

# The Athletic Preparticipation Evaluation: Cardiovascular Assessment

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Thousands of young athletes receive preparticipation evaluations each year in the United States. One objective of these evaluations is to detect underlying cardiovascular abnormalities that may predispose an athlete to sudden death. The leading cardiovascular causes of sudden death in young athletes include hypertrophic cardiomyopathy, congenital coronary artery anomalies, repolarization abnormalities, and Marfan syndrome. Because these abnormalities are rare and difficult to detect clinically, it is recommended that family physicians use standardized history questions and examination techniques. Athletes, accompanied by their parents, if possible, should be asked about family history of cardiac disease and sudden death; personal cardiac history; and exercise-related symptoms, specifically syncope, chest pain, and palpitations. The physical examination should include blood pressure measurement, palpation of radial and femoral pulses, dynamic cardiac auscultation, and evaluation for Marfan syndrome. Athletes with "red flag" signs or symptoms may need activity restriction, special testing, and referral if the diagnosis is unclear. (*Am Fam Physician* 2007;75:1008-14. Copyright © 2007 American Academy of Family Physicians.)



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This article is one in a series on sports medicine created in collaboration with the American Medical Society for Sports Medicine. Coordinators of the series are Francis O'Connor, M.P.H., COL, MC, USA, Virginia Sports Medicine Institute, Fairfax, and Karl (Bert) Fields, M.D., Moses Cone Family Physician Residency and Sports Medicine Fellowship, Greensboro, N.C.

**T**he preparticipation physical evaluation includes screening for issues that may affect an athlete's health and safety (e.g., medication, illicit drug, and supplement use; illness; eating disorders; obesity; vision or hearing problems). The American Academy of Family Physicians has joined a number of organizations in endorsing guidelines to help physicians perform adequate preparticipation evaluations.<sup>1</sup>

Cardiovascular abnormalities that contribute to nontraumatic sudden deaths are estimated to occur in one out of 200,000 young athletes per year.<sup>2-4</sup> Many of these abnormalities have no symptoms or signs, which compounds the difficulty in detecting these conditions. Only a fraction of athletes with serious cardiac anomalies are diagnosed before sudden death.<sup>2-4</sup> Despite the

elusive nature of these disorders, a carefully performed cardiovascular history and physical examination are recommended during preparticipation evaluations.<sup>1</sup> Participation eligibility for athletes diagnosed with cardiovascular abnormalities can be judged further using guidelines from the 36th Bethesda Conference consensus panel.<sup>3</sup>

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Because cardiovascular abnormalities are difficult to detect, family physicians should use standardized history questions and physical examination techniques to maximize the effectiveness of the preparticipation evaluation. The leading cardiovascular causes of nontraumatic exertional sudden death in young athletes are listed in *Table 1*,<sup>2,4</sup> and common clinical clues are identified in *Table 2*.<sup>2,4,5</sup>

## SORT: KEY RECOMMENDATIONS FOR PRACTICE

<i>Clinical recommendation</i>	<i>Evidence rating</i>	<i>References</i>
A careful personal and family history and physical examination designed to identify cardiovascular conditions in young athletes should be included in preparticipation examinations.	C	1, 2
Athletes with a murmur that becomes softer with squatting or louder or longer with standing or during a Valsalva maneuver should be evaluated for hypertrophic cardiomyopathy and mitral valve prolapse.	C	1, 9
Routine screening with noninvasive tests, such as echocardiography, exercise stress testing, and electrocardiography, is not recommended.	C	1-3
Athletes with suspicious cardiovascular examination findings or a history of unexplained exercise-related symptoms (e.g., syncope, presyncope, chest pain) after initial testing should be restricted from athletic participation pending further cardiologic evaluation.	C	1, 3, 15
Athletes with stage 2 hypertension (i.e., blood pressure above the 99th percentile [based on age, sex, and height] plus 5 mm Hg; or blood pressure more than 160/100 mm Hg for athletes older than 18 years) should be restricted from participation until hypertension is controlled.	C	8, 18

*A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, see page 957 or <http://www.aafp.org/afpsort.xml>.*

### CLINICAL HISTORY

The clinical history (*Table 3*<sup>1</sup>) detects more abnormalities than the physical examination and is more likely to prompt physicians to limit athletic participation.<sup>5,6</sup> When possible, the athlete's parents should be involved in answering history questions.<sup>7</sup> If any of the history questions yields an affirmative response, further questioning and evaluation are recommended.

