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ORIGINAL ARTICLE

One year cardiac follow up of young world cup football team compared to nonathletes

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KEYWORDS

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Football;
Training;
Sudden death

Abstract *Introduction:* Sudden cardiac death among professional young athletes has become a significant concern mainly attributed to structural heart changes and ECG abnormalities.
Objectives: We aimed primarily to compare echocardiographic and electrocardiographic changes in young professional athletes versus a control group of sedentary lifestyled nonathletic individuals of the same age group. Secondly, we aimed to follow up echocardiographic and electrocardiographic changes in young professional athletes after one year.
Methods: We conducted the study from May 2008 to May 2009 by clinical examination, transthoraxic echocardiography and 12 lead ECG. Our study group was the national football team candidates for the youth world cup occurring in Cairo 2009. This study group was compared to a control group of randomly picked nonathletic third year medical students after exclusion of anyone with a known medical illness. The study group was classified into Athletes I representing athletes at the beginning of the study and Athletes II representing athletes after one year follow up.
Results: The Study group comprised 34 males, mean age 18.82 ± 1.56 years while the Control group comprised 28 males, age mean 19.64 ± 2.31 years. There was not a significant difference between the two groups regarding number, age, height or weight ($P > 0.05$).
Athletes I vs control: Clinical parameters showed significantly lower Systolic Blood Pressure SBP (athletes 117.79 ± 6.536, control 126.43 ± 17.043, $P = 0.008$) and Heart Rate HR (athletes 68.88 ± 5.044, control 77.43 ± 6.033, $P = 0.001$). ECG parameters showed a significantly longer RR interval (athletes 0.88 ± 0.065, control 0.76 ± 0.078, $P = 0.001$), while Corrected QTc interval was not significantly different (athletes 0.41 ± 0.029, control 0.42 ± 0.022, $P > 0.05$). Echo parameters showed a significant increase in Ejection fraction EF (athletes 60.94 ± 3.084 vs control

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54.14 ± 13.063, $P = 0.005$) and Left atrial dimension LA (athletes 3.28 ± 0.392 vs control 2.58 ± 1.321, $P = 0.005$). On the other hand Septal wall in diastole SWD, Right ventricle dimension RV, Left ventricular end systolic dimension LVESD, Left Ventricular End Diastolic Dimension LVEDD, Aortic Root AO, and Posterior wall in diastole PWD were not significantly different ($P > 0.05$).

Athletes II vs control: QTc became significantly longer (athletes 0.43 ± 0.028 vs control 0.42 ± 0.022, $P = 0.05$). SWD was significantly thicker (athletes 1.21 ± 0.23 vs control 1.07 ± 0.17, $P = 0.04$). SBP, HR remained significantly lower and RR, EF, LA remained significantly greater ($P < 0.05$), while RV, LVESD, LVEDD, AO, PWD remained not significantly different both at the beginning and also after 1 year ($P > 0.05$).

Athletes I vs athletes II: ECG parameters showed a significant increase in QTc (0.41 ± 0.029 vs 0.43 ± 0.028, $P = 0.005$) and RR interval (0.81 ± 0.167 vs 0.88 ± 0.065, $P = 0.046$). Echo parameters showed a significant increase in SWD (1.21 ± 0.232 vs 0.93 ± 0.124, $P < 0.001$), LA (3.62 ± 0.423 vs 3.28 ± 0.392, $P = 0.001$), RV (2.37 ± 0.565 vs 2.09 ± 0.234, $P = 0.011$), PWD (1.00 ± 0.200 vs 0.90 ± 0.200, $P = 0.008$), and a significant decrease in LVESD (3.19 ± 0.679 vs 3.48 ± 0.190, $P = 0.016$). Other parameters were not statistically significant ($P > 0.05$).

Conclusions: Professional football playing in young males results in significant changes compared to their control of sedentary nonathletic medical students of similar age. Clinical parameters showed a significant decrease in systolic blood pressure SBP and heart rate HR, ECG parameters showed significant increase in RR interval and QTc interval, and Echocardiographic parameters showed a significant increase in Left atrium diameter LA, Septal wall in diastole SWD, and ejection fraction EF. One year of professional football playing in young males causes a continuing significant increase in ECG parameters QTc, RR interval, and echocardiographic parameters SWD, LA, Right ventricle dimension RV, Posterior wall in diastole PWD and decrease in Left ventricular end systolic diameter LVESD compared to themselves one year earlier. The international concern of Sudden cardiac death among professional young athletes may be attributed to Structural heart changes and ECG abnormalities acquired with professional training.

