of the pericardial layers, resulting in chronic constrictive pericarditis.

Diagnosis. Useful diagnostic findings are:

- symptoms: they are characterized by acute chest pain and fever usually accompanied by an elevation in inflammation markers. Sometimes, the clinical course can be insidious, with stabbing chest pain, fatigue, and transient fever⁴²;
- physical examination: it can reveal pericardial or pleuro-pericardial rubs, associated with faint cardiac tones, usually with significant pericardial effusion;
- ECG: it can show ventricular repolarization abnormalities (diffuse elevation of the ST-segment followed by inverted T-waves) mimicking an acute myocardial infarction⁴³;
- ECHO: in the acute phase, it can demonstrate pericardial effusion, even mild, associated with an increased reflectivity and thickening of the pericardial layers.

Indications. Subjects with a definite diagnosis of pericarditis cannot participate in competitive sports of any type until complete recovery and in all cases not before 6 months from the onset of the disease (after this time interval, recurrences are less frequent).

In the absence of any clinical and biochemical signs, sports eligibility can be re-evaluated. Athletes can return to competitive sports if the clinical and non-invasive tests are normal. In case of a remarkable myocardial involvement (myocardio-pericarditis), indications regarding myocarditis will be applied.

References

1. Report of the WHO/ISFC task force on the definition and classification of cardiomyopathies. *Br Heart J* 1980; 44: 672-3.
2. Richardson P, McKenna W, Bristow M, et al. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the Definition and Classification of Cardiomyopathies. *Circulation* 1996; 93: 841-2.
3. Maron BJ. Hypertrophic cardiomyopathy. *Curr Probl Cardiol* 1993; 18: 639-704.
4. Maron BJ, Gardin JM, Flack JM, Gidding SS, Kurosaki TT, Bild DE. Prevalence of hypertrophic cardiomyopathy in a general population of young adults. Echocardiographic analysis of 4111 subjects in the CARDIA Study. Coronary Artery Risk Development in (Young) Adults. *Circulation* 1995; 92: 785-9.
5. Maron BJ, McKenna WJ, Danielson GK, et al. Task Force on Clinical Expert Consensus Documents. American College of Cardiology; Committee for Practice Guidelines. European Society of Cardiology. American College of Cardiology/ European Society of Cardiology clinical expert consensus document on hypertrophic cardiomyopathy. A report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines. *J Am Coll Cardiol* 2003; 42: 1687-713.
6. Chung MW, Tsoutsman T, Semsarian C. Hypertrophic cardiomyopathy: from gene defect to clinical disease. *Cell Res* 2003; 13: 9-20.
7. Spirito P, Chiarella F, Carratino L, Berisso MZ, Bellotti P, Vecchio C. Clinical course and prognosis of hypertrophic cardiomyopathy in an outpatient population. *N Engl J Med* 1989; 320: 749-55.
8. Nienaber CA, Hiller S, Spielmann RP, Geiger M, Kuck KH. Syncope in hypertrophic cardiomyopathy: multivariate analysis of prognostic determinants. *J Am Coll Cardiol* 1990; 15: 948-55.
9. Savage DD, Seides, Clark CE, et al. Electrocardiographic findings in patients with obstructive and non-obstructive hypertrophic cardiomyopathy. *Circulation* 1978; 58: 402-8.
10. Maron BJ, Wolfson JK, Cirò E, Spirito P. Relation of electrocardiographic abnormalities and patterns of left ventricular hypertrophy identified by 2-D echocardiography in patients with hypertrophic cardiomyopathy. *Am J Cardiol* 1983; 51: 189-94.
11. Shapiro LM, McKenna WJ. Distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy: a two-dimensional echocardiographic study. *J Am Coll Cardiol* 1983; 2: 437-44.
12. Maron BJ, Gottdiener JS, Epstein SE. Patterns and significance of distribution of left ventricular hypertrophy in hypertrophic cardiomyopathy. *Am J Cardiol* 1981; 48: 418-28.
13. Maron BJ, Pelliccia A, Spataro A, Granata M. Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes. *Br Heart J* 1993; 69: 125-8.
14. Maron BJ, Spirito P, Wesley Y, Arce J. Development and progression of left ventricular hypertrophy in children with hypertrophic cardiomyopathy. *N Engl J Med* 1986; 315: 610-4.
15. Maron BJ, Cecchi F, McKenna WJ. Risk factors and stratification for sudden death in patients with hypertrophic cardiomyopathy. *Br Heart J* 1994; 72: S13-S18.
16. Ciampi Q, Betocchi S, Lombardi R, et al. Haemodynamic determinants of exercise-induced abnormal blood pressure response in hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2002; 40: 278-84.
17. Savage DD, Seides SF, Maron BJ, Myers DJ, Epstein SE. Prevalence of arrhythmias during 24-hour electrocardiographic monitoring and exercise testing in patients with obstructive and non obstructive hypertrophic cardiomyopathy. *Circulation* 1979; 59: 866-75.
18. McKenna WJ, Chetty S, Oakley CM, Goodwin JF. Arrhythmia in hypertrophic cardiomyopathy. Exercise and 48-hour ambulatory electrocardiographic assessment with and without beta adrenergic blocking therapy. *Am J Cardiol* 1980; 45: 1-5.
19. Spirito P, Maron BJ. Relation between extent of left ventricular hypertrophy and occurrence of sudden cardiac death in hypertrophic cardiomyopathy. *J Am Coll Cardiol* 1990; 15: 1521-6.
20. Dec GW, Fuster V. Medical progress: idiopathic dilated cardiomyopathy. *N Engl J Med* 1994; 331: 1564-75.
21. Grunig E, Tasman JA, Kucherer H, Franz W, Kubler W, Katus HA. Frequency and phenotypes of familial dilated cardiomyopathy. *J Am Coll Cardiol* 1998; 31: 186-94.
22. Wilensky RL, Yudelman P, Cohen AI, et al. Serial electrocardiographic changes in idiopathic dilated cardiomyopathy confirmed at necropsy. *Am J Cardiol* 1988; 62: 276-83.
23. Pelliccia A, Culasso F, Di Paolo FM, Maron BJ. Physiologic left ventricular cavity dilatation in elite athletes. *Ann Intern Med* 1999; 130: 23-31.
24. Tomai F, Ciavolella M, Crea F, et al. Left ventricular vol-

