

# Abnormal ECGs Predict Heart Disease in Athletes

BY KATE JOHNSON  
Montreal Bureau

**E**lectrocardiograms showing marked repolarization abnormalities in young athletes may predict their subsequent development of heart disease, according to Italian researchers.

"Contrary to previous reports describing such ECG patterns as innocent manifestations of 'athlete's heart' without adverse clinical consequences, the present study shows that these abnormal ECGs may represent the initial expression of genetic cardiac disease, preceding by many years phenotypic expression and adverse clinical outcomes," wrote Dr. Antonio Pelliccia from the Institute of Sports Medicine and Science, Italian National Olympic Committee, Rome, and colleagues (N. Engl. J. Med. 2008;358:152-61).

The study included high-level athletes from the Italian Institute of Sports Medicine and Science database, all of whom are required by law to undergo preparticipation screening to rule out the presence of cardiovascular disease.

Of 12,880 athletes screened at the institute between 1979 and 2001, 81 were identified as having sufficient data showing marked repolarization abnormalities without evidence of structural heart disease, over a mean 10 years of serial clinical, ECG, and echocardiographic studies. A control group of 229 athletes of similar age, sex, and duration of follow-up was also selected from the same database. Marked repolarization abnormalities were defined as inverted T waves of 2 mm or more in depth in at least three leads (exclusive of standard lead III), and predominantly in the anterior and lateral precordial leads V<sub>2</sub> through V<sub>6</sub>.

The 81 subjects in the study group included 63 men and 18 women, with a mean age of 23 years at their initial evaluation and 32 years at their most recent assessment. They were most commonly involved in soccer, rowing or canoeing, track and field, swimming, and cycling. They had participated in regular training and competition for a mean duration of 12 years, and 70% had achieved recognition at national or international events, including 14 who had participated in the Olympic Games. Among the 229 control subjects there were 157 men and 72 women who were a mean age of 22 years at initial evaluation. They participated in rowing or canoeing, soccer, water polo, track and field, shooting, and judo, and 80% had reached national or international levels of competition.

In the study group of 81 subjects with abnormal ECGs, evidence of

cardiomyopathy developed in 5 (6%), and evidence of other cardiovascular disorders developed in 6 (7%) during the follow-up period, for a total of 11 subjects (14%), reported the authors. Among the five with evidence of cardiomyopathy, one died at age 24 years (1 year after the initial evaluation) from clinically undetected arrhythmogenic right ventricular cardiomyopathy.

Clinical and phenotypic features of hypertrophic cardiomyopathy developed in three other subjects (at ages 27, 32, and 50 years), including one who survived a cardiac arrest after 16 years of follow-up. The fifth athlete developed dilated cardiomyopathy over a 9-year follow-up.

Among the other six athletes in the study group who developed other cardiovascular conditions, there was systemic hypertension in three, atherosclerotic coronary artery disease (requiring bypass grafting) in one, myocarditis in one, and supraventricular tachycardia (requiring radiofrequency ablation) in one. The remaining 70 (86%) subjects in the study group had unremarkable clinical courses.

In contrast, there was no evidence of cardiomyopathy in any of the athletes in the control group over an average of 9 years of follow-up, and only four (2%) had evidence of other cardiovascular disorders. These included myocarditis in one athlete at age 19 years, 1 year after the initial evaluation; pericarditis in one athlete at age 28 years, 2 years after the initial evaluation; and supraventricular tachycardia in two athletes, identified after 2 and 3 years of follow-up.

"The negative predictive value of a normal ECG was 100% to exclude the development of cardiomyopathy and 98% to exclude the development of any cardiac abnormalities," said the authors. "The positive predictive value of an abnormal ECG was 6% for cardiomyopathy and 14% for any cardiac condition."

Although they noted that ECGs showing marked repolarization abnormalities "may be useful for identifying athletes at risk for the subsequent development of structural heart disease," they suggested that such findings "underscore the importance of greater diagnostic scrutiny and continued clinical surveillance." Serial ECG alone may not be sufficient for such surveillance, they added, suggesting "echocardiography and selective additional testing are necessary to clarify the cardiac diagnosis." On the other hand, a normal ECG "can be regarded as reasonably reliable evidence to exclude the presence of potentially lethal cardiac disease," they concluded. ■

## CLINICAL GUIDELINES FOR PHYSICIANS

### Hyperlipidemia in Children and Teens

BY NEIL S. SKOLNIK, M.D., AND GABOR PERNYESZI, M.D., M.P.H.