1. Introduction

Regular physical activity has several beneficial effects on health and is linked with reduced cardiovascular and all-cause mortality. However, in certain circumstances, an acute bout of exertion may cause dangerous health effects and, in extreme cases, sudden death (the so called ''paradox of exercise'').

Competitive athletics has been defined as ''participation in an organized team or individual sport that requires regular competition against others as a central component, that places a high premium on excellence and achievement and requires some form of systematic training''.[1]

Although rare, the sudden death of a young athlete is a dramatic event with a devastating impact on his family, sports community, and medical staff. It was the case of professional soccer player Marc-Vivien Foe from Cameroon (28 years old), who died suddenly while playing in the Confederations Cup, due to a hypertrophic cardiomyopathy, or, more recently, of the Spanish soccer player Antonio Puerta (22 years old) who had a syncope on the field, with spontaneous recovery and a new collapse in the locker room due to a fatal cardiac arrest.[2]

Dealing with exercise-related sudden death (ESD), we usually refer to an unexpected and witnessed sudden cardiac arrest occurring within 1 hour from an exercise bout in an apparently healthy person.

Following this definition, ESD is rare, with an incidence of 1/100 000 to 1/300 000 per year in males. This explains why, still today, experts discuss on the usefulness of a routine medical pre-participation screening in preventing ESD.[3]

It is largely demonstrated that cardiovascular diseases play the dominant role in ESD, accounting for 80–85% of cases. Usually, the fatal cardiac arrest occurs either during or immediately after strenuous exercise, suggesting that autonomic nervous system changes related to effort may trigger malignant arrhythmias in subjects with cardiac disease. This aspect is indirectly confirmed by the fact that ESD is more frequent during official events in respect to training sessions, probably because of the greater psychological involvement.Aim of the work:

1. Compare echocardiographic and electrocardiographic changes in young professional athletes versus a control group of sedentary life styled nonathletic individuals of the same age group.
2. Follow up echocardiographic and electrocardiographic changes in those young professional athletes group after one year.
3. Providing limitation criteria for eligibility of competition and suitable sports for athletes at risk for SCD.

2. Patients and methods

This study was conducted in Critical Care Medicine Department Cairo University in the period from May 2008 to May

2009.Our study group was picked up from the national football team candidates for the youth world cup occurring in Cairo 2009. This study group was compared to a control group of randomly picked nonathletic third year medical students after exclusion of anyone with a known medical illness.

The study group was classified into:

[I] Athletes I representing athletes at the beginning of the study and
[I] Athletes II representing athletes after one year follow up.

2.1. Patients

2.1.1. Inclusion Criteria

1. Study group:

Athletes, males, age between 17 and 22 years.
Practicing football in a professional manner.
The study group was examined in the beginning of the study (athletes I) and one year later (athletes II).

2. Control group:
Nonathletic, males, age between 17 and 22 years.

The study group was compared to a control group of randomly picked nonathletic third year medical students.

2.1.2. Exclusion Criteria

Known congenital heart disease.
Implantable cardioverter defibrilator or pacemaker implantation.
Hypertension.
Any chronic disease as liver, kidney, autoimmune,...,etc.

2.2. Methods

2.2.1. All individuals in both groups were subjected to

Thorough history taking: Chest pain, syncopal attacks, palpitation, high blood pressure and family history of sudden cardiac death.

(1) General and clinical examination:

1. Weight: using a standard weight calculating machine to all subjects.
2. Height: Was measured by a standard against the wall measure.
3. Arm span to height ratio and other examinations to exclude Marfan syndrome (ratio more than 1.05 is significant) criteria.

General Clinical Examination including:

1. Blood pressure SPB and DBP
2. Cardiac Auscultation
3. Abdominal examination

(2) Electrocardiogram ECG:

12-lead ECG was done to all subjects to evaluate the cardiovascular condition.
Variables of comparison were:
RR interval: from the R to the next R.

PR interval: from the beginning of the P to the beginning of the QRS.

QT interval: from the beginning of the QRS to the end of T wave.

$$\text{Corrected } QT(\text{QTc}) = \frac{QT \text{ interval in second}}{\sqrt{R} - \text{Rinterval in second}}.$$

(3) Echocardiography:

Transthoracic echocardiography was done to every candidate in both groups at the beginning and to the study group after one year follow up.

The following variables were measured:

Left ventricular end diastole (LVEDD 3.5–5.6 cm)
Left ventricular end systole (LVESD)
Left atrial dimensions (LA 2.5–4 cm).
Ejection fraction (EF more than 50%).
Interventricular septum (IVS 0.6–1.1 cm) in diastole.
Posterior wall thickness (PWd 0.6–1.1 cm) in diastole.