### PHYSICAL EXAMINATION

The four components of the preparticipation cardiovascular evaluation are blood pressure measurement, palpation of radial and femoral pulses, dynamic cardiac auscultation, and evaluation for Marfan syndrome.<sup>1-3</sup> Blood pressure should be measured using an appropriately sized cuff at the brachial artery while the patient is seated.<sup>1,8</sup>

Palpation of the radial pulse allows for quick evaluation of the patient's heart rate and rhythm. A normal arterial pulse has a smooth, rapid upstroke; a smooth summit; and a more gradual downstroke. *Table 4* summarizes abnormal arterial pulsations and associated conditions.<sup>9,10</sup> Radial and femoral pulses should be palpated simultaneously to screen for coarctation of aorta. If the femoral pulse is diminished or delayed relative to the radial pulse, further evaluation is needed.

The cardiac assessment should be performed in a quiet setting to facilitate auscultation. The assessment should include a brief visual inspection of the precordium to evaluate for asymmetry and abnormal impulses. Precordial palpation can detect thrills, abnormal apical impulse location, and parasternal heave. The physician then should carefully auscultate for splitting of first and second heart sounds (i.e., S<sub>1</sub> and S<sub>2</sub>), extra heart sounds, and murmurs. S<sub>2</sub> is composed of closure of the aortic valve followed by closure of the pulmonic valve. Inspiration causes increased blood flow into the pulmonary vascular bed, slightly delaying closure of the pulmonic valve. This widening of S<sub>2</sub> ("physiologic split") is a reassuring sign during an athlete's cardiac examination. A "fixed split" of S<sub>2</sub> can be a sign of an atrial septal defect, and a "paradoxical split" (i.e., narrowing on inspiration) can be a sign of severe aortic stenosis, hypertrophic cardiomyopathy, or left bundle branch block.<sup>9</sup>

**Table 1. Cardiovascular Causes of Sudden Death in Young Athletes**

Hypertrophic cardiomyopathy
Coronary artery anomalies
Commotio cordis (i.e., blunt trauma to the chest causing ventricular fibrillation)
Left ventricular hypertrophy
Myocarditis
Marfan syndrome (i.e., aortic root dilatation, aneurysm and subsequent rupture)
Arrhythmogenic right ventricular cardiomyopathy
Tunneled coronary artery
Aortic stenosis
Dilated cardiomyopathy
Myxomatous mitral valve degeneration
Mitral valve prolapse
Drug abuse
Long QT syndrome
Cardiac sarcoidosis
Brugada syndrome (a genetic disorder of myocardial sodium ion channels)

*NOTE: Causes are listed in descending order of prevalence. Information from references 2 and 4.*

**Table 2. Common Etiologies of Sudden Death in Young Athletes**

<i>Condition</i>	<i>Historical features</i>	<i>Physical examination findings</i>
Aortic stenosis	Personal history of exercise-induced chest pain, breathlessness, light-headedness, syncope, or dizziness	Constant apical ejection click; harsh systolic ejection murmur heard best at the upper right sternal border; crescendo-decrescendo murmur, normally grade 3 murmur or higher
Brugada syndrome (a genetic disorder of myocardial sodium ion channels)	Family history of premature sudden death, particularly in men of Southeast Asian descent	Unremarkable
Coronary artery diseases (congenital or acquired)	Family history of early coronary artery disease, premature sudden death, or coronary anomalies Personal history of exercise-induced chest pain, syncope, or fatigue	Usually normal
Hypertrophic cardiomyopathy	Family history of hypertrophic cardiomyopathy, premature sudden death, recurrent syncope, or lethal arrhythmias requiring urgent treatment Personal history of exertional chest pain or syncope	Wide range of auscultatory findings, from normal examination to a harsh midsystolic murmur that accentuates with standing or the Valsalva maneuver
Long QT syndrome	Family history of premature sudden death Personal history of palpitations or recurrent syncope	Unremarkable
Marfan syndrome	Family history of Marfan syndrome or premature sudden death	See Table 5
Myocarditis	Personal history of fatigue, exertional dyspnea, syncope, palpitations, arrhythmias, or acute congestive heart failure	May be normal Palpable or auscultated extra systoles, third or fourth heart sound gallops, and other clinical signs of heart failure should be considered suspicious
Arrhythmogenic right ventricular cardiomyopathy	Family history of premature sudden death; more common in persons of Mediterranean descent Personal history of palpitations or recurrent syncope	Unremarkable