- umes during exercise in normal subjects and patients with dilated cardiomyopathy assessed by first-pass radionuclide angiography. *Am J Cardiol* 1993; 72: 1167-71.
25. Grimm W, Christ M, Bach J, Muller HH, Maisch B. Noninvasive arrhythmia risk stratification in idiopathic dilated cardiomyopathy: results of the Marburg Cardiomyopathy Study. *Circulation* 2003; 108: 2883-91.
 26. McKenna WJ, Thiene J, Nava A, et al. Diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy. Task Force of the Working Group Myocardial and Pericardial Disease of the European Society of Cardiology and of the Scientific Council on Cardiomyopathies of the International Society and Federation of Cardiology. *Br Heart J* 1994; 71: 215-8.
 27. 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: 129-33.
 28. 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: 1959-63.
 29. Nava A, Thiene G, Canciani B, et al. Familial occurrence of right ventricular dysplasia: a study involving nine families. *J Am Coll Cardiol* 1988; 12: 1222-8.
 30. Coonar AS, Protonotarios N, Tsatsopoulou A, et al. Gene for arrhythmogenic right ventricular cardiomyopathy with diffuse nonepidermolytic palmoplantar keratoderma and woolly hair (Naxos disease) maps to 17q21. *Circulation* 1998; 97: 2049-58.
 31. Scognamiglio R, Rahimtoola SH, Thiene G, et al. Concealed phase of familial arrhythmogenic right ventricular cardiomyopathy (ARVC): early recognition and long-term follow-up. (abstr) *J Am Cardiol* 1997; 29: 194A.
 32. Ricci C, Longo R, Pagnan L, et al. Magnetic resonance imaging in right ventricular dysplasia. *Am J Cardiol* 1992; 70: 1589-95.
 33. Keller DI, Osswald S, Bremerich J, et al. Arrhythmogenic right ventricular cardiomyopathy: diagnostic and prognostic value of the cardiac MRI in relation to arrhythmia-free survival. *Int J Cardiovasc Imaging* 2003; 19: 537-43.
 34. Corrado D, Basso C, Thiene G, et al. Spectrum of clinicopathologic manifestations of arrhythmogenic right ventricular cardiomyopathy/dysplasia: a multicenter study. *J Am Coll Cardiol* 1997; 30: 1512-20.
 35. Wynne J, Braunwald E. *Cardiomiopatie e miocarditi*. In: Braunwald E, ed. *Malattie del cuore*. Trattato di medicina cardiovascolare. V edizione italiana. Padova: Piccin, 1998: 1953.
 36. Probst V, Kyndt F, Allouis M, Schott JJ, Le Marec H. Genetic aspects of cardiac conduction defects. *Arch Mal Coeur Vaiss* 2003; 96: 1067-73.
 37. Maisch B, Bultman, Factor S, et al. WHO consensus conference on definition of inflammatory cardiomyopathy (myocarditis): report from two expert committees on histology of viral cardiomyopathy. *Heartbeat* 1999; 4: 3.
 38. Hyypia T. Etiological diagnosis of viral heart disease. *Scand J Infect Dis Suppl* 1993; 88: 25-31.
 39. Peters NS, Poole-Wilson PA. Myocarditis - continuing clinical and pathologic confusion. *Am Heart J* 1991; 121: 942-7.
 40. James KB, Lee K, Thomas JD, et al. Left ventricular diastolic dysfunction in lymphocytic myocarditis as assessed by Doppler echocardiography. *Am J Cardiol* 1994; 73: 282-5.
 41. Aretz HT, Billingham ME, Edwards W, et al. Myocarditis. A histopathologic definition and classification. *Am J Cardiovasc Pathol* 1987; 1: 3-14.
 42. Lorell BH. *Malattie del pericardio*. In: Braunwald E, ed. *Malattie del cuore*. Trattato di medicina cardiovascolare. V edizione italiana. Padova: Piccin, 1998: 2057.
 43. Kouvaras G, Soufras G, Chronopoulos G, et al. The ST seg-

ment axis as a differential diagnostic feature between acute pericarditis and acute inferior myocardial infarction. *Angiology* 1990; 41: 207-12.