Right ventricle dimention (RV 0.7–2.5 cm).

Aortic root (AO 2.5–4 cm).

2.3. Statistical methods

Data were coded and entered using the statistical package SPSS (Self-Propelled Semi-Submersible) version 15.
Data were summarized using mean and standard deviation for quantitative variables & percent for quantitative variables, compare between groups was done using chi-square for qualitative variables and independent samples T-test for normal distributed quantitative variables while quantitative variables not normally distributed were compared using non-parametrical Mann-Whitney test and Wilcoxon signed rank test.
P-value < 0.05 was considered statistically significant.

3. Results

We conducted the study from May 2008 to May 2009 in the critical care medicine department, Cairo University.

The athletes study group comprised 34 males picked from members of the football youth teams at Al Ahly football club.

This study group was classified into:

1. Athletes I: represented the athletes at the beginning of the study.
2. Athletes II: represented the athletes one year later in follow up.

The control group comprised 28 males randomly picked nonathletic third year medical students after exclusion of anyone with a known medical illness.

Our results will be discussed under the following items:

(A) Baseline and clinical parameters
(B) Electrocardiographic parameters.
(C) Echocardiographic parameters.

(A) Baseline and clinical data:

1. The mean age for athletes was 18.82 ± 1.566 years while it was 19.64 ± 2.313 years in the control group. There was no significant statistical difference between the two groups regarding the age ($P > 0.05$).
2. The mean heig**ht** Ht in athletes was 176.15 ± 5.533 cm while it was 177.04 ± 6.472 cm in the control group without significant statistical difference between the two groups ($P > 0.05$).
3. The mean weight Wt of athletes was 72.29 ± 5.579 kg while in the control group it was 74.82 ± 13.944 kg with no significant statistical difference between the two groups regarding the weight ($P > 0.05$).
4. Body Surface Area BSA in athletes was 1.87 ± 0.1 while it was 1.91 ± 0.180 in control group without significant statistical difference between the two groups ($P > 0.05$).
5. S*ystolic blood pressure* SBP was *significantly lower* in athletes compared to control (117.79 ± 6.536 mmHg vs 126.43 ± 17.043 mmHg, $P = 0.008$), respectively.
6. *Diastolic blood pressure* DBP showed no significant statistical difference between the two groups (74.41 vs 76.79, $P = 0.32$).
7. The mean *Heart Rate* HR in (athletes I) (68.88 ± 5.044) was significantly lower than control (77.43 ± 6.033, $P = 0.001$) (Table 1).

While there was no such difference between athletes II (72.97 ± 12.537) and control group (77.43 ± 6.033, $P = 0.09$), as well as between (athletes I) (68.88 ± 5.044) and athletes II (72.97 ± 12.537) ($P = 0.1$) (Fig. 1).

(B) Electrocardiographic data:

RR interval:

(1) Athletes I vs control: RR interval was significantly longer (0.88 + 0.065 vs 0.76 + 0.078, P 0.001)in athletes I vs control, respectively (Table 3).

Table 1 Baseline and clinical data.

	Athletes (34 pts)	Control (28 pts)	*P*-value
Age	18.82 ± 1.566	19.64 ± 2.313	0.103
Ht	176.15 ± 5.533	177.04 ± 6.472	0.562
Wt	72.29 ± 5.579	74.82 ± 13.944	0.337
BSA	1.87 ± 0.101	1.91 ± 0.180	0.285
SBP	117.79 ± 6.536	126.43 ± 17.043	0.008
DBP	74.41 ± 5.609	76.79 ± 12.488	0.324
HR	68.88 ± 5.044	77.43 ± 6.033	0.001

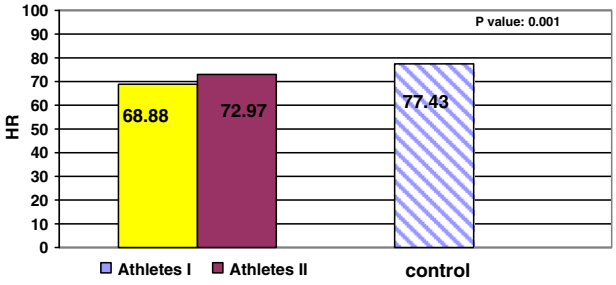

Figure 1 Mean heart rate in study versus control groups.

(2) Athlete II vs control: RR interval was not significantly longer (0.81 ± 0.167 vs 0.76 ± 0.078, P: 0.153) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: RR interval was significantly longer (0.88 + 0.065 vs 0.81 + 0.167, P 0.046) in athletes I vs athletes II, respectively (Fig. 2, Table 2).

