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ZIPES AND GARSON TASK FORCE 6

Recommendations

1. Athletes with myocardial bridging of an epicardial coronary artery and no evidence of myocardial ischemia at rest or during exercise can participate in all competitive sports.

2. Athletes with myocardial bridging of an epicardial coronary artery and objective evidence of myocardial ischemia should be restricted to low intensity competitive sports (class IA).

Recommendations for other coronary artery anomalies, including anomalous origin of the left main coronary artery, are included in Task Force 1.

References

- Thompson PD, Funk EJ, Carleton RA, Sturner WQ. Incidence of death during jogging in Rhode Island from 1975 through 1980. JAMA 1982;247: 2535-8.
- Willich SN, Lewis M, Lowel H, Arntz H-R, Schubert F, Schroder R. Physical exertion as a trigger of acute myocardial infarction. N Engl J Med 1993;329: 1684–90.
- Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE. Triggering of acute myocardial infarction by heavy physical exertion: protection against triggering by regular exertion. N Engl J Med 1993;329:1677–83.
- Siscovick DS, Weiss NS, Fletcher RH, Lasky T. The incidence of primary cardiac arrest during vigorous exercise. N Engl J Med 1984;311:874-7.
- Thompson PD, Stern MP, Williams P, Duncan K, Haskell WL, Wood PD. Death during jogging or running: a study of 18 cases. JAMA 1979;242:1265-7.
- Northcote RJ, Evans ADB, Ballantyne D. Sudden death in squash players. Lancet 1984;21:148-51.
- Epstein SE, Blomqvist CG, Buja LM, Haskell WL, Ryan TJ, Thompson PD. Task Force V: ischemic heart disease. 16th Bethesda Conference: cardiovascular abnormalities in the athlete: recommendations regarding eligibility for competition. J Am Coll Cardiol 1985;6:1222-4.
- Ambrose JA, Tannenbaum MA, Alexopoulos D, et al. Angiographic progression of coronary artery disease and the development of myocardial infarction. J Am Coll Cardiol 1988;12:56-62.
- Davies MJ. Anatomic features in victims of sudden coronary death. Circulation 1992;85 Suppl 1:1-119-24.
- Black A, Black MM, Gensini G. Exertion and acute coronary artery injury. Angiology 1975;26:759–83.
- Ciampricotti R, EL Gamal MIH, Bonnier JJ, Relik THFM. Myocardial infarction and sudden death after sport: acute coronary angiographic findings. Cathet Cardiovasc Diagn 1989;17:193–7.
- Yasue H, Omote S, Takizawa A, Nagao M, Miwa K, Tanaka S. Circadian variation of exercise capacity in patients with Prinzmetal's variant angina: role of exercise-induced coronary arterial spasm. Circulation 1979;59:938–48.
- Gordon JB, Ganz P, Nabel EG, et al. Atherosclerosis influences the vasomotor response of epicardial coronary arteries to exercise. J Clin Invest 1989;83:1946-52.

- Wilson RF, Marcus ML, Christensen BV, Talman C, White CW. Accuracy of exercise electrocardiography in detecting physiologically significant coronary arterial lesions. Circulation 1991;83:412–21.
- Maron BJ, Roberts WC, McAllister HA, Rosing DR, Epstein SE. Sudden death in young athletes. Circulation 1980;62:218–29.
- Van Camp SP, Peterson RA. Identification of the high risk cardiac rehabilitation patient. J Cardiopulmonary Rehabil 1989;9:103–9.
- Specchia G, De Servi S, Falcone C, et al. Coronary arterial spasm as a cause of exercise-induced ST-segment elevation in patients with variant angina. Circulation 1979;59:948–54.
- Ardissino D, De Servi S, Falcone C, et al. Role of hypocapnic alkalosis in hyperventilation-induced coronary artery spasm in variant angina. Am J Cardiol 1987;59:707–9.
- Pie A, Broustet JP, Saliou B, Gosse P, Guern P. Coexistence of vigorous exercise and heavy smoking in triggering acute myocardial infarction in menunder 35 years--fact or fiction? In: Roskamm H. editor. Myocardial Infarction at Young Age. Berlin, New York: Springer-Verlag, 1981:108–14.
- Delaye J, Beaune J, Delahaye JP. Myocardial infarction at young age during high physical exercise. In ref 19:115–21.
- Miller LW, Schlant RC, Kobashigawa J, Kubo S, Renlund DG. Task Force 5: complications. 24th Bethesda Conference: cardiac transplantation. J Am Coll Cardiol 1993;22:41–54.
- Uretsky BF, Murali S, Reddy PS, et al. Development of coronary artery disease in cardiac transplant patients receiving immunosuppressive therapy with cyclosporine and prednisone. Circulation 1987;76:827–34.
- Uretsky BF, Kormos RJ., Zerbe TR, et al. Cardiac events after heart transplantation: incidence and predictive value of coronary arteriography. J Heart Lung Transplant 1992;11:S45–51.
- Takahashi M. Inflammatory disease of the coronary artery in children. Cor Artery Dis 1993;4:133-8.
- Ledford DK. Immunologic aspects of cardiovascular disease. JAMA 1992; 268:2923-9.
- Paridon SM, Ross RD, Kuhns LD, Pinsky WW. Myocardial performance and perfusion during exercise in patients with coronary artery disease caused by Kawasaki disease. J Pediatr 1992;121:985–6.
- Morales AR, Romanelli R, Boucek RJ. The mural left anterior descending coronary artery, strenuous exercise and sudden death. Circulation 1980;62: 230-7.
- Waller BF, Catellier MJ, Clark MA, Hawley DA, Pless JE. Cardiac pathology in 2007 consecutive forensic autopsies. Clin Cardiol 1992;15:760–5.
- Corrado D, Thiene G, Cocco P, Frescura C. Non-atherosclerotic coronary artery disease and sudden death in the young. Br Heart J 1992;68:601–7.
- Betriu A, Tabau J, Sanz G, Magrina J, Navarro-Lopez F. Relief of angina by periarterial muscle resection of myocardial bridges. Am Heart J 1980;100: 223-6.
- Hill RC, Chitwood WR Jr, Bashore TM, Sink JD, Cox JL, Wechsler AS. Coronary flow and regional function before and after supraarterial myotomy for myocardial bridging. Ann Thorac Surg 1981;31:176-81.