SYSTEMIC HYPERTENSION

Definition and classification

According to the most recent WHO-ISH guidelines, systemic hypertension is defined as a systolic BP ≥ 140 mmHg and/or a diastolic BP ≥ 90 mmHg in subjects not taking antihypertensive drugs¹⁻³. The greatest innovation is the growing importance given to systolic BP whose measurement is now considered as valuable as that of diastolic BP to the purpose of classification. The present classification of systemic hypertension in adults is reported in table I.

Obviously, threshold BP values for normotensive and hypertensive subjects are lower in younger age. A recent epidemiologic study regarding a large Italian population aged 5 to 17 years has brought to the conclusion that standard BP values are higher in Italy when compared to those reported in the charts of the American Task Force, especially in the prepubertal age. In particular, in children aged 5 to 12 years, systolic and diastolic BP values were 2-6 mmHg higher in both genders. For this reason, in different age groups, the normal limits should be as follows^{4,5}:

- 6-9 years: < 126/82 mmHg,
- 10-12 years: < 130/82 mmHg,
- 13-15 years: < 136/86 mmHg.

Blood pressure measurement

The diagnosis of hypertension mainly relies upon a correct BP measurement; therefore, in order to avoid errors the following simple rules should be adopted^{6,7}:

- patients should be in the sitting position and should avoid smoking and caffeine for at least 30 min;
- patients should rest for at least 5 min before BP measurement;
- the arm should be free from any clothing, should lay on a comfortable surface and should be kept at the heart level;
- arm cuff should be of the correct length (12-13 cm in children, up to 35 cm in subjects with fat arms) and should cover at least 80% of the arm circumference;
- sphygmomanometer can be standard mercury, aneroid (recently calibrated) or electronic certified;
- arm cuff should be inflated quickly to a BP exceeding systolic BP by 20-30 mmHg; the pace of deflation should be 2-3 mmHg/s;
- systolic and diastolic BP should be measured in both arms (fifth Korotkoff sounds in adults, fourth Korotkoff sounds in children);
- a second measurement should be taken after 2 min, taking into account the mean value.

Table I. Classification of systemic hypertension.

Category	Systolic (mmHg)	Diastolic (mmHg)
Optimal	< 120	< 80
Normal	< 130	< 85
High-normal	130-139	85-89
Grade 1 hypertension (mild)	140-159	90-99
Borderline	140-149	90-94
Grade 2 hypertension (moderate)	160-179	100-109
Grade 3 hypertension (severe)	≥ 180	≥ 110
Isolated systolic hypertension	≥ 140	< 90
Borderline	140-149	< 90

From 1999 World Health Organization-International Society of Hypertension guidelines¹.

Even when complying with these rules, measured BP might not reflect the actual BP of that subject. This is mainly due to two reasons: the “alarm reaction” to BP measurement (due to emotional causes, so-called “white coat hypertension”) and the spontaneous BP variability. With the aim at avoiding a normal individual to be labeled as hypertensive, three strategies are currently available: to re-evaluate the subject by serial visits over a long time interval (3-6 months), to perform the self-measurement of BP at home, or to undergo an ambulatory 24-hour recording^{7,8}.

In athletes, only the first and last options are of use, in that the self-measurement of BP is not reliable and, moreover, sports eligibility should be granted or rejected over a short time.

Current monitoring devices allow for a continuous BP measurement with the subject attending his normal daily life. Many different devices are available for ambulatory recording (auscultatory, oscillometric or mixed) but only those validated according to the criteria stated by the International Society of Hypertension should be employed. The parameters to be considered are: mean 24-hour BP, mean daytime BP, and mean night-time BP. Unfortunately, the interpretation of the results is partially challenged by the lack of reference values of normality widely accepted. Moreover, one should recall that BP values resulting from self-measurement and ambulatory recording are lower than those measured in the clinical setting (BP values 125/80 mmHg by ambulatory recording are equal to BP values 140/90 mmHg in the clinical setting).

Last, there is also the possibility of recording BP invasively, directly “on field” during sports. Because of the high costs and complexity, such a procedure should be limited to selected cases⁹.

Etiology and target organ damage

In case of rest BP values ≥ 140/90 mmHg, sports physicians should ascertain whether the subject is really hypertensive, search for any causes of secondary hyper-

tension and additional cardiovascular risk factors, target organ damage, and associated clinical conditions.

- Ascertain the existence of a real hypertensive condition. This goal can be achieved, in the first instance, by performing a correct measurement following the rules described above. Then, it is necessary to perform an accurate clinical evaluation, including family and personal history, a thorough physical examination, and a laboratory screening as follows:

- blood test (glycemia, total and fractionated cholesterol, serum creatinine and/or blood urea nitrogen, potassium and sodium);
- urine analysis;
- ECG;
- ECHO;
- fundoscopy.

This protocol represents a mandatory premise before further specific tests.