PR interval:

(1) Athletes I vs control: PR interval showed no significant statistical difference (149.03 ± 11.821 vs 139.57 ± 27.67) ($P = 0.076$) in athletes I vs control, respectively (Table 3).
(2) Athlete II vs control: PR interval showed no significant statistical difference (143.59 ± 24.87 vs 139.57 ± 27.67

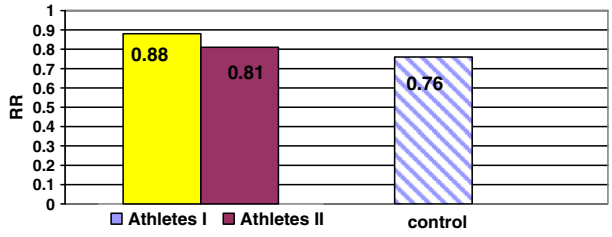

Figure 2 Mean RR interval in study versus control groups.

Table 2 The comparison between athletes I at the beginning of study and athletes II after 1 year of professional sport activity.

	Athletes I (34 pts)	Athletes II (34 pts)	*P*-value
ECG parameters			
HR	68.88 ± 5.044	72.97 ± 12.537	0.1
RR	0.88 ± 0.065	0.81 ± 0.167	0.046
PR	149.03 ± 11.821	143.59 ± 24.87	0.237
QRS	101.15 ± 3.66	102.74 ± 6.561	0.2771
QTc	0.41 ± 0.029	0.43 ± 0.028	0.005
Echo parameters			
SWd	0.93 ± 0.124	1.21 ± 0.232	0.001
PWd	0.90 ± 0.106	1 ± 0.20	0.008
EF	60.94 ± 3.08	60.48 ± 10.53	0.85
LVEDd	4.79 ± 0.507	4.79 ± 1	0.987
LVESd	3.48 ± 0.19	3.19 ± 0.679	0.016
LA	3.28 ± 0.392	3.62 ± 0.423	0.001
AO	2.79 ± 0.329	2.7 ± 0.56	0.228
RV	2.09 ± 0.237	2.37 ± 0.565	0.01

($P = 0.55$), in athletes II vs control, respectively (Table 4).

(3) Athletes I vs athletes II: PR interval showed no significant statistical difference (149.03 ± 11.821 vs 143.95 ± 24.844, $P = 0.237$) in Athletes I vs athletes II, respectively (Fig. 3, Table 2).

The QRS:

(1) Athletes I vs control: The QRS showed no significant statistical difference (101.15 ± 3.661 vs 100.52 ± 5.867 $P = 0.465$) in athletes I vs control, respectively (Table 3).

(2) Athlete II vs control: The QRS showed no significant statistical difference (102.74 ± 6.561 vs 100.25 ± 5.867, $P = 0.125$) in athletes II vs control, respectively (Table 4).

(3) Athletes I vs athletes II: The QRS showed no significant statistical difference (101.15 ± 3.66 vs 102.74 ± 6.561, $P = 0.2771$) in athletes I vs athletes II, respectively (Fig. 4, Table 2).

Table 3 The comparison between athletes I and control.

	Athletes I (34 pts)	Control	*P*-value
ECG parameters			
HR	68.88 ± 5.044	77.43 ± 6.033	0.001
RR	0.88 ± 0.065	0.76 ± 0.078	0.001
PR	149.03 ± 11.821	139.57 ± 27.67	0.076
QRS	101.15 ± 3.66	100.25 ± 5.867	0.465
QTc	0.41 ± 0.029	0.42 ± 0.022	0.327
Echo parameters			
SWd	0.93 ± 0.124	1.07 ± 0.179	0.001
PWd	0.90 ± 0.106	1.07 ± 0.225	0.0001
EF	60.94 ± 3.08	54.14 ± 13.06	0.005
LVEDd	4.79 ± 0.507	4.69 ± 0.635	0.461
LVESd	3.48 ± 0.19	3.48 ± 0.19	0.483
LA	3.28 ± 0.392	2.58 ± 1.321	0.005
AO	2.79 ± 0.329	2.63 ± 0.414	0.108
RV	2.09 ± 0.237	2.27 ± 0.492	0.073

Table 4 The comparison between athletes II after 1 year and control.