Task Force 6: Arrhythmias

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General Considerations

Guidelines for athletic participation are needed to reduce the risk for arrhythmia-related morbidity or mortality. However, it is often difficult to establish the importance of a cardiac rhythm disturbance in assessing an athlete's eligibility for competition. Few data exist that have been obtained prospectively from well designed, scientifically acceptable studies to determine whether a particular rhythm disturbance predisposes an athlete to sudden death or to symptoms, such as syncope or presyncope, that could precipitate severe injury. Sudden unexpected cardiac death in the young is rare (1,2). It has been estimated that the incidence is <1% of that in adults (3). Nonetheless, a significant proportion of these deaths occur in relation to exercise. The recent deaths of Reggie Lewis and Hank Gathers have focused attention on athletes with known arrhythmias (4).

Arrhythmias commonly are evanescent, often disappearing unpredictably for long periods of time, in some cases years. If they recur when the athlete is not exercising, the arrhythmia may not be noted or may not produce significant symptoms. The same arrhythmia may minimally affect a competitive golfer but severely incapacitate other athletes, such as cross-country skiers performing at peak physical effort. The athlete may not develop the arrhythmia during each sporting event. Although the reasons for this are not known, factors related to the autonomic nervous system probably play a very important role in determining whether an arrhythmia occurs and its rate and effect on hemodynamic responses and symptoms (5). Autonomic "tone" probably varies greatly and perhaps unpredictably between and within athletic events and from one athlete to another. Mental stress during competition can produce important electrophysiologic and hemodynamic changes that are probably mediated through the autonomic nervous system.

It is important to understand the range of normal heart rate and rhythm for the trained athlete (6,7): Heart rates of 25 beats/min and sinus pauses lasting >2 s may be found on 24-h Holter ambulatory electrocardiographic (ECG) recordings. Type I second-degree atrioventricular (AV) block and single uniform premature ventricular complexes each may occur in ~40% of athletes. Complex ventricular arrhythmias (multiform premature ventricular complexes, couplets, nonsustained ventricular tachycardia) are rare (7).

Many of our conclusions result from data obtained in nonathletes, from general perceptions or experience and from a heavy input of "what seems reasonable." Decision making based on this type of logic is often faulty but is the best available. Recommendations and guidance need to be balanced between an effort to avoid restricting activity unduly and the hope of reducing the risk of death and injury due to a rhythm disturbance.

Despite the lack of certain information, some firm conclusions can be reached. Certain arrhythmias create symptoms and are dangerous in and of themselves regardless of the clinical situation in which they occur (8). These arrhythmias generally are characterized by very rapid or very slow heart rates that significantly compromise cardiac output, coronary or cerebral blood flow or maintenance of blood pressure. Such arrhythmias may include atrial flutter or fibrillation with uncontrolled ventricular rates of 200 to 300 beats/min, usually (but not exclusively) in athletes with Wolff-Parkinson-White syndrome, rapid sustained ventricular tachycardias and AV block or sinus node disease with very slow ventricular rates. Certain persistent arrhythmias, such as chronic supraventricular tachycardia, can worsen cardiac function (9). Other arrhythmias, such as AV node reentrant tachycardia, generally well tolerated in most people, may not produce important symptoms at rest but only during exercise in athletes with structural heart disease due, in part, to an increase in the tachycardia rate. Arrhythmias that might otherwise be innocuous and no more than a nuisance might, under conditions of participation in certain sports involving body contact or high speed, place the athlete at risk of injury or death because of transient impaired mental function causing loss of physical control. For example, an athlete with supraventricular tachycardia participating in potentially dangerous sports, such as diving, downhill skiing or auto racing, may be at greater risk because of dizziness, near syncope (a feeling of impending loss of consciousness) or syncope than if he or she were playing basketball or baseball.