- Search for any causes of secondary hypertension. From the etiologic point of view, in most cases hypertension is not caused by a specific condition, and is therefore defined as idiopathic or essential. In few cases, however, hypertension is caused by definite conditions (secondary hypertension). The most common causes of secondary hypertension are:

- congenital or acquired heart diseases (ACo, aortic regurgitation);
- neuroendocrine diseases (hyperthyroidism, pheochromocytoma, etc.);
- nephrovascular and nephroparenchymal diseases (glomerulonephritis, fibrodysplasia, renal artery stenosis, etc.);
- exogenous factors, such as a large intake of sodium, alcohol, liquorice abuse;
- drugs and substances (estrogenic, steroids, sympathetic agonists, erythropoietin, cocaine, amphetamines).

- Assess additional risk factors, target organ damage, and associated clinical conditions. In hypertensive subjects risk stratification is of paramount importance. In accordance with the most recent guidelines, it should be carried out by measuring systolic and diastolic BP and assessing additional risk factors, target organ damage, and associated clinical conditions (Table II).

Table II. Ten-year absolute risk of cardiovascular events.

Additional risk factors, TOD, ACC	Blood pressure (mmHg)		
	Grade 1 SBP 140-159 or DBP 90-99	Grade 2 SBP 160-179 or DBP 100-109	Grade 3 SBP ≥ 180 or DBP ≥ 110
None	Low risk	Medium risk	High risk
1-2 risk factors	Medium risk	Medium risk	Very high risk
≥ 3 risk factors, TOD or diabetes	High risk	High risk	Very high risk
ACC	Very high risk	Very high risk	Very high risk

Low risk < 15%; medium risk 15-20%; high risk 20-30%; very high risk > 30%. ACC = associated clinical conditions; DBP = diastolic blood pressure; SBP = systolic blood pressure; TOD = target organ damage. From 1999 World Health Organization-International Society of Hypertension guidelines¹, modified.

- *Major risk factors:* degree of hypertension (Table I); age (> 55 years for men, > 65 years for women); cigarette smoking; hypercholesterolemia; diabetes mellitus; cardiovascular family history.

- *Target organ damage:* left ventricular hypertrophy*; proteinuria and/or mild increase in serum creatinine (1.2-2.0 mg/dl); diffuse or focal narrowing of retinal arteries; atherosclerotic lesions in the carotid, aortic, iliac, femoral arteries documented at ECHO and X-ray.

- *Associated clinical conditions:* cerebrovascular diseases; heart diseases (coronary artery diseases, heart failure); renal diseases (diabetic nephropathy, chronic renal disease); atherosclerotic vasculopathies; hypertensive retinopathy.

Sports eligibility

- Secondary hypertension: sports eligibility is subordinated to the resolution of the underlying disease.
- Essential hypertension: general criteria for granting sports eligibility should be defined and established on the basis of the different types of sports.

In this view, EST is of considerable value. It should be performed on the cycloergometer or treadmill, with continuous and increasing workloads up to the maxi-

mal workload or 85% of the maximal age-predicted heart rate. According to recent guidelines, BP values should be considered as abnormal when > 240/115 mmHg on exertion and/or if they do not return to baseline values within 6 min of exercise interruption. However, as for systolic BP, the value of 240 mmHg should be considered as a merely conventional limit. Though EST remains essential in athletes, ambulatory BP monitoring may be a useful diagnostic tool in subjects with high BP values at rest even if, as stated above, results should be interpreted with caution.

- In hypertensive subjects with high or very high cardiovascular risk (Table II), eligibility to competitive sports of any type should be rejected.

In hypertensive subjects with low cardiovascular risk: eligibility to competitive sports of any type can be granted provided that systolic BP values at EST are < 240 mmHg and return to baseline values within 6 min of exercise interruption.

- In hypertensive subjects with medium cardiovascular risk: eligibility to competitive sports can be granted provided that systolic BP values at EST are < 240 mmHg and return to baseline values within 6 min of exercise interruption. However, eligibility to extremely demanding sports, even if short-lasting, should be rejected, in particular to those with a constant and significant "pressure" cardiovascular workload, such as weight lifting, body building, etc.

- In hypertensive subjects at low or medium cardiovascular risk with an abnormal BP pattern at EST (systolic BP values > 240 mmHg, not returning to baseline values within 6 min after exercise interruption): eligibility to competitive sports will be subordinated to the establishment of a good BP control, at rest and on effort, by drug treatment. However, eligibility can be granted for 6 months, and BP should be checked serially in order to verify the effect of exercise and the efficacy of therapy. Moreover, athletes should subscribe a statement testifying the assumption of the prescribed drugs.

- Hypertensive subjects to whom competitive sports eligibility has been rejected, non-competitive athletic activities of group A (with minimum to moderate aerobic

* One peculiar aspect concerning hypertensive athletes is the differentiation of pathologic hypertrophy, secondary to hypertension, from the "physiologic" hypertrophy caused by physical training. The ECG can provide some useful indications: besides high-voltage QRS, athletes often present with sinus bradycardia and/or other bradyarrhythmias, high T-waves, an ECG pattern of "early repolarization", and intraventricular conduction delay involving the right bundle branch. On the other hand, hypertensive subjects often show atrial wave abnormalities, QRS alterations with positivity of the Sokolow and Lewis indexes and of the more recent "Perugia score", left-axis deviation with intraventricular conduction delay involving the left bundle branch (intrinsic deflection in leads I-V₅-V₆ > 0.04 s). Even more important is ECHO and its correct interpretation: in hypertensive subjects, at least in the early phases, wall thickening increases, especially involving the interventricular septum without a proportional increase in the dimensions of the ventricular chamber (increased mass to volume ratio). In the uncertain cases, the study of the diastolic function can be discriminating (assessed by radionuclide techniques) since it is always impaired in pathologic hypertrophy.

cardiovascular involvement) can be allowed with therapeutic aims, pending a thorough evaluation of the whole clinical picture.