*Information from references 2, 4, and 5.*

**Table 3. Cardiovascular Screening Questions for the Athletic Preparticipation Examination**

- Have you ever passed out or nearly passed out during or after exercise?
- Have you ever had discomfort, pain, or pressure in your chest during exercise?
- Does your heart race or skip beats during exercise?
- Has a doctor ever told you that you have high blood pressure, high cholesterol, a heart murmur, or a heart infection?
- Has a doctor ever ordered a test for your heart (e.g., electrocardiography, echocardiography)?
- Has anyone in your family died for no apparent reason?
- Does anyone in your family have a heart problem?
- Has anyone in your family died of heart problems or of sudden death before 50 years of age?
- Does anyone in your family have Marfan syndrome?

*Adapted with permission from Preparticipation Physical Evaluation. 3rd ed. Minneapolis, Minn.: McGraw-Hill/Physician and Sportsmedicine, 2005:19.*

Murmurs should be characterized based on timing, location, character, and intensity. Dynamic auscultation (e.g., squat-to-stand and Valsalva maneuvers) can further clarify the murmur type and bring out other murmurs that were not heard initially. Squatting increases venous return to the heart, thereby increasing left ventricular blood volume, chamber size, and stroke volume. In contrast, standing and the Valsalva maneuver decrease venous return, thereby decreasing left ventricular size and stroke volume. Increased stroke volume causes murmurs to become louder with squatting and quieter with standing or the Valsalva maneuver. However, if a murmur is softer when the patient squats or is louder or longer when he or she returns to a standing position or during the Valsalva maneuver, hypertrophic cardiomyopathy or mitral valve prolapse should be suspected.<sup>1,9</sup>

Marfan syndrome is an autosomal dominant disorder of the connective tissue that has characteristic clinical manifestations (Table 5).<sup>1</sup> Aortic root dilatation is common in patients with Marfan syndrome and can cause aortic dissection and sudden death.<sup>1,11</sup> A family history of Marfan syndrome should prompt an evaluation for the disorder because not all persons have a classic presentation. Evaluation also is recommended for males taller than 6 ft (1.83 m) and females taller than 5 ft 10 in (1.78 m) who have two or more physical manifestations of Marfan syndrome. The evaluation includes electrocardiography (ECG), slit-lamp eye examination, and echocardiography to assess the aortic root. The physician should consult with a subspecialist if indicated<sup>1</sup>; a listing of subspecialists by location is available at <http://www.marfan.org>.

**SPECIAL TESTS**

ECG and echocardiography have been proposed to detect underlying cardiac anomalies.<sup>1-4,12,13</sup> However, the low prevalence of these anomalies limits the usefulness of screening tests.<sup>3</sup> Routine use of ECG, echocardiography, or exercise stress testing is not recommended in young athletes.<sup>1-3</sup>

**Indications for Referral or Special Testing**

Athletes with “red flag” signs, symptoms, or physical examination findings may need activity restriction, special testing, or referral.

**RED FLAG SIGNS AND SYMPTOMS**

Patients with exercise-related syncope or presyncope require further evaluation to rule out structural cardiovascular disease.<sup>1,14,15</sup> Syncope or presyncope may indicate left ventricular outflow tract obstruction, arrhythmia, or congenital coronary anomalies. More benign causes include vasovagal syncope, exercise-associated collapse, hypoglycemia, and medication use.<sup>1,3,14</sup> Patients should not participate in athletics during this evaluation.<sup>15</sup> If syncope or presyncope remains unexplained, the patient should be referred to a cardiologist. Symptomatic patients should be evaluated thoroughly before returning to athletic participation.<sup>14-16</sup>

Exercise-induced asthma is a common cause of exertional chest pain in young athletes. Although significant cardiac disease is an uncommon cause,<sup>1</sup> exertional chest pain in this population can be a sign of left ventricular outflow tract obstruction or coronary artery

