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Echocardiographic Characteristics of Professional Football Players

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OBJECTIVES
We examined the echocardiographic characteristics of highly trained American football players.

BACKGROUND
Intense physical training is associated with morphologic and physiologic cardiac changes often referred to as the "athlete's heart." Echocardiographic features peculiar to elite football players have not been described.

METHODS
We studied cardiac morphology and function as assessed by rest and stress echocardiography in 156 asymptomatic National Football League players. Resting and stress ejection fraction (EF), wall thickness, and diastolic left ventricular internal diameter (LVID) were measured. Left ventricular (LV) mass was calculated, as was relative wall thickness (RWT) defined as septal and posterior wall thickness divided by LVID. Control data were obtained from published studies.

RESULTS
The mean LVID (53 ± 0.5 mm) and maximal wall thickness (11.2 ± 0.2 mm) were increased over normal reported control subjects. There was a correlation between LVID and body weight (p = 0.01) and body surface area (BSA) (p = 0.01). The average LVID indexed to BSA was 23 ± 2 mm/M². There was also a correlation between maximal wall thickness and body weight (p = 0.01) and BSA (p = 0.01). The average wall thickness indexed to BSA was 5.05 ± 0.88 mm/M². Of the players, 23% had evidence of LV hypertrophy. Two players had an increased septal-to-posterior-wall-thickness ratio (≥1.3), although no player had an outflow gradient. The RWT for the players was 0.424 (± 0.1). The mean resting EF was 58% (± 4.4%), and every player undergoing exercise testing had an appropriate hyperdynamic response in cardiac function.

CONCLUSIONS
Both wall thickness and LVID of elite American football players are increased and correlate with body size. There is a high RWT, reflecting an emphasis on strength training. The LV EF was normal and not supranormal, as is sometimes believed. Regardless of the resting EF, all players had hyperdynamic cardiac responses with exercise. (J Am Coll Cardiol 2003;41:280–4) © 2003 by the American College of Cardiology Foundation

Intense physical training is associated with characteristic changes in cardiac function and morphology that have been termed the "athlete's heart" (1). Physiologic alterations from training include an increased stroke volume and decreased heart rate (2), while morphologic changes include increased left ventricular (LV) cavity dimension, wall thickness, and mass (1,3,4). The extent of morphologic changes varies between sports (5,6), and complicates the differentiation between these normal physiologic alterations and a cardiomyopathy. There is also a misperception among some that the LV ejection fraction (EF) of a well-trained athlete should be supranormal. Although the exercise-induced cardiac changes in athletes participating in various sports have been well described, changes associated with American football have been less well studied. Accordingly, we reviewed the echocardiographic features of 156 professional American football players.

METHODS
Between 1996 and 1999, 1,282 apparently healthy collegiate football players underwent a routine physical exam and electrocardiography as part of their evaluation for selection into the National Football League. No athlete was excluded for any reason. Each of the athletes had performed at an exceptional level and was being assessed for professional employment. Steroid use among the participants was not known. All players had similar training methods emphasizing efforts to improve power and speed rather than endurance, although there was some variability based on the player’s position. The blood pressure in all players was consistently or predominantly <140/90 mm Hg. All subjects were asymptomatic. The 156 athletes undergoing echocardiography had a clinical suspicion of possible heart disease based on mild abnormalities on history, clinical evaluation (often a family history of hypertension), or the electrocardiogram. In other respects they were representative of the elite football players entering the National Football League. Many players with echocardiographic abnormalities did not have an echocardiogram. The players studied underwent M-mode, Doppler, and two-dimensional echocardiography at rest and with exercise using commercially available equipment (Hewlett-Packard, Andover, Massachusetts). Wall thickness, left ventricular internal diameter (LVID) at end-diastole, resting and exerci-
surface area (BSA) were determined by calculation of
between wall thickness, LVID, body weight, and body
ally for wall motion abnormalities. The relationships
predicted. The images were qualitatively evaluated visu-
ton protocol achieving a maximum heart rate of 85%
/determination by two-tailed
tformed utilizing two-stage stress with a modi-
two-chamber views (7). Stress echocardiograms were per-
taken from endocardial contour measured in the apical and
measured by biplane Simpson'
/LVID) and expressed as a fraction (4). Left ventricular mass
was calculated from end-diastolic wall thickness and cavity
dimension using the Penn-cube formula: LVM = 1.04
([LVID + PWT + IVST]³ – LVID) – 13.6 g (8). Left ventricular hypertrophy was considered present when mass
index exceeded 116 g/M² (9). Left ventricular EF was
measured by biplane Simpson’s rule with measurements
taken from endocardial contour measured in the apical and
two-chamber views (7). Stress echocardiograms were
-performed utilizing two-stage stress with a modified Naught-
ton protocol achieving a maximum heart rate of 85%
predicted. The images were qualitatively evaluated visu-
ally for wall motion abnormalities. The relationships
between wall thickness, LVID, body weight, and body
surface area (BSA) were determined by calculation of
Pearson’s correlation collation with statistical significance
determined by two-tailed t testing. Data are presented as
± SE with a p value <0.05 considered significant.
Interobserver reproducibility assessment of the measure-
ments was not available.