Antihypertensive treatment and sports activity

As stated before, to the purpose of eligibility to competitive sports, antihypertensive treatment can be allowed, even though some drugs are prohibited by national and international regulations (for instance, beta-blockers in shot sports and diuretics)^{10,11}.

The choice of the ideal drug for hypertensive athletes should consider the hemodynamic impact, metabolism, and athletic performance. In fact, the aim of medications in sports medicine is to normalize BP without worsening athletic performance. The ideal drug should not:

- depress the cardiac response to exertion;
- be arrhythmogenic;
- interfere with the physiologic blood supply to the muscles;
- interfere with normal utilization of energy sources.

Nowadays, such "ideal" drugs are available: ACE-inhibitors, angiotensin receptor blockers (sartans), calcium antagonists, especially dihydropyridines^{12,13}.

Physical activity for the prevention and treatment of hypertension

Epidemiologic trials have shown that regular physical activity can lower BP and demonstrated an inverse relation between BP and physical activity^{14,15}. At first BP reduction seems unremarkable, but it is not far from that obtained in many pharmacological trials^{16,17}.

The most widely accepted hypothesis over the years is that exercise acts by causing relevant modifications in the neurovegetative system¹⁸⁻²⁰. One of the most important is the physiologic reduction of the sympathetic tone caused by aerobic training which leads to a reduction in BP and exerts a beneficial effect on many cardiovascular risk factors: tendency to tachycardia, dyslipidemia, decreased glucose tolerance, increased plasma renin activity, and alterations in membrane ionic carriers²¹⁻²⁴.

References

1. 1999 World Health Organization-International Society of Hypertension Guidelines for the Management of Hypertension. Guidelines Subcommittee. *J Hypertens* 1999; 17: 151-83.
2. Chobanian AV, Bakris GL, Black HR, et al, for the National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003; 289: 2560-71.
3. European Society of Hypertension-European Society of Cardiology Guidelines Committee. 2003 European Society of Hypertension-European Society of Cardiology guidelines for the management of arterial hypertension. *J Hypertens* 2003; 21: 1011-53.
4. Menghetti E, Virdis R, Strambi M, et al. Blood pressure in childhood and adolescence: the Italian normal standards. Study Group on Hypertension of the Italian Society of Pediatrics. *J Hypertens* 1999; 17: 1363-72.
5. Ardissino D, Bianchetti M, Braga M, et al. Raccomandazioni sull'ipertensione arteriosa in età pediatrica: il Progetto CHild. *Ital Heart J Suppl* 2004; 5: 398-412.
6. Palatini P, Pessina AC. Tecniche di misurazione della pressione arteriosa. *Ipertensione Oggi* 1986; 1: 27.
7. Palatini P. Ipertensione arteriosa ed attività sportiva. In: Zeppilli P, ed. *Cardiologia dello sport*. Roma: CESI, 2001: 685-712.
8. Vriz O, Mos L, Frigo G, et al. Effects of physical exercise on clinic and 24-hour ambulatory blood pressure in young subjects with mild hypertension. *J Sports Med Phys Fitness* 2002; 42: 83-8.
9. Palatini P, Mos L, Mormino P, et al. Blood pressure changes during running in humans: the beat phenomenon. *J Appl Physiol* 1989; 67: 52-9.
10. Disciplina della tutela sanitaria delle attività sportive e della lotta contro il doping. Legge 14 dicembre 2000, n. 376.
11. Codice antidoping del movimento olimpico. Appendice A - Lista delle classi di sostanze vietate e dei metodi proibiti. 1 settembre 2001-31 dicembre 2002.
12. Palatini P, Bongiovì S, Mario L, Mormino P, Raule G, Pessina AC. Effects of ACE inhibition on endurance exercise haemodynamics in trained subjects with mild hypertension. *Eur J Clin Pharmacol* 1995; 48: 435-9.
13. Dumoulin P. Systemic hypertension and sports practice. Epidemiology and therapeutics. *Rev Prat* 2001; 51: S19-S23.
14. Fagard R, M'Buyamba JR, Staessen J. Physical activity and blood pressure. In: Bulpitt CJ, ed. *Handbook of hypertension*. Vol 6. Epidemiology of hypertension. Amsterdam: Elsevier Science Publishing, 1984: 104.
15. Paffenbarger RS, Thorne MC, Wing AL. Chronic disease in former college students. VIII. Characteristics in youth predisposing to hypertension in later years. *Am J Epidemiol* 1968; 88: 25-32.
16. Gilders RM, Dudley GA. Endurance exercise training and treatment of hypertension. The controversy. *Sports Med* 1992; 13: 71-7.
17. De Plaen JF, Detry JM. Hemodynamic effects of physical training in established arterial hypertension. *Acta Cardiol* 1950; 35: 179.
18. Seals DR, Hagberg JM. The effect of exercise training on human hypertension: a review. *Med Sci Sports Exerc* 1984; 16: 207-15.
19. Arakawa K. Antihypertensive mechanism of exercise. *J Hypertens* 1993; 11: 223-9.
20. Fagard R, Amery A. Physical exercise in hypertension. In: Laragh JH, Brenner BM, eds. *Hypertension*. New York, NY: Raven Press, 1995: 2367.
21. MacKnight JM. Exercise considerations in hypertension, obesity, and dyslipidemia. *Clin Sports Med* 2003; 22: 101-21.
22. Krzesinski JM, Ancion G. Sports and hypertension. *Rev Med Liege* 2001; 56: 306-12.
23. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine position stand. Exercise and hypertension. *Med Sci Sports Exerc* 2004; 36: 533-53.
24. Sachtleben T, Fields KB. Hypertension in the athlete. *Curr Sports Med Rep* 2003; 2: 79-83.