	Athletes II (34 pts)	Control	*P*-value
ECG parameters			
HR	72.97 ± 12.537	77.43 ± 6.033	0.09
RR	0.81 ± 0.167	0.76 ± 0.078	0.153
PR	143.59 ± 24.87	139.57 ± 27.67	0.550
QRS	102.74 ± 6.561	100.25 ± 5.867	0.125
QTc	0.43 ± 0.028	0.42 ± 0.022	0.058
Echo parameters			
SWd	1.21 ± 0.232	1.07 ± 0.179	0.012
PWd	1 ± 0.20	1.07 ± 0.225	0.202
EF	60.48 ± 10.53	54.14 ± 13.06	0.04
LVEDd	4.79 ± 1	4.69 ± 0.635	0.638
LVESd	3.19 ± 0.679	3.48 ± 0.19	0.142
LA	3.62 ± 0.423	2.58 ± 1.321	0.001
AO	2.7 ± 0.56	2.63 ± 0.414	0.61
RV	2.37 ± 0.565	2.27 ± 0.492	0.431

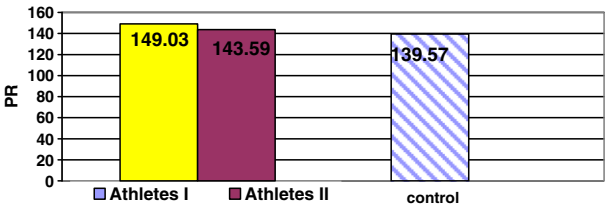

Figure 3 Mean PR interval in study versus control groups.

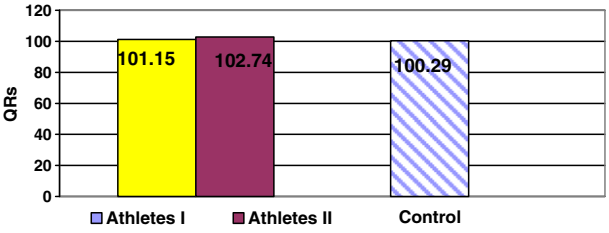

Figure 4 Mean QRS interval in study versus control groups.

QTc:

(1) Athletes I vs control: Corrected QTc interval was not significantly different (0.41 ± 0.029 vs 0.42 ± 0.022, $P > 0.05$) in athletes I vs control, respectively (Table 3).

(2) Athletes II vs control: QTc became significantly longer (0.43 ± 0.028 vs 0.42 ± 0.022, $P = 0.05$) in athletes II vs control, respectively (Table 4).

(3) Athletes I vs athletes II: Showed a significant increase in QTc (0.41 ± 0.029 vs 0.43 ± 0.028, $P = 0.005$) in athletes I vs athletes II, respectively (Fig. 5, Table 2).

(C) Echocardiographic data:
The septal wall in diastole (SWd):

(1) Athletes I vs control: The septal wall in diastole (SWd) was significantly lower (0.93 ± 0.124 vs 1.07 ± 0.179 $P = 0.001$) in athletes I vs control, respectively (Table 3).

(2) Athletes II vs control: SWd was significantly higher (1.21 ± 0.232 vs 1.07 ± 0.179, $P = 0.012$) in athletes II vs control, respectively.

(3) Athletes I vs athletes II: SWd showed a significant increase in (0.93 ± 0.124 vs 1.21 ± 0.232, $P < 0.001$) in athletes I vs athletes II, respectively (Fig. 6).

The posterior wall in diastole (PWd):

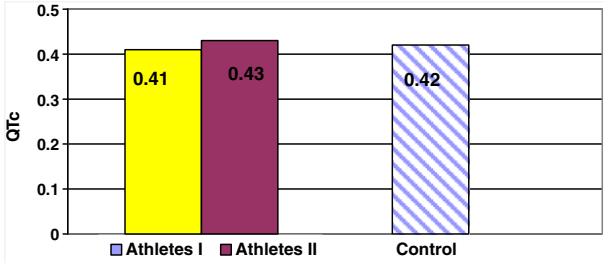

Figure 5 Mean QTc interval in study versus control groups.

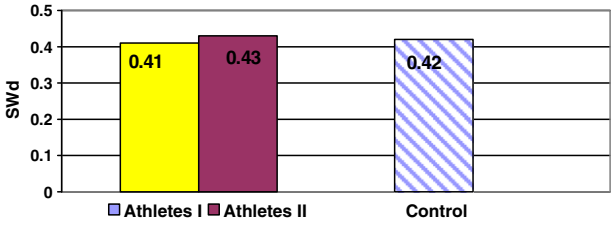

Figure 6 Mean SWd in study versus control groups.