**N**ew recommendations by the American Heart Association on the screening and treatment of hyperlipidemia update the National Cholesterol Education Program Expert Panel on Blood Cholesterol Levels in Children and Adolescents of 1992. They are based on new data on the development of atherosclerosis and new treatment studies (Circulation 2007;115:1948-67).

#### Screening

Consider screening with a fasting lipid profile in children who have a family history of coronary disease (that is, a parent or grandparent who developed coronary artery disease, peripheral vascular disease, or cerebrovascular disease, with onset before the age of 55 years).

Children with a parent with high blood cholesterol (great than 240 mg/dL) or who are overweight or obese should be screened for lipid abnormalities. Obese children with lipid abnormalities should also be screened for other aspects of metabolic syndrome (such as insulin resistance and type 2 diabetes, hypertension, or central adiposity). Measurements of creatine kinase (CK), alanine aminotransferase (ALT), and aspartate aminotransferase (AST) should be done before pharmacologic intervention.

#### Treatment Parameters

Drug therapy should be considered in children older than 10 years (although therapy can usually wait until menarche for girls), and after a 6- to 12-month trial of dietary management. Patients should ideally be at Tanner stage 2 or higher. Consider drug therapy if the LDL cholesterol level remains greater than 4.90 mmol/L (190 mg/dL), or if the level is greater than 4.10 mmol/L (160 mg/dL) and there is a positive family history of premature cardiovascular disease, or if two other risk factors are present in the patient after vigorous attempts are made to control the factors.

The treatment goal for LDL cholesterol is at least a level less than 3.35 mmol/L (130 mg/dL), but ideally less than 2.85 mmol/L (110 mg/dL). For children with high-risk lipid abnormalities, the presence of additional risk factors or high-risk conditions lower the LDL cholesterol goals, and starting drug therapy before the child reaches 10 years of age may be appropriate.

Other risk factors could include male gender; a strong family history of premature cardiovascular disease; presence of low HDL cholesterol, high triglycerides, and small dense LDL cholesterol; overweight or obesity and aspects of the metabolic syndrome; and other medical conditions associated with an increased atherosclerotic risk such as diabetes.

#### Treatment

Statins are recommended as first-line therapy. Choice of statin is a matter of preference. Start with the lowest dose given once daily.

The patient should report all potential adverse effects, especially myopathy (muscle cramps, weakness, asthenia, and more diffuse symptoms). If myopathy is present, its relationship to recent physical activity should be assessed, the

medication stopped, and CK assessed. The patient should be monitored for resolution of the myopathy and any associated increases in CK. Consideration can be given to restarting the medication once symptoms and laboratory abnormalities have resolved.

After 4 weeks, measure the fasting lipoprotein profile, CK, ALT, and AST, and compare with laboratory-specific normal values. The threshold for a worrisome level of CK is 10 times above the upper limit of normal; consider the effect of physical activity. The threshold for a worrisome level of ALT and AST is three times above the upper limit of normal.

Advise female patients on concerns relating to pregnancy and the need for contraception. Advise about drug interactions, especially cyclosporine, fibric acid derivatives, niacin, erythromycin, azole antifungals, nefazodone, and many HIV protease inhibitors.

If target LDL cholesterol levels are achieved and there are no laboratory abnormalities, then continue therapy and recheck in 8 weeks and again in 3 months. If laboratory abnormalities are noted or symptoms are reported, temporarily withhold the drug and repeat the blood work in about 2 weeks. When abnormalities return to normal, the drug may be restarted with close monitoring. If target LDL cholesterol levels are not achieved, double the dose, and repeat the blood work in 4 weeks. Continue stepped titration to the maximum recommended dose until target LDL cholesterol levels are achieved or there is evidence of toxicity.

Monitor growth (height, weight, and body mass index), sexual maturation, and development (Tanner staging). Monitor fasting lipoprotein profile, CK, ALT, and AST every 3-6 months. Monitor and encourage compliance with lipid-lowering dietary and drug therapy. Serially assess and counsel for other risk factors, such as weight gain, smoking, and inactivity.

#### The Bottom Line

Appropriate risk factors should trigger lipid screening in children and adolescents. Consider drug therapy where lifestyle modification has not adequately reduced LDL cholesterol levels. Careful monitoring of history and laboratory findings is needed for children on statins.



DR. SKOLNIK is an associate director of the family Medicine Residency Program at Abington (Pa.) Memorial Hospital and a coauthor of "Redi-Reference Clinical Guidelines." DR. PERNYESZI is a second-year resident in the program.

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