Some athletes with coronary heart disease (10), hypertrophic cardiomyopathy (11,12), arrhythmogenic right ventricular dysplasia (13), aortic stenosis (14) and other forms of congenital heart disease (1) are probably at greater risk for cardiac arrest and sudden death during and just after exercise. This is probably true whether or not arrhythmias have been recognized previously (15-17). In general, athletes with symptoms possibly related to cardiac arrhythmia, such as syncope, near syncope and palpitations, should be carefully evaluated before being permitted to participate in competitive sports. A consideration of cardiac hemodynamic status is critical because right or left ventricular dysfunction is an additional important predictor of arrhythmic death (18,19). The presence of a significant rhythm disturbance in athletes with abnormal cardiac hemodynamic status (from any cause unrelated to the arrhythmia), itself is definitely incompatible with participation in all competitive sports. However, it is important to emphasize that some disease states that produce arrhythmias can be self-limited, with subsequent full recovery.

In general, all athletes with significant cardiac arrhythmias being considered for athletic activity should have a 12-lead ECG, echocardiogram, exercise test and a long-term 24-h Holter ambulatory ECG recording, if possible during the specific type of exercise being considered. Resuscitation equipment and trained personnel may be needed on a standby basis for the latter test, which is deemed important because a conventional exercise test may not replicate the specific clinical situation produced by actively participating in the sport. In this regard, exercise tests may need to be adapted specifically for the athlete; that is, to begin exercise at peak energy expenditure, as a sprinter in a race might, rather than with the slow increase in work load commonly used in testing athletes with coronary artery disease.

All athletes with an arrhythmia who are permitted to engage in athletics should be reevaluated at intervals after they are "trained," to determine whether the conditioning process affected the arrhythmia. It should also be stressed that athletes with arrhythmias controlled by antiarrhythmic drugs may stop taking these drugs for a variety of reasons, and therefore compliance with recommended therapy must be established periodically. Coaches and team physicians must continually question players about palpitations, with or without physical activity. Abuse with drugs like cocaine can be responsible for life-threatening arrhythmias, and such considerations are an important part of the evaluation. Of note, the use of certain cardioactive drugs, such as beta-adrenergic blocking agents, is banned in some competitive sports.

It may be difficult for a team physician and consultants from the local community to make objective decisions to restrict or proscribe sports for a competitive athlete. It has been suggested (4) that borderline cases be reviewed by nonbiased experts for the purpose of making these decisions more efficiently. Whether that approach can be practically implemented has not been determined. Alternatively, it has also been suggested that in such borderline cases, both appropriate emergency equipment and medical professionals versed in the operation of that equipment be present at all practices and games. However, there is considerable concern with regard to this practice because its safety and efficacy have not been established. Indeed, the practical implementation and reliability of this approach to reverse potentially lethal arrhythmias in nonhospital settings is highly questionable.

Syncope

Unexplained syncope in an athlete is a potentially important symptom that requires a thorough evaluation. It may be due to a variety of causes, including cardiovascular disease; alternatively, it may be unassociated with structural heart disease but due to mechanisms such as vasovagal syncope, a not uncommon finding in highly trained athletes. Although vasovagal syncope is compatible with continued participation in all competitive sports, caution should be used in making this diagnosis in highly trained athletes without first deficitively excluding underlying structural cardiovascular disease. A cardiac arrhythmia should be considered, particularly when syncope occurs during or immediately after exercise.

A cause of syncope can be established in \sim 50% of patients. Usually, a careful history and physical examination will identify the etiology (20). When such an evaluation does not determine the cause, further testing in search of an arrhythmia is indicated. Ambulatory ECG recordings are often unrevealing but nevertheless probably worthwhile to obtain during the initial evaluation. Event or loop recorders can be used to increase the ECG sampling time. Tilt table testing has been used to assess patients at risk for vasovagal syncope, but the lack of specificity of this test (particularly in endurance-trained athletes) requires a particularly cautious interpretation of the results. Exercise testing is useful and is optimally performed while recording the ECG during the sport activity in which the athlete participates. Invasive electrophysiologic testing is most likely to identify an arrhythmia responsible for syncope in those patients with cardiac disease or an abnormal ECG but should be considered in other athletes when no other cause of the syncope has been identified.

Types of Arrhythmias

Disturbances of Sinus Node Function

Sinus tachycardia and sinus bradycardia appropriate for the clinical situation are not considered abnormal, and no tests are necessary. Sinus arrhythmia and wandering pacemaker are generally considered normal, and no tests are necessary unless the arrhythmias result in inappropriately slow rates accompanicd by symptoms. Sinus arrhythmia and sinus bradycardia are particularly common in the trained athlete.

Asymptomatic sinus pause or sinus arrest <3 s is probably of no significance (7). Longer pauses, sinoatrial exit block and sick sinus syndrome (21) are considered abnormal, and athletes should have a 12-lead and 24-h ECG and an exercise test. In an occasional athlete experiencing syncope or near syncope, an electrophysiologic study may be indicated. Echocardiography should be performed to exclude structural heart disease, and other tests to evaluate ventricular or valvular function may be indicated.

Recommendations

1. Athletes with a normal or structurally abnormal heart in whom the bradycardic rate is increased appropriately by physical activity can participate in all competitive sports. They should be reassessed periodically to determine that training does not aggravate the bradycardia.