RESULTS

The subjects had a mean age of 22 years. The height ranged
from 172 to 201 cm (69 to 79 in.) with a mean 185 cm (73
in.), and their weight ranged from 85 to 157 kg (187 to 347
lbs), with a mean 105 kg (231 lbs). The BSA ranged from
1.95 to 2.84 M², with a mean 2.29 M².

The mean LVID was 53 mm ± 0.5 mm). While over
one-third of the players (43 of 156) had a mildly enlarged
LVID of >55 mm, only 10 players (6%) had markedly
enlarged ventricles (LVID >60 mm) (Fig. 1). There was a
correlation between LVID and body weight (Pearson cor-
relation of 0.468, p = 0.01) as well as between LVID and
BSA (Pearson correlation of 0.479, p = 0.01). The average
LVID indexed to BSA was 23 ± 2 mm/M².

The mean value for maximal wall thickness was 11.2 cm
(± 0.2 cm) (Figs. 2 and 3). There was a correlation between
wall thickness and body weight (Pearson correlation of
0.490, p = 0.01) as well as between wall thickness and BSA
(Pearson correlation of 0.457, p = 0.01). The average wall
thickness indexed to BSA was 5.05 ± 0.88 mm/M².
Seventeen athletes (11%) had a wall thickness >13 mm, and
two players had a wall thickness >15 mm. Both of these
players with markedly thickened walls weighed more than
118 kg (260 lbs) and had LVIDs >55 mm. One of the
athletes had increased wall thickness with a relatively small
LV cavity (wall thickness of 15 mm, LVID of 40 mm).
There was no asymmetry in wall thickness or resting outflow
gradient in that athlete, although this was suggestive of
hypertrophic nonobstructive cardiomyopathy. The mean
septal-to-PWT ratio was 1.02. Only two players had a
markedly increased ratio (≥1.3), and neither of these players
had a murmur or outflow tract gradient.

The mean LVM was 236 g (Fig. 4). Only 3% of players
had an LVM >350 g. The mean LVM/BSA ratio was 103
g/M² (± 20.9 g/M²). Left ventricular hypertrophy was
present in 23% of the athletes (26 of the 113 subjects for
which sufficient information was available to allow calcula-
tion of the LVM). The RWT was 0.424. Thirty-six athletes

Figure 1. Distribution plot of left ventricular diastolic diameter. Six percent of the players had a left ventricular internal diameter (LVID) at end-diastole >6.0 cm. There was a correlation between LVID and body weight (p = 0.01) as well as body surface area (p = 0.01).
(23%) had an RWT >0.44 (considered the upper limit of normal of control subjects).

The mean resting EF was 58% (± 4.4%). A total of 39% of players had an EF of 50% to 55%, although no player had an EF <50% (Fig. 5). All subjects had an appropriate hyperdynamic EF with exertion to an average EF with stress of 76% (± 14%).

While trivial regurgitant valvular abnormalities were common, only five players had moderate regurgitation (four with pulmonic insufficiency and one with tricuspid insufficiency). Two players had mitral valve prolapse, one had mild pulmonic stenosis, and one had a bicuspid aortic valve. No valvular abnormalities were found that would prompt limitation of participation in professional football.