(1) Athletes I vs control: The posterior wall in *diastole* (PWd) was significantly lower (0.90 ± 0.106 vs 1.07 ± 0.225, $p = 0.001$) in athletes I vs control, respectively.
(2) Athletes II vs control: PWd showed no significant statistical difference (1 ± 0.20 vs 1.07 ± 0.225, $P = 0.202$) in athletes II vs control, respectively.
(3) Athletes I vs athletes II: PWd showed a significant increase (0.90 ± 0.106 vs 1 ± 0.20, *P*: 0.008) in athletes I vs athletes II, respectively (Fig. 7, Table 2).

Ejection Fraction:

(1) Athletes I vs control: Ejection fraction EF showed a significant increase (60.94 ± 3.084 vs 54.14 ± 13.063, $P = 0.005$) in athletes I vs control, respectively (Table 3).
(2) Athletes II vs control: EF remained significantly greater (60.48 ± 10.53 vs 54.14 ± 13.06, $P = 0.04$) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: EF was not significantly different (60.94 ± 3.08 vs 60.48 ± 10.53, $P = 0.85$) in athletes I vs athletes II, respectively (Fig. 8, Table 2).

The left ventricular end diastole (LVEDd):

(1) Athletes I vs control: LVEDd showed no significant statistical difference (4.79 ± 0.507 vs 4.69 + 0.635, $P = 0.461$) in athletes I vs control, respectively (Table 3).

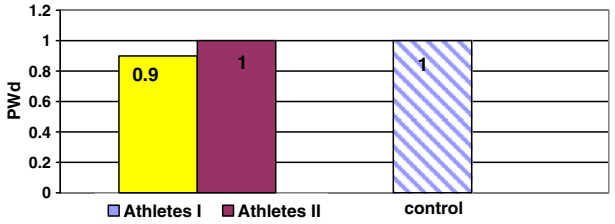

Figure 7 Mean PWd in study versus control groups.

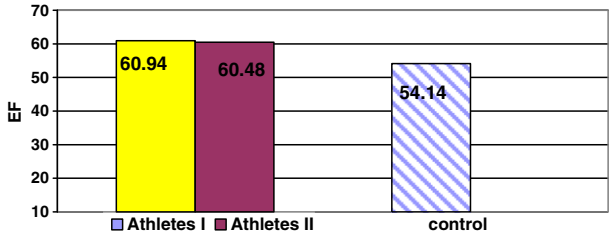

Figure 8 Mean ejection fraction in study versus control groups.

(2) Athletes II vs control : LVEDd showed no significant statistical difference (4.79 ± 1 vs 4.69 ± 0.635, *P*: 0.638) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: LVEDd showed no significant statistical difference (4.79 ± 0.507 vs 4.79 ± 1, *P*: 0.987) in athletes I vs athletes II, respectively (Fig. 9, Table 2).

The left ventricular end systole (LVESd):

(1) Athletes I vs control: The left ventricular end systole LVESd showed no significant statistical difference (3.48 ± 0.19 vs 3.48 ± 0.19, $P = 0.483$) in athletes I vs control, respectively (Table 3).
(2) Athletes II vs control: LVESd showed no significant statistical difference (3.19 ± 0.679 vs 3.48 ± 0.19, *P*: 0.142) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: LVESd show significant decrease (3.48 ± 0.19 vs 3.19 ± 0.679, $P = 0.016$) in athletes I vs athletes II, respectively (Fig. 10, Table 2).

The left atrium LA:

(1) Athletes I vs control: LA show a significant increase (3.28 ± 0.392 vs 2.58 ± 1.321, $P = 0.005$) in athletes I vs control, respectively (Table 3).
(2) Athletes II vs control: LA show a significant increase (3.62 ± 0.423 vs 2.58 ± 1.321, $P = 0.001$) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: LA showed a significant increase (3.28 ± 0.392 vs, 3.62 ± 0.423 *P*: 0.001) in athletes I vs athletes II, respectively (Fig. 11, Table 2).

The aortic root AO:

(1) Athletes I vs control: The aortic root AO showed no significant statistical difference (2.79 ± 0.329 vs 2.63 ± 0.414, $P = 0.108$) in athletes I vs control, respectively (Table 3).

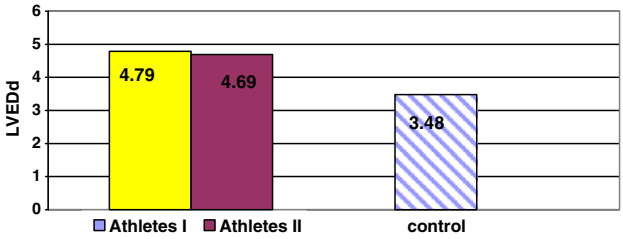

Figure 9 Mean LVEDd in study versus control groups.