2. Athletes with syncope or near syncope should not participate in sports where the likelihood of even a momentary loss of consciousness may be hazardous until the cause has been determined and treated, if necessary.

3. Athletes with symptoms such as impaired consciousness and fatigue clearly attributed to the arrhythmias should be treated and if asymptomatic for 3 to 6 months during treatment, can participate in all competitive sports after physician reevaluation.

4. Athletes with symptomatic tachycardia/bradycardia syndrome or inappropriate sinus tachycardia should be treated. If asymptomatic for 3 to 6 months, they can participate in low intensity competitive sports (class IA [see Table 1 in Classification of Sports]).

5. Athletes with pacemakers should not engage in sports with a danger of bodily collision because such trauma may damage the pacemaker system.

Premature Atrial Complexes

In the absence of evidence obtained from a careful history or physical examination suggesting the presence of structural heart disease, and in the absence of symptoms other than occasional palpitation, evaluation other than a 12-lead ECG is not necessary.

Recommendation. Athletes can participate in all competitive sports.

Atrial Flutter (in the Absence of Wolff-Parkinson-White Syndrome)

In the absence of an acute, limiting illness, sustained atrial flutter is an uncommon rhythm disturbance in athletes without structural heart disease. Therefore, an echocardiogram should be performed to evaluate cardiovascular structure and function. Because the potential for very rapid ventricular rates exists if the atrial flutter conducts 1:1 to the ventricles (22), ECG determination of the ventricular response during an exercise test or athletic event during treatment is essential. For some patients with paroxysmal atrial flutter, induction of the arrhythmia by electrical stimulation may be necessary before the exercise test. A 12-lead and long-term 24-h ECG are also necessary.

Recommendations

1. Athletes with atrial flutter in the absence of structural heart disease who maintain a ventricular rate comparable to that of an appropriate sinus tachycardia during physical activity while receiving therapy with a digitalis glycoside, a beta-blocker or calcium channel blocker, can participate in low intensity competitive sports (class IA), with the warning that rapid 1:1 conduction still may occur. However, full participation in all competitive sports should not be allowed unless the athlete has been without atrial flutter for 3 to 6 months with or without treatment.

2. Athletes with structural heart disease who have atrial flutter can participate in low intensity competitive sports only (class IA) after 6 months have elapsed without an episode of atrial flutter.

Atrial Fibril!ation (in the Absence of Wolff-Parkinson-White Syndrome)

Atrial fibrillation is far more common than atrial flutter and may be present intermittently or chronically (23). Evaluation should include a search for the cause, such as thyrotoxicosis. More often atrial fibrillation occurs in association with diseases such as coronary artery disease or hypertension. Evaluation includes determination of the ventricular response during athletic activity or an exercise test comparable to the intended athletic competition. For some patients with paroxysmal atrial fibrillation, electrical induction of atrial fibrillation before the exercise test may be necessary. A 12-lead ECG is necessary, and long-term 24-h ECG recordings and an echocardiogram are helpful in establishing the presence of structural heart disease.

Recommendations

1. Athletes with atrial fibrillation in the absence of structural heart disease who maintain a ventricular rate comparable to that of an appropriate sinus tachycardia during physical activity while receiving no therapy or therapy with a digitalis glycoside, a beta-blocker or calcium channel blocker can participate in all competitive sports. Note that the use of beta-blockers is prohibited in some competitive sports.

2. Athletes who have atrial fibrillation in the presence of structural heart disease who maintain a ventricular rate comparable to that of an appropriate sinus tachycardia during physical activity while receiving no therapy or therapy with a digitalis glycoside, a beta-blocker or calcium channel blocker can participate in sports consistent with the limitations of the structural heart disease.

3. Athletes who require anticoagulation should not participate in sports with danger of bodily collision.

Sinus Node Reentry and Atrial Tachycardia (in the Absence of Wolff-Parkinson-White Syndrome)

Sinus node reentry and atrial tachycardia, including automatic atrial tachycardia, atrial tachycardia due to reentry, atrial tachycardia with block and chaotic atrial tachycardia, should be evaluated as described for atrial flutter (23).

Recommendations

1. Athletes with sinus node reentry or atrial tachycardia in the absence of structural heart disease who maintain a ventricular rate comparable to that of an appropriate sinus tachycardia during physical activity with or without therapy can participate in all competitive sports.

2. Athletes with underlying structural heart disease can participate only in competitive sports consistent with the limitations of the heart disease.

Atrioventricular Junctional Escape Beats/Rhythm

Atrioventricular junctional escape beats and junctional rhythm are common in athletes. The clinical approach and final recommendations are the same as those given earlier for symptomatic athletes with disturbances of sinus node function (see earlier).

Premature AV Junctional Complexes

If the athlete is asymptomatic except for occasional episodes of palpitations that do not suggest a sustained tachycardia, evaluation need include only a 12-lead ECG. In some athletes, a 24-h ECG recording (during athletic activity if possible), echocardiogram and an exercise test may be indicated.

Recommendations

1. Athletes with a structurally normal heart and a normal heart rate response to activity without evidence of a sustained tachycardia can participate in all competitive sports.