**Table 4. Clinical Conditions Associated with Abnormal Arterial Pulsation Findings**

<i>Findings</i>	<i>Associated conditions</i>
Large amplitude, rapidly rising pulse, water-hammer pulse	Hypertrophic cardiomyopathy, aortic regurgitation, severe mitral regurgitation, patent ductus arteriosus
Small amplitude, slowly rising pulse (i.e., pulsus parvus et tardus)	Aortic stenosis, low cardiac output (i.e., heart failure)
Alternating strong and weak arterial pulse (i.e., pulsus alternans)	Left ventricular systolic dysfunction
Pulse amplitude decreases or disappears on inspiration (i.e., pulsus paradoxus)	Cardiac tamponade, severe congestive heart failure, severe chronic obstructive pulmonary disease or asthma, constrictive pericarditis
Double-peak pulse (i.e., pulsus bisferiens)	Aortic regurgitation with or without aortic stenosis, hypertrophic cardiomyopathy (may not be palpable)
Abnormal rate or irregular rhythm	Arrhythmia or conduction disturbance

*Adapted with permission from Chizner MA. The diagnosis of heart disease by clinical assessment alone. Dis Mon 2002;48:42, with additional information from reference 10.*

**Table 5. Clinical Manifestations of Marfan Syndrome**

Aortic insufficiency murmur
Arachnodactyly (i.e., long, spidery fingers)
Arm span that is greater than body height
High-arched palate
Kyphosis
Lenticular dislocation
Mitral valve prolapse
Myopia
Pectus excavatum
Thumb sign (i.e., entire nail of the thumb projects beyond the ulnar border of the hand when the hand is clenched without assistance)
Wrist sign (i.e., thumb overlaps last joint of fifth finger when hand is wrapped around opposite wrist)

*Adapted with permission from Preparticipation Physical Evaluation. 3rd ed. Minneapolis, Minn.: McGraw-Hill/Physician and Sportsmedicine, 2005:23.*

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anomalies. In patients with hypertrophic cardiomyopathy, labile outflow tract obstruction that is provoked by changes in preload, afterload, and contractility may cause symptoms only during peak physical exertion or in volume-depleted states.<sup>17</sup> A congenital coronary anomaly, most often a coronary artery that arises from the opposite aortic sinus, should be suspected when an athlete presents with chest pain or syncope. Because of the transient or episodic nature of this ischemia, it often is not detected on static or exercise ECG. Diagnosis requires echocardiography, cardiac magnetic resonance imaging, cardiac computed tomography, or cardiac catheterization.<sup>4</sup>

Palpitations may signify an arrhythmia. The patient history should explore the abruptness of onset, the heart rate, and the regularity of episodes. A gradual onset and relief of palpitations on exertion suggests a sinus tachycardia, whereas a sudden onset of a fast heart rate suggests supraventricular tachycardia, especially if it can be resolved with vagal maneuvers.<sup>9</sup> Palpitations also may be of ventricular origin, suggesting right ventricular outflow tachycardia, arrhythmogenic right ventricular cardiomyopathy, or long QT syndrome. The physician should question athletes with palpitations about their use of tobacco, caffeine, alcohol, over-the-counter medications, supplements, and illicit drugs. If the etiology of palpitations is not clear after a basic work-up (i.e., electrolyte testing, thyroid function testing, and careful ECG evaluation), activity restriction is needed pending further evaluation for malignant arrhythmia with Holter or event monitoring. Consultation with a cardiologist should be considered.

Dyspnea on exertion and easy fatigue are nonspecific symptoms that may simply represent poor conditioning, or they may be initial signs of primary pulmonary hypertension, profound anemia, exercise-induced asthma, or an underlying cardiovascular disorder. If an athlete previously has been diagnosed with hypertension, dyslipidemia, myocarditis, or other cardiac abnormalities, the physician should obtain information on previous participation restrictions. Echocardiography should be performed prior to exercise testing to rule out aortic stenosis and hypertrophic cardiomyopathy.

A family history of premature sudden death (i.e., younger than 50 years) or significant cardiovascular disease is a red flag,<sup>1,12,13</sup> and further details should be sought. A family history of premature sudden death is particularly relevant in identifying hypertrophic cardiomyopathy and is a major risk factor for long QT syndrome, arrhythmogenic right ventricular cardiomyopathy, Marfan syndrome, and coronary anomalies. ECG, echocardiography, exercise stress testing, and lipid panels should be used to evaluate for structural cardiovascular disease. Specialized testing or cardiology referral should be considered.