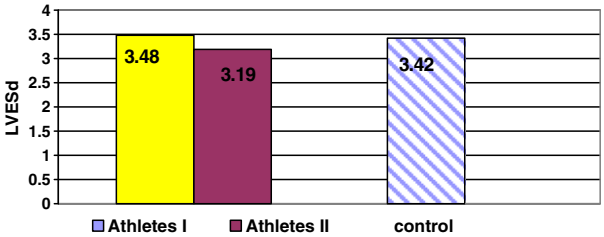

Figure 10 Mean LVESd in study versus control groups.

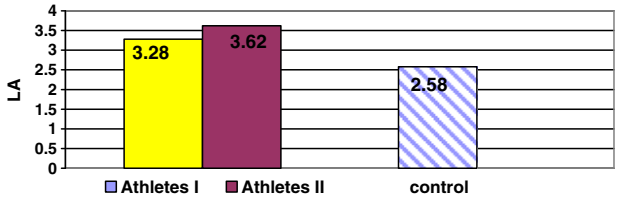

Figure 11 Mean LA in study versus control groups.

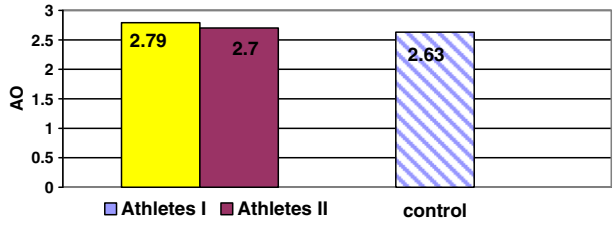

Figure 12 Mean AO in study versus control groups.

(2) Athletes II vs control: AO showed no significant statistical difference (2.7 ± 0.56 vs 2.63 ± 0.414, $P = 0.61$) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: AO showed no significant statistical difference (2.79 ± 0.329 vs 2.7 ± 0.56, P: 0.228) in athletes I vs athletes II, respectively (Fig. 12, Table 2).

The right ventricle diameter RV:

(1) Athletes I vs control: The right ventricle RV showed no significant statistical difference (2.09 ± 0.237 vs 2.27 ± 0.492, $P = 0.073$) in athletes I vs control, respectively (Table 3).
(2) Athletes II vs control: RV showed no significant statistical difference (2.37 ± 0.565 vs 2.27 ± 0.492, $P = 0.431$) in athletes II vs control, respectively (Table 4).
(3) Athletes I vs athletes II: RV was significantly increased (2.09 ± 0.237 vs 2.37 ± 0.565, $P = 0.01$) in athletes I vs athletes II, respectively (Fig. 13, Table 2).

4. Discussion

Although regular physical activity has several beneficial effects on health and is linked with reduced cardiovascular and all-cause mortality, in certain circumstances, an acute bout of exertion may cause dangerous health effects and, in extreme cases, sudden death.

We aimed primarily to compare echocardiographic and electrocardiographic changes in young proffessional athletes versus a control group of sedentary lifestyled nonathletic individuals of the same age group. Secondly, we aimed to follow up echocardiographic and electrocardiographic changes in young proffessional athletes after one year, and finally to recognize silent cardiovascular abnormalities to provide limitation criteria for eligibility of competition for athletes at risk of sudden cardiac death (SCD).

This study was conducted in Critical Care Medicine Department Cairo University in the period from may 2008 to may 2009. Our study group was picked up from the national football team candidates for the youth world cup occurring in Cairo 2009.Our control group was randomly picked nonathletic third year medical students.

4.1. Regarding clinical parameters

In our athletes, systolic blood pressure (SBP) was 117.79 ± 6.536 mmHg while in control group, it was 126.43 ± 17.043 mmHg. SBP was significantly lower in athletes I vs control ($P = 0.008$).

While diastolic blood pressure (DBP) showed no significant statistical difference in athletes I vs control, respectively (74.41 vs 76.79, $P > 0.05$).

This comes in agreement with Eric Abergel et al.[4] who showed in a study done on 286 cyclists and 52 matched sedentary volunteers that Systolic blood pressure (120 ± 9 mm Hg vs. 126 ± 13 mmHg, $P < 0.05$) and diastolic blood pressure (68 ± 9 mmHg vs. 77 ± 10 mm Hg, $P < 0.05$) were lower in athletes than in controls, as was heart rate.

In a study done by Hernelahti et al.[5] on 264 male orienteering runners among men aged 35–59 years concluded that long-term vigorous endurance training is associated with a low prevalence of hypertension. Some of the effect can be explained by a lower body mass, but exercise seems to induce a lower rate of hypertension by other mechanisms than by decreasing body weight.