2. Athletes with an abnormal heart, depending on the type and extent of the heart disease, can participate in competitive sports consistent with the limitations of the structural cardiac disease.

Nonparoxysmal AV Junctional Tachycardia

Noninvasive tests required include a 12-lead ECG, echocardiogram, exercise test and 24-h ECG recording during activity. Invasive studies may be necessary for some symptomatic patients or for those with very rapid ventricular rates (24).

Recommendations

1. Athletes without structural heart disease or symptoms who have a controlled ventricular rate that increases and slows appropriately in relation to the level of activity with or without therapy can participate in all sports.

2. Athletes who have no symptoms but who have structural heart disease or incompletely controlled ventricular rates can engage in low intensity competitive sports (class IA), depending on the nature and extent of the structural heart disease and the ventricular rate.

Supraventricular Tachycardia

Atrioventricular node reentry tachycardia and AV reentry over a concealed accessory pathway (with only retrograde conduction) (8) are included in this category. If the diagnosis of the supraventricular tachycardia cannot be made with certainty, and if other clinical therapeutic circumstances warrant it, invasive electrophysiologic studies may be indicated. It is important to identify the rate response of the supraventricular tachycardia during exercise. If the exercise does not induce the tachycardia, attempts to induce the supraventricular tachycardia (possibly with atrial or esophageal pacing) may be useful, followed once again by an exercise test performed by the athlete when the supraventricular tachycardia has been initiated.

Recommendations

1. Athletes who are asymptomatic and have reproducible exercise-induced supraventricular tachycardia in whom prevention of recurrences by therapy has been demonstrated by appropriate testing can participate in all competitive sports.

2. Athletes who do not have exercise-induced supraventricular tachycardia but experience sporadic recurrences should be treated. However, because of the unpredictable nature of the tachycardia, end points for adequate therapy may be difficult to achieve; but once established, these athletes can participate in all sports. Asymptomatic athletes who have episodes of supraventricular tachycardia of 5 to 10 s that do not increase in duration during exercise can participate in all sports.

3. Athletes with syncope, near syncope or significant palpitations secondary to arrhythmia or who have significant structural heart disease in addition to the arrhythmia should not participate in any competitive sports until they have been adequately treated and have no recurrence for ≥ 6 months (4). At that time they can participate in low intensity competitive sports (class IA).

4. For those athletes with successful catheter or surgical ablation (25,26) who are asymptomatic and have no inducible arrhythmia on follow-up electrophysiologic testing (or no recurrence of tachycardia for 3 to 6 months after ablation), all competitive sports are permitted.

Ventricular Pre-Excitation (Wolff-Parkinson-White Syndrome)

Required noninvasive tests include a 12-lead ECG, a 24-h ECG recording during athletic activity, exercise test and echocardiogram to exclude associated cardiovascular abnormalities. Electrophysiologic studies may be indicated in some athletes with palpitations who appear at risk for developing tachycardias, such as those with symptoms of impaired consciousness, long-lasting palpitations or rapid rates (27).

In asymptomatic athletes with no history of palpitations or tachycardia and no evidence of structural cardiac abnormalities, further evaluation is probably not necessary. However, the optimal management for these athletes is uncertain, and continues to be debated (6,28,29). Sudden death in athletes with pre-excitation is rare (28,29), and it appears to be confined largely to those with accessory pathways that have short refractory periods. Therefore, it may be advisable in selected athletes to undertake esophageal pacing or intracardiac electrophysiologic studies to identify the presence of an accessory pathway that has a short refractory period. Atrial fibrillation should be induced and the shortest RR interval assessed. If a short refractory period is found, radiofrequency catheter ablation may be considered. However, in those athletes with a history of palpitations, syncope or near syncope, it is mandatory to assess the functional capabilities and electrophysiologic properties of the accessory pathway (30).

Recommendations

1. Athletes without structural heart disease, a history of palpitations or tachycardia (particularly those >20 years old) can participate in all competitive sports. However, in younger age groups, a more in-depth evaluation may be recommended before allowing participation in moderate to high intensity competitive sports.

2. Athletes with episodes of AV reciprocating tachycardia should be treated as previously recommended (see Supraventricular Tachycardia). However, it should be appreciated that they can develop atrial fibrillation with rapid ventricular rates. Electrical induction of atrial fibrillation to determine the shortest QRS interval between two complexes conducted over the accessory pathway during isoproterenol administration or exercise is recommended.

3. Athletes with episodes of atrial flutter/fibrillation whose maximal ventricular rate at rest (without therapy) as a result of conduction over the accessory pathway is \leq 240 beats/min and who have no episodes of syncope or near syncope appear to be at low risk for sudden cardiac death and can participate in all competitive sports (31). However, despite this apparent risk-stratifying test, those with very rapid ventricular rates probably should be considered for radiofrequency ablation of the accessory pathway. Athletes with syncope or near syncope or episodes of atrial flutter/fibrillation whose maximal ventricular rate at rest (without therapy) as a result of conduction over the accessory pathway exceeds 240 beats/min are restricted to low intensity competitive sports (class IA). They should be considered for radiofrequency ablation of the accessory pathway.