CORONARY ARTERY DISEASE

Introduction

The prevalence of coronary atherosclerosis and the incidence of cardiac ischemic events increase with age. Autopsy examinations of adults who died from sudden cardiac death have often shown coronary atherosclerosis and/or acute coronary lesions with plaque rupture. Conversely, cardiac events in athletes < 30 years are rarely related to such a clinical condition¹. These observations suggest that in subjects > 35-40 years exercise-related sudden death is commonly related to arrhythmic complications of myocardial ischemia induced by exertion associated with significant coronary atherosclerosis or acute progression of a previously "non-significant" lesion²⁻⁶.

These issues should be taken into account in order to choose the better diagnostic tool for assessing myocardial ischemia in adult sportsmen, especially when presenting with cardiovascular risk factors (hypertension, dyslipidemia, smoking, family history of premature cardiovascular events)⁷⁻¹². Recently, in the prognostic stratification of subjects with cardiovascular risk factors, the evaluation of the overall cardiovascular risk has been added (see Hypertension section). To the purpose of competitive sports eligibility this means that exercise ECG must always be performed, preferably in pharmacologic washout, by means of EST in subjects > 40 years with no cardiovascular risk factors or at low risk, and in those < 40 years at medium or high risk¹³⁻¹⁹.

Finally, it should be pointed out that, in recent years, requests for sports eligibility are made not only by subjects with ECG abnormalities suggestive of coronary artery disease or without any clinical signs (silent ischemic heart disease) but also by an ever growing number of subjects with known coronary artery disease²⁰⁻²⁴.

Sports eligibility in subjects with known coronary artery disease

This group includes subjects with:

- history of myocardial infarction, confirmed by clinical, ECG and cardiac enzyme data;
- history of angina pectoris, confirmed by symptom-related alterations in ST segment at standard ECG or HM, or by laboratory signs of myocardial ischemia at EST or different provocative tests (ST segment alterations, regional wall motion abnormalities, unequal distribution of radioisotopes, etc.)^{25,26};
- history of myocardial revascularization (coronary artery bypass grafting or angioplasty).

In these cases, the main prognostic factors are:

- degree of left ventricular dysfunction;
- extent of the coronary artery disease;
- possible inducible ischemia;
- electric instability (ventricular arrhythmias)²⁷.

For this reason, it is necessary to evaluate the global ventricular function at rest (ECHO, radionuclide ventriculography, etc.), and to perform a symptom-limited EST in pharmacologic washout and HM. Based on test results, subjects will be divided into two groups:

- mild risk:
 - ejection fraction > 50%;
 - normal exercise tolerance according to age;
 - no inducible ischemia;
 - no ventricular arrhythmias at rest and/or on exertion;
 - no coronary artery stenosis > 50% in the main subepicardial vessels (if coronary angiography has been obtained); and
- high risk:
 - ejection fraction < 50%;
 - myocardial ischemia inducible on exertion;
 - complex ventricular arrhythmias at rest and/or on exertion;
 - significant coronary stenosis (> 50%) involving at least one main subepicardial vessel (if coronary angiography has been obtained).

Subjects at mild risk can be granted eligibility to sports of minimum to moderate cardiovascular involvement of group B2, and non-competitive athletic activities of group A. However, clinical and laboratory examination for risk assessment should be repeated with a tailored schedule, since the extent and progression of the coronary disease may vary over time.

Patients with single-vessel stenosis and good angioplasty results (effective revascularization), not before one year post-intervention, can be re-evaluated to the purpose of eligibility to some sports of moderate to high cardiovascular involvement, including overall risk assessment, besides with mandatory evaluation every 6 months.

Subjects at high risk will not be granted competitive sports eligibility. Recreational sports activities may be suggested in view of a complete psychological and physical rehabilitation and of secondary prevention of coronary artery disease, provided that the clinical picture is accurately evaluated and sports activities are performed under strict control on behalf of sports physicians or experts in cardiac rehabilitation.

