Well-trained athletes may have alterations of cardiovascular function which resemble organic heart disease on examination with noninvasive diagnostic techniques. Systolic murmurs, slow heart rates, a variety of arrhythmias and alterations of S-T segments and T waves are all features of the athletic heart syndrome.

We report selected external pulse recordings and vectorcardiograms from two basketball players with features of the athletic heart syndrome. Both subjects had atypical chest pain and physical findings suggestive of heart disease. Complete right and left heart catheterization, ventriculography and selective coronary cineangiography were completely normal in both subjects. Furthermore, there was no evidence of left ventricular subaortic pressure gradient during or after isoproterenol administration and catheter evoked ventricular extrasystoles.

Figure 1 shows the phonocardiograms, external carotid pulse tracing and apexcardiogram from a 29-year-old man (professional basketball player) who had third and fourth heart sounds on cardiac auscultation and a prominent sustained apical impulse on cardiac palpation. In panel A, there is a prominent dicrotic retraction and diastolic wave on the external carotid pulse tracing. A sustained apical systolic wave can be seen on the apexcardiogram shown in panel B. Figure 2 demonstrates the abnormalities of the Frank vectorcardiogram and 12 lead scalar electrocardiogram in this patient. Note the increased QRS voltage suggestive of left ventricular hypertrophy, with ischemic-like S-T segment and T loop abnormalities in the anterolateral and inferior wall leads. The administration of potassium and exercise testing did not resolve these S-T segment and T wave alterations. Figure 3 shows the sagittal plane timed vectorcardiogram from a 31-year-old athlete who had an irregular cardiac rhythm. Marked respiratory sinus arrhythmia was related to unexpected antegrade block of sinus impulses during longer P-P cycles.

Despite repeated demonstration of cardiographic and hemodynamic changes resulting from physical training, there are only a few reports of subjects with the athletic heart syndrome who were studied with cardiac catheterization. The explanation for the graphic abnormalities described here remains obscure. Experimental animals subjected to prolonged exercise develop cardiac hypertrophy with a more pronounced Starling effect.

Electrocardiographic and vectorcardiographic abnormalities previously documented in well-trained athletes include sinus bradycardia, second degree atrioventricular block, QRS loop

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**Figure 1.** Panel A: Simultaneously recorded phonocardiograms, external carotid pulse tracing (CT) and lead 2 (LII) of the electrocardiogram from a 29-year-old basketball player. MA, TA, PA, AA = mitral, tricuspid, pulmonic and aortic areas respectively. Panel B: Simultaneously recorded phonocardiograms, apexcardiogram (ACG) and lead 2 of the electrocardiogram.
changes and "ischemic" S-T segment and T wave alterations. Increased vasal tone consequent to athletic conditioning may play a role in the genesis of the arrhythmias. Redistribution of the potassium within the myocardial cells or a physiologic potassium perfusion resulting from strenuous muscular effort may in part account for chronic or acute electrovectorcardiographic T loop abnormalities.

In conclusion, it appears that further study will be required before the "abnormal" graphic changes in these subjects with apparently "normal" hearts are explained.

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