4. Athletes with successful catheter or surgical ablation of the accessory pathway who are asymptomatic and have normal AV conduction and no inducible arrhythmia by follow-up electrophysiologic study or no spontaneous recurrence of tachycardia for 3 to 6 months after ablation can participate in all competitive sports. In selected athletes, the desired participation in sports can be an indication for catheter ablation of the accessory pathway. Noninvasive tests recommended include a 12-lead ECG and exercise test. If there is evidence to suggest the presence of structural heart disease, an echocardiogram is indicated, and a 24-h ECG recording may be beneficial. Even without evidence of structural heart disease, if an increase in the number of premature ventricular complexes or complex ventricular arrhythmias occurs during exercise, further evaluation may be indicated (6,32). In some of these athletes thought to have a structurally normal heart, cardiac catheterization and angiography may reveal otherwise undetected abnormalities, including occult coronary artery disease, congenital coronary anomalies, arrhythmogenic right ventricular dysplasia, cardiac tumor or evidence of cardiomyopathy.

Recommendations

1. Athletes *without* structural heart disease who have premature ventricular complexes at rest, during exercise and exercise testing (comparable to the sport in which they compete) can participate in all competitive sports. Should the premature ventricular complexes increase in frequency during exercise or exercise testing to the extent that they produce symptoms of impaired consciousness, significant fatigue or dyspnea, the athlete can participate in low intensity competitive sports only (class IA).

2. Athletes *with* structural heart disease who are in high risk groups and have premature ventricular complexes (with or without treatment) can participate in low intensity competitive sports only (class IA). Athletes with premature ventricular complexes that are suppressed by drug therapy (as assessed by ambulatory ECG recordings) during participation in the sport can compete in low intensity competitive sports only (class IA).

Ventricular Tachycardia

Nonsustained or sustained monomorphic or polymorphic ventricular tachycardia is always a potentially serious occurrence. Noninvasive tests to be performed include a 12-lead ECG, exercise test, 24-h ECG recording during exercise and echocardiography. Cardiac catheterization and electrophysiologic study should be considered to verify that the heart is structurally normal and to establish the mechanism or location, or both, of the ventricular tachycardia.

Recommendations

1. Athletes with nonsustained or sustained ventricular tachycardia on any of the aforementioned tests generally should not compete in all sports for at least 6 months after the last episode of ventricular tachycardia. This recommendation applies whether the athlete is untreated or treated with either drugs or catheter or surgical ablation. If there have been no clinical recurrences, and ventricular tachycardia is not inducible by exercise or exercise testing and electrophysiologic study, and the athlete has no structural heart disease, all competitive sports may be permitted. For the athlete with structural heart disease and ventricular tachycardia, moderate and high intensity competition is contraindicated regardless of whether the ventricular tachycardia is suppressed. Only low intensity competitive sports (class IA) are permitted.

An exception to this general recommendation is the asymptomatic athlete with brief (generally <8 to 10 consecutive ventricular beats) episodes of nonsustained monomorphic ventricular tachycardia, rates generally <150 beats/min and no structural heart disease established by noninvasive and invasive tests. These athletes do not appear to be at increased risk for sudden cardiac death. If exercise testing (preferably by ambulatory ECG recording during the specific competitive sport) demonstrates suppression of the ventricular tachycardia or no significant worsening compared with baseline, participation in all competitive sports is permissible.

2. Continued athletic competition should not represent the primary indication for an implantable defibrillator. The efficacy with which these devices will terminate a potentially lethal arrhythmia under the extreme conditions of competitive sports is currently unknown. For athletes with implantable defibrillators or antitachycardia devices, all moderate and high intensity sports are contraindicated. Low intensity competitive sports activity (class IA) that does not constitute a significant risk of trauma to the defibrillator is also contraindicated for at least 6 months after the last ventricular arrhythmia requiring intervention (pacing, cardioversion or defibrillation). Subsequently, these athletes can participate in low intensity competitive sports only (class IA).

Ventricular Flutter and Ventricular Fibrillation

Recommendation. Athletes with these conditions that result in cardiac arrest in the presence or absence of structural heart disease cannot participate in any moderate or high intensity competitive sports. However, athletes who have had no episodes of ventricular flutter or ventricular fibrillation for 6 months with treatment may engage in low intensity competitive sports (class IA). Recommendations in the section on ventricular tachycardia also apply.

First-Degree AV Block

If the QRS complex is normal, no further evaluation other than a 12-lead ECG is necessary. If the QRS complex is abnormal, or the PR interval is excessively prolonged (≥ 0.3 s), an exercise stress test, 24-h ECG recording and echocardiogram are indicated, as well as possibly an electrophysiologic study to determine the site and duration of conduction delay.

Recommendation. Athletes who are asymptomatic, without evidence of structural heart disease, in whom the firstdegree AV block does not worsen with exercise, can participate in all competitive sports. If underlying heart disease is present, its nature and severity can independently dictate alternative restrictions.

Type 1 Second-Degree (Wenckebach) AV Block

Wenckebach AV node block can be present in otherwise normal, well trained endurance athletes (7). Recommended tests include a 12-lead ECG, exercise test and echocardiogram. A 24-h ECG recording during athletic activity may be indicated in some athletes. In those athletes with type 1 second-degree AV block and coexisting bundle branch block, electrophysiologic study may be indicated to identify the presence of His-Purkinje Wenckebach block.