DISCUSSION

Physiologic hypertrophy is a common feature of the “athlete’s heart.” The extent of change in cardiac dimensions varies between athletes and training methods, and adds to the clinical dilemma when attempting to distinguish athlete’s heart from pathologic heart disease (1,4,9,10). Football remains very popular in the U.S., and cardiac adaptations peculiar to training for this sport have not been well described. We intended to assess the cardiac structural changes commonly seen among athletes intensely training and competing in American football.

Although our study did not include cardiac morphology on normal controls, a recent meta-analysis summarized such findings among the control subjects of previous reports (4). Cardiac structure in 78 original studies comparing athletes to control subjects included the findings of over 800 controls, and reported an LVID of 49.6 mm, wall thickness of 8.8 mm, and LVM of 174 g (4). The cardiac morphologies found in our study of football players are similar to average values reported among athletes pursuing a variety of other sports (4,6,11). While both the wall thickness and LV cavity size were increased, the increased LVM among the football players was primarily due to an enlarged LV cavity.

This is an important point, as highly trained competitive athletes (particularly rowers and cyclists) without apparent heart disease can develop markedly thickened ventricular walls that may resemble hypertrophic cardiomyopathy (1,5,12,13). Previous studies have demonstrated that a LV wall thickness of >13 mm was very uncommon among highly trained athletes, and the upper limit to which the ventricle wall thickens with training is 16 mm (11). In large part, our findings on elite American football players have been similar. Although nearly a one-quarter of the subjects would be classified as having evidence of LV hypertrophy, only a small minority had extreme levels of hypertrophy. In

![Figure 2. Maximal wall thickness. The mean value was 11.2 mm (± 0.2 mm). Six percent of players had a wall thickness >14 mm.](image)

![Figure 3. Maximal wall thickness related to player weight. There was a correlation between wall thickness and body weight (p = 0.01) as well as body surface area (p = 0.01).](image)
our study both LVID and wall thickness correlated with body size of the athlete. Six percent of the football players in our series had a wall thickness in the borderline range of >13 mm, but no player had a thickness >16 mm, and no player was found to have a dynamic outflow tract obstruction.

Morganroth et al. (5) first suggested two different morphologic types of athlete’s heart: a strength-trained heart and an endurance-trained heart. The pressure overload states accompanying strength training result in predominantly increased LV wall thickness with little change in LV chamber size. The sustained episodes of high cardiac output and volume overload that occur in endurance training would preferentially increase LV chamber size with less effect on wall thickness. Such divergent cardiac adaptations between static and more dynamic sports were recently confirmed in a large meta-analysis (4). The football players in our series had cardiac changes more similar to other athletes that have emphasized strength training, with a relatively larger increase in LV wall thickness and a slight increase in LVID (4). This is not surprising, as most players emphasize weight lifting during their training, an activity that causes transient large increases in blood pressure (14,15). This training favors more ventricular wall hypertrophy, as reflected in the high RWT among the football players. The RWT of 0.424 among the football players in this series is more than the control patients (0.356) and endurance-trained athletes (0.389) reported in past studies, although not as great as values of RWT reported for classic strength trained athletes (0.442) (4).

Although auscultation of a murmur prompted obtaining the echocardiogram in some of the subjects, most players were found to have only trivial regurgitant valves. There were two cases of mitral valve prolapse, one of bicuspid aortic valve, and one mild pulmonic stenosis, but no lesions were found that would prompt surgical intervention or limitation of activities. A physiologic flow murmur is a well-recognized phenomenon in highly trained athletes.

Left ventricular systolic function is commonly assessed by EF (7). There is a misperception among some that the EF of a well-trained athlete should be supranormal. However, studies suggest this is not the case. The largest study of athletes has shown LV systolic function as assessed by EF or fractional fiber shortening is similar to sedentary control subjects (4). We found similar results, with a large percentage (nearly 40%) of the football players having low normal values between 50% and 55%. All subjects who underwent stress echocardiography had an appropriate hyperdynamic response in systolic function.

In summary, elite American football players in the National Football League have cardiac dimensions similar to other well-trained athletes. There is relatively more hypertrophy of the LV wall as opposed to larger LV chamber size, although both are increased. Both wall thickness and LV chamber size correlate with body size. Resting EFs were normal and not hyperdynamic, but all increased with exercise.
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