### PHYSICAL EXAMINATION FINDINGS

Hypertension is defined as an average systolic blood pressure and/or diastolic blood pressure that on at least three occasions is above the 95th percentile for patient sex, age, and height. Updated guidelines on follow-up for hypertension in children and adolescents are summarized in *Table 6*.<sup>8</sup> If an athlete's blood pressure is initially above the 90th percentile, the measurement should be repeated

**Table 6. Recommended Follow-up for Hypertension in Children and Adolescents**

Hypertension classification	Definition*	Follow-up	Athletic participation
Prehypertension	Blood pressure is between the 90th and 95th percentiles	Recheck blood pressure in six months	Full participation is appropriate
Stage 1	Blood pressure is between the 95th and 99th percentiles plus 5 mm Hg	Recheck blood pressure during two additional visits in one to two weeks, or sooner if patient is symptomatic	Participation is appropriate, although the patient should avoid power lifting
Stage 2	Blood pressure is above the 99th percentile plus 5 mm Hg	Refer for immediate evaluation and treatment	Participation restriction is needed until hypertension is controlled

\*—Percentiles based on age, sex, and height.

Adapted with permission from National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* 2004;114(2 suppl):560. Accessed February 2, 2007, at: [http://www.nhlbi.nih.gov/health/heart/hbp/hbp\\_ped.pdf](http://www.nhlbi.nih.gov/health/heart/hbp/hbp_ped.pdf).

twice after the patient sits quietly for at least five minutes.<sup>8</sup> If the blood pressure remains high after these two additional checks, it should be rechecked in six months. If the average blood pressure is above the 95th percentile, the blood pressure should be checked twice in the next one to two weeks, or sooner if the patient is symptomatic. If the blood pressure is still above the 95th percentile, consider referring the patient for further evaluation and possible treatment.<sup>8</sup>

Prompt referral should be made if the patient's blood pressure is above the 99th percentile plus 5 mm Hg (i.e., stage 2 hypertension).<sup>8</sup> In athletes older than

18 years, stage 1 hypertension is defined as blood pressure more than 140/90 mm Hg, and stage 2 hypertension is defined as blood pressure more than 160/100 mm Hg.<sup>18</sup> Stage 2 hypertension or evidence of target-organ damage warrants discontinuation of athletic participation until the hypertension is controlled.<sup>8,18</sup>

Hypertension with a forceful upper extremity pulse and relatively weaker or delayed femoral pulse suggests possible aortic coarctation.<sup>19</sup> Cessation of vigorous athletic participation is advised, and prompt consultation with a cardiologist is needed.

Determining which murmurs are pathologic and which are benign (*Table 7*) is perhaps the most challenging aspect of preparticipation evaluations. Athletes with signs or symptoms of a pathologic murmur should be referred to a cardiologist before returning to athletic participation.<sup>1,9</sup>

**Table 7. Clues to Identifying Benign vs. Pathologic Heart Murmurs**

**Benign murmurs**

- Absence of associated symptoms
- Absence of family history
- Associated with normal, physiologic splitting of S<sub>2</sub>; absence of other abnormal heart sounds (e.g., clicks, gallops)
- Early to midsystolic
- Crescendo-decrescendo murmur
- Musical, vibratory, or buzzing quality
- Normal blood pressure, pulse contour, electrocardiography, or precordial examination
- Often heard best over pulmonic area or mid-left sternal border
- Soft murmur (grade 1 or 2)

**Pathologic murmurs**

- Associated arrhythmia
- Associated left ventricular apical or right ventricular parasternal heave
- Associated with abnormal jugular venous pulse; wide pulse pressure; or brisk, rapidly rising pulse or weak, slowly rising pulse
- Change in intensity with physiologic maneuvers (especially if murmur becomes louder with Valsalva or squat-to-stand maneuvers)
- Diastolic murmur
- Family history of sudden death or cardiac disease
- Long duration (mid- or late-peak or holosystolic murmur)
- Loud murmur (grade 3 or more)
- Other abnormal heart sounds (e.g., loud S<sub>1</sub>, fixed or paradoxically split S<sub>2</sub>, midsystolic click)
- Presence of associated symptoms (e.g., chest pain, dyspnea on exertion, syncope)
- Radiation to axilla or carotids

S<sub>2</sub> = second heart sound; S<sub>1</sub> = first heart sound.

Adapted with permission from Chizner MA. *The diagnosis of heart disease by clinical assessment alone.* *Dis Mon* 2002;48:60.

**Liability**

When making eligibility and disqualification decisions, it is important to follow current clinical guidelines. There often are pressures from athletes, parents, or coaches to maintain athletes' eligibility. However, the law supports limiting high school and college athletes with cardiovascular abnormalities from participation in competitive sports if there are unacceptable medical risks.<sup>20</sup>

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the U.S. Army Medical Department or the U.S. Army Service at large.

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