Recommendations

1. Athletes with a structurally normal heart and no worsening or actual improvement of AV block with exercise or recovery can participate in all competitive sports.

2. Athless with a structurally abnormal heart in whom AV block disappears or does not worsen with exercise or recovery can participate in all competitive sports, as determined by the limitations of the cardiac abnormality.

3. Athletes without symptoms in whom type 1 seconddegree AV block initially appears or worsens with exercise or during the recovery period should be evaluated further (e.g., for possible intra- or infra-His block) and may require pacemaker therapy. Such athletes can participate in low intensity competitive sports (class IA).

4. Athletes treated with pacemakers should not engage in competitive sports with a danger of bodily collision because such trauma may damage the pacemaker system.

Type 2 Second-Degree (Mobitz) AV Block

The evolution and treatment of this abnormality is considered to be the same as in acquired complete heart block. It should be treated with permanent pacing before any athletic activity (see Congenital Complete Heart Block, recommendations 2 and 3).

Congenital Complete Heart Block

The clinical approach to evaluating the severity of the cardiovascular abnormality includes an echocardiogram, 12-lead ECG, 24-h ECG recording during exercise and exercise stress test (exercise testing should be performed at the same exercise level as that during the sports activity).

Recommendations

1. Athletes with a structurally normal heart and normal cardiac function, no history of syncope or near syncope, a narrow QRS complex, ventricular rates at rest >40 to 50 beats/min increasing with exertion, no or only occasional premature ventricular complexes and no ventricular tachycardia during exertion can participate in all competitive sports (32).

2. Athletes with ventricular arrhythmia, symptoms of fatigue, near syncope or syncope should have a pacemaker implanted before they participate in competitive sports. Athletes with pacemakers should not participate in competitive sports with a danger of bodily collision because such trauma may damage the pacemaker system. Before allowing athletes to engage in these activities, an exercise test should be performed at the level of activity demanded by the particular sport to be certain that the paced heart rate increases appropriately.

3. Athletes with abnormal hemodynamic status, as in an intracardiac shunt, cannot participate in any competitive sports without a pacemaker. Restrictions are the same as those in recommendation 2.

Acquired Complete Heart Block

This condition should be treated with pacing before any athletic activity (see Congenital Complete Heart Block, recommendations 2 and 3). Athletes with a pacemaker should not participate in competitive sports with a danger of bodily collision because such trauma may damage the pacemaker system.

Complete Right Bundle Branch Block

Evaluation includes a 12-lead ECG, 24-h ECG recording, exercise test and echocardiogram.

Recommendation. Athletes without ventricular arrhythmias who do not develop AV block with exercise and who have no symptoms can participate in all competitive sports. This also applies to athletes with associated left-axis deviation.

Complete Left Bundle Branch Block

The evaluation includes a 12-lead ECG, 24-h ECG, exercise test and echocardiogram. Because of the rarity of acquired left bundle branch block in children and its association with syncope from presumed paroxysmal AV block, an invasive electrophysiologic study should be considered in young patients (33).

Recommendations

1. Older athletes with acquired left bundle branch block should follow the recommendations under Complete Right Bundle Branch Block.

2. Athletes with a normal HV interval and a normal AV conduction response to pacing can participate in all competitive sports.

3. Athletes with abnormal AV conduction characterized by an HV interval >90 ms or a His-Purkinje block should have pacemaker implantation. They should be restricted from competitive sports with a danger of bodily collision because such trauma may damage the pacemaker system.

Congenital Long QT Interval Syndrome

The diagnosis of the long QT-QTU syndrome may be complex in some patients (34,35). Although a corrected QT interval (Bazett) of 440 to 450 ms has been used as the upper limit of normal, the diagnosis of long QT syndrome should be made using not only the QT interval but also a history of symptoms, family history and ECG changes, such as T wave alternans or abnormal configuration.