4.2. Regarding ECG parameters

Compared to controls, in our study the heart rate in athletes was lower with RR interval longer significantly than in control (HR 68.88 ± 5.044 with RR 0.88 ± 0.065 vs HR 77.43 ± 6.033 with RR 0.76 ± 0.078, P 0.001) in athletes I vs control, respectively. HR and RR interval was not significantly chan-

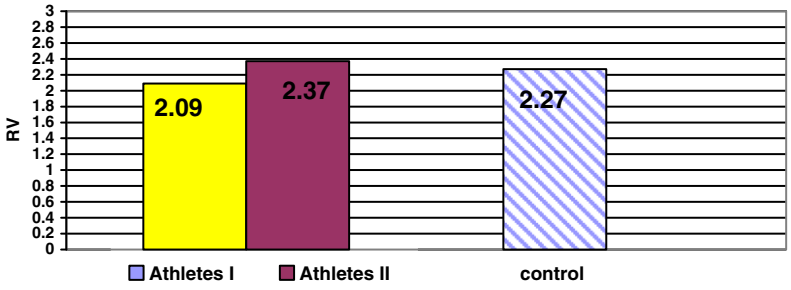

Figure 13 Shows mean RV in study versus control groups.

ged (HR 68.2 with RR 0.81 ± 0.167 vs HR 78.9 with RR 0.76 ± 0.078, P 0.153) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, HR was lower with RR longer significantly (HR 68.2 with RR 0.88 ± 0.065 vs HR 74.1 with RR 0.81 ± 0.167, $P = 0.046$) in athletes I vs athletes II, respectively.

This comes in agreement with Martinelli[6] who found that athletes at rest had lower heart rates in a study done on two groups 10 cyclists and 11 sedentary group showed (HR 65.6 ± 10.49 with RR 0.91 vs HR 70.9 ± 10.75 with RR 0.85, $P < 0.05$) in athletes vs control, respectively. Similarly, in a study done by Piraye Kervancioglu and Savas Hatipoglu[7] on 40 male football players and 25 sedentary males showed (HR 60.48 ± 6.71 with RR 1.00 vs HR 73.20 ± 3.44 with RR0.82, $P < 0.05$) in athletes vs control, respectively. Similarly, this comes in agreement with the study done by Langdeau et al.[8] who made the study on 100 athletes and 50 control and found that athletes had significantly longer RR intervals (HR 54.5 with RR1.168.5 ± 21.6 vs HR 63.8 with RR 0.943.6 ± 21.8, $P < 0.05$) in athletes vs control, respectively.

The cause of bradycardia in athletic subjects is uncertain. Most evidence suggests that the sinoatrial node and atrioventricular nodes are suppressed by an increase in vagal tone, which can be abolished by atropine or stopping training. In athletes the resting bradycardia seems to be much more related to changes in intrinsic mechanisms than to modifications in autonomic control.[9]

Compared to controls, in our study QTc interval was not significantly different (0.41 ± 0.029 vs 0.42 ± 0.022, $P > 0.05$) in athletes I vs control, respectively. QTc became significantly longer (0.43 ± 0.028 vs 0.42 ± 0.022, $P = 0.05$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, QTc showed a significant increase (0.41 ± 0.029 vs 0.43 ± 0.028, $P = 0.005$) in athletes I vs athletes II, respectively.

This comes in agreement with Sharma et al.[10] who showed that athletes have a significantly longer QTc than nonathletes ($P < 0.05$) in a study done to more than 1000 athletes (391 ± 27 vs 379 ± 29, $P < 0.05$) in athletes vs control, respectively, but in disagreement with J-B Langdeau et al.[7] who did his study on 100 athletes vs 50 control (415.8 ± 9.4 vs 406.4 ± 2.1, $P > 0.05$) in athletes vs control, respectively.

Compared to controls, in our study PR interval showed no significant statistical difference (149.03 ± 11.821 vs 139.57 ± 27.67, $P = 0.076$) in athletes I vs control, respectively PR interval showed no significant statistical difference (143.59 ± 24.87 vs 139.57 ± 27.67, $P = 0.55$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, PR interval showed a non-significant increase (149.03 ± 11.821 vs 143.95 ± 24.844 ms ($P = 0.237$) in athletes I vs athletes II, respectively.

This comes in disagreement with S Sharma, 2002 who showed in a study done on 1000 junior athletes in the same age and sex of our study group that PR interval is more prolonged in athletes than nonathletes (153 ± 20 vs 140 ± 18, $P < 0.05$