Sports eligibility in subjects with silent coronary artery disease

It is well known that ventricular repolarization abnormalities (VRA) at rest and/or on effort are frequent in the athletic population, even in the absence of other clinical and laboratory signs. In top-level athletes, these alterations are related to the degree of conditioning, as they normalize after detraining. However, VRA can also be the hallmark of cardiac structural anomalies (MVP, HCM, ARVC, etc.) and, therefore, a thorough and specific clinical investigation should be performed (ECHO and magnetic resonance imaging when indicated, EST, etc.).

When VRA do not recover, worsen, or even new ones develop on exertion without definite cardiac morphologic alterations, further investigation is needed to rule out coronary artery disease, especially in subjects > 35-40 years and/or with increased cardiovascular risk. Whenever available, it is useful to compare the results of previous ECG at rest, and subjects should be submitted to provocative test contemporary cardiovascular imaging in order to detect regional motion abnormalities. In this case, stress ECHO is the diagnostic tool of choice due to low costs and greater specificity, when compared to radionuclide study of myocardial perfusion. When stress ECHO yields to a positive result, or myocardial perfusion scintigraphy when indicated, coronary angiography should be obtained.

When stress ECHO or myocardial perfusion scintigraphy results are positive, obviously, competitive sports eligibility is rejected, and may be re-evaluated provided that coronary angiography results are negative.

When stress ECHO and/or myocardial perfusion scintigraphy results are negative, sports eligibility may be considered. However, in subjects > 35-40 years or at medium or high cardiovascular risk, sports eligibility should be restricted to a limited time interval (maximum 6 months) by repeating the protocol followed at the first visit.

Coronary artery disease and exercise in a gym setting

In view of the ever growing request of practicing sports and physical exercise, also made by subjects with known coronary artery disease with increased overall cardiovascular risk, the COCIS Committee, regardless of competitive sports eligibility, deemed useful to state some general indications for engaging in the so-called “gym activities” safely.

First one should recall that, besides traditional aerobic exercise (cyclette, treadmill, etc.) included in rehabilitation and secondary prevention programs for patients who had experienced ischemic episodes or had undergone transcatheter or surgical revascularization, at present resistance activities are also allowed with the aim at strengthening the muscle tone, increasing the general well-being, and helping patients to cope with everyday life needs²⁸⁻³⁰.

In the outpatient phase of rehabilitation, experts in cardiac rehabilitation and sports physicians can allow patients to start with or return to “gym activities”, after a close clinical and functional evaluation to determine the type and amount of workloads³¹⁻³³. This evaluation must be tailored to the individual patients and should be carried out in Sports Medicine Centers by specialists with adequate competence able to define the proper “training programs”. Regardless of the individual program, gym activities in these subjects should be based on the following general rules:

- heart rate should always be < 70% of the maximum age-predicted: this parameter should be monitored by telemetric systems or heart rate monitors. In case of a variable cardiovascular and metabolic involvement, as it occurs in non-purely aerobic exercise, the upper reference limit must be lower than that used in the same subject when performing exercise at a constant workload. Moreover, it is recommended to check the BP response;
- muscle overloads should not be high, the force-generating capacity being always within 50% of the maximum voluntary contraction. Series and number of repetitions should be clearly determined, taking care that, during recovery and before the next exercise, heart rate will not exceed baseline values by > 20-30%;
- in case of team activities, when it becomes hard to place correctly the subject with heart disease within a proper homogeneous class, it is recommended to avoid high-intensity exercise (high-impact aerobics, spinning, etc.).

Finally, to the purpose of allowing patients with coronary artery disease or cardiovascular anomalies to exercise in a gym setting, it is recommended that two more fundamental prerequisites are met:

- the gym should be “accredited” to this scope, that is, personnel specifically trained to apply the planned workloads, availability of systems for clinical parameters monitoring, tools and expertise to perform basic life support with defibrillation;
- availability of a Sports Medicine Center or sports physicians able to carry out first evaluation and serial controls and to make tailored exercise schedules.

References

1. European Atherosclerosis Society Consensus Conference Strategy for the prevention of coronary artery disease. A policy statement of the European Atherosclerosis Society. Naples, 1986.
2. Keys A. A multivariate analysis of death and coronary heart disease. Cambridge, MA: Harvard University Press, 1980.
3. Amsterdam EA, Laslett L, Holly R. Exercise and sudden death. *Cardiol Clin* 1987; 5: 337-43.
4. Anversa P, Ricci R, Olivetti G. Effects of exercise on the capillary vasculature of the rat heart. *Circulation* 1987; 75: 112-118.
5. Mittleman MA. Triggering of myocardial infarction by physical activity, emotional stress and sexual activity. In: Willich SN, Muller JE, eds. Triggering of acute coronary syndromes. Dordrecht, Boston, London: Kluwer Academic Publishers, 1996: 71.
6. Hambrecht R, Wolf A, Gielen S, et al. Effect of exercise on coronary endothelial function in patients with coronary artery disease. *N Engl J Med* 2000; 342: 454-60.
7. Kraus H, Raab W. Hypokinetic disease. Springfield, IL: Thomas Publisher, 1961.
8. Cassel J, Heyden S, Bartel AG, et al. Occupation and physical activity and coronary heart disease. *Arch Intern Med* 1971; 128: 920-8.
9. Kannel WB, Sorlie P. Some health benefits of physical activity. The Framingham Study. *Arch Intern Med* 1979; 139: 857-61.

10. Bonanno JA, Lies JE. Effects of physical training on coronary risk factors. *Am J Cardiol* 1974; 33: 760-4.
11. Hartung G, Foreyt JP, Mitchell RE, Vlasek I, Gotto AM Jr. Relation of diet to high-density-lipoprotein-cholesterol in middle-aged marathon runners, joggers and inactive men. *N Engl J Med* 1980; 302: 357-61.
12. Williams P, Wood P, Haskell W, et al. Effect of exercise intensity and duration on plasma lipoprotein cholesterol levels. (abstr) *Circulation* 1981; 64: IV-185.
13. Mitchell JH, Maron BJ, Epstein SE. 16th Bethesda Conference. Cardiovascular abnormalities in the athlete: recommendations regarding eligibility for competition. October 3-5, 1984. *J Am Coll Cardiol* 1985; 6: 1186-232.
14. Thompson PD, Klocke FJ, Levine BD, Van Camp SP. 26th Bethesda Conference. Recommendations for determining eligibility for competition in athletes with cardiovascular abnormalities. Task Force 5: coronary artery disease. *J Am Coll Cardiol* 1994; 24: 888-92.
15. Ward A, Malloy P, Rippe J. Exercise prescription guidelines for normal and cardiac populations. *Cardiol Clin* 1987; 5: 197-210.
16. Deliri MM, Mullins CB. Physiologic effects and importance of exercise in patients with coronary artery disease. *Cardiovasc Med* 1977; 2: 365.
17. Vuori L, Svumakki L, Svumakki T, Kekkonen V. Risk of sudden cardiovascular death in exercise. *Med Science Sport Exer. Proceedings of American College of Sports Medicine*, 1982: 1-14.
18. Maron BJ. The paradox of exercise. *N Engl J Med* 2000; 343: 1409-11.
19. Zeppilli P. *Cardiologia dello Sport*. Roma: CESI, 2001.
20. Garcia-Palmieri MR, Costas R, Cruz-Vidal M, Sorlie PD, Havlik RJ. A protective factor against heart attacks in Puerto Rico. *Am J Cardiol* 1982; 50: 749-55.
21. Rose G. Physical activity and coronary heart disease. *Proc R Soc Med* 1969; 62: 1183-8.
22. Morris JN, Everitt MG, Pollard R, Chave SP, Semmence AM. Vigorous exercise in leisure time: protection against coronary heart disease. *Lancet* 1980; 2: 1207-12.
23. Kannel WB, Belanger A, D'Agostino R, Israel I. Physical activity and physical demand on the risk of cardiovascular disease and death. The Framingham Study. *Am Heart J* 1986; 112: 820-5.
24. May GS, Furberg CD, Eberlein KA, Geraci BJ. Secondary prevention after myocardial infarction: a review of short-term acute phase trial. *Prog Cardiovasc Dis* 1983; 25: 335-59.
25. Todd IC, Ballantyne D. Cardiac adaptations to training: relevance to angina pectoris. *Int J Cardiol* 1986; 10: 91-7.
26. Ehsani AA, Heath GW, Hagberg JM, Sobel BE, Holloszy JO. Effects of 12 months of intensive exercise training on ischemic ST-depression in patients with coronary artery disease. *Circulation* 1981; 64: 1116-24.
27. Schwartz PJ, Billman GE, Stone HL. Autonomic mechanism in ventricular defibrillation introduced by myocardial ischemia during exercise in dog with healed myocardial infarction. An experimental preparation for sudden cardiac death. *Circulation* 1984; 69: 790-800.
28. Hammond HK, Kelly L, Froelicher VF, Pewen W. Use of clinical data in predicting improvement in exercise capacity after cardiac rehabilitation. *J Am Coll Cardiol* 1985; 6: 19-26.
29. Verani MS, Hartung GH, Hoepfel-Harris J, Welton DE, Pratt CM, Miller RR. Effects of exercise training on left ventricular performance and myocardial perfusion in patients with coronary artery disease. *Am J Cardiol* 1981; 47: 797-803.
30. D'Andrea L, D'Andrea A. Possiamo prescrivere al cardiopatico stabilizzato un'attività non protetta in palestra? *Int J Sports Cardiol* 1999; 8: 173-9.
31. Tavazzi L, Ignone G, Giordano A, Giannuzzi P. Cardiac rehabilitation in patients with recent myocardial infarction and left ventricular dysfunction. Hemodynamic aspects. *Adv Cardiol* 1986; 34: 156-69.
32. Giordano A, Giannuzzi P, Tavazzi L. Feasibility of physical training in post-infarct patients with left ventricular aneurysm: a hemodynamic study. *Eur Heart J* 1988; 9 (Suppl F): 11-5.
33. EAMI Study. Attenuation of unfavorable remodeling by long-term physical training in post infarct patients with exertional ischemia and left ventricular dysfunction. (abstr) *Circulation* 1994; 90: 14.

Appendix

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