References

- Garson A Jr, McNamara DG. Sudden death in a pediatric cardiology population, 1958 to 1983: relation to prior arrhythmias [abstract]. J Am Coll Cardiol 1985;5 Suppl B:134B-7.
- Vetter VL. Postoperative arrhythmias after surgery for congenital heart defects. Cardiol Rev 1994;2:83–97.
- Driscoll DJ, Edwards WD. Sudden unexpected death in children and adolescents [abstract]. J Am Coll Cardiol 1985;5 Suppl B:118B–21.
- Maron BJ. Sudden death in young athletes: lessons from the Hank Gathers affair. N Engl J Med 1993;329:55–7.
- Counael P. Cardiac arrhythmias and the autonomic nervous system. J Cardiovasc Electrophysiol 1993;4:338–55.
- Zehender M, Meinertz T, Keul J, Just H. ECG variants and cardiac arrhythmias in athletes: clinical relevance and prognostic importance. Am Heart J 1990;119:1378–91.
- Bjornstad H, Storstein L, Meen HD, Hals O. Ambulatory electrocardiographic findings in top athletes, athletic students and control subjects. Cardiology 1994;84:42–50.
- Zipes DP. Specific arrhythmias: diagnosis and treatment. In: Braunwald E, editor. Heart Disease: A Textbook of Cardiovascular Medicine. 4th ed. Philadelphia: Saunders, 1992:667–725.
- Ludomirsky A, Garson A Jr. Supraventricular tachycardia. In: Gillette PC, Garson A Jr, editors. Pediatric Arrhythmias: Electrophysiology and Pacing. Philadelphia: Saunders, 1990;380–426.
- Muller JE, Abela GS, Nesto RW, Tofler GH. Triggers, acute risk factors and vulnerable plagues: the lexicon of a new frontier. J Am Coll Cardiol 1994;23:809–13.
- DeRose JJ Jr, Banas JS Jr, Winters SL. Current perspectives on sudden cardiac death in hypertrophic cardiomyopathy. Prog Cardiovasc Dis 1994; 36:475-84.
- Stewart JT, McKenna WJ. Arrhythmias in hypertrophic cardiomyopathy. J Cardiovasc Electrophysiol 1991;2:516–24.
- Furlanello F, Bettini R, Bertoldi A, et al. Arrhythmias patterns in athletes with arrhythmogenic right ventricular dysplasia. Eur Heart J 1989;10 Suppl D:16-9.
- Michael PL, Mandagout O, Vahanian A, et al. Ventricular arrhythmias in aortic valve disease before and after aortic valve replacement. Acta Cardiol 1992;47:145–56.
- Topaz O, Edwards JE. Pathologic features of sudden death in children, adolescents, and young adults. Chest 1985;87:476-82.
- Vohra J, Sathe S, Warren R, Tatoulis J, Hunt D. Malignant ventricular arrhythmias in patients with mitral valve prolapse and mild mitral regurgitation. PACE 1993;16:387–93.
- Maron BJ, Epstein SE, Roberts WC. Causes of sudden death in competitive athletes. J Am Coll Cardiol 1986;7:204-14.

- Murphy JG, Gersh BJ, Mair DD, et al. Long-term outcome in patients undergoing surgical repair of tetralogy of Fallot. N Engl J Med 1993;329: 593-9.
- Stevenson WG, Stevenson LW, Middlekauff HR, Saxon LA. Sudden death prevention in patients with advanced ventricular dysfunction. Circulation 1993;88:2953-61.
- Kapoor WN. Evaluation and management of the patient with syncope. JAMA 1992;268:2553-60.
- Wang Y-G, Hariman RJ, Wiber DJ, et al. Various electrocardiographic and electrophysiologic presentations of normal and abnormal sinus node. J Cardiovasc Electrophysiol 1992;3:187–95.
- Garson A Jr, Allendet JH, Baron PJ, et al. Atrial flutter in the young: a collaborative study of 380 cases [abstract]. Pediatr Cardiol 1984;4:307.
- Stanton MS, Miles WM, Zipes DP. Atrial fibrillation and flutter. In: Zipes DP, Jalife J, editors. Cardiac Electrophysiology. From Cell to Bedside. Philadelphia: Saunders, 1990:735-42.
- Bharati S, Moskowitz WB, Scheinman M, Estes NAM III, Lev M. Junctional tachycardias: anatomic substrate and its significance in ablative procedures. J Am Coll Cardiol 1991;18:179-86.
- Gursoy S, Schluter M, Kuck K-H. Radiofrequency current catheter ablation for control of supraventricular arrhythmias. J Cardiovasc Electrophysiol 1993;4:194–205.
- Jackman WM, Beckman KJ, McClelland JH, 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.
- Jackman WM, Wang X, Friday KJ, et al. Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. N Engl J Med 1991;324:1605–11.
- Furlanello F, Bertoldi A, Bettini R, Dallago M, Vergara G. Life-threatening tachyarrhythmias in athletes. PACE 1992;15:1403–11.
- Munger TM, Packer DL, Hammill SC, et al. A population stury of the natural history of Wolff-Parkinson-White syndrome in Olmsted County, Minnesota, 1953–1989. Circulation 1993;87:866–73.
- Wiedermann CJ, Becker AE, Hopferwieser T, Muhlberger V, Knapp E. Sudden death in a young competitive athlete with Wolß-Parkinson-White syndrome. Eur Heart J 1987;8:651–5.
- Leitch JW, Klein GJ, Yee R, Murdock C. Prognostic value of electrophysiology testing in asymptomatic patients with Wolff-Parkinson-White pattern. Circulation 1990;82:1718–23.
- Karpawich PP, Gillette PC, Garson A Jr, Hesslein PS, Porter C-B, Mc-Namara DG. Congenital complete atrioventricular block: clinical and electrophysiologic predictors of need for pacemaker insertion. Am J Cardiol 1981;48:1098-102.
- Gillette PC. Syncope in a young girl with left bundle branch block. Pediatr Cardiol 1979;1:75-6.
- Garson A Jr, MacDonald D II, Fournier A, et al. The Long QT syndrome in children: an international study of 287 patients. Circulation 1993;87:1866– 72.
- Schwartz PJ, Locati E, Priori SG, Zaza A. The long QT syndrome. In ref 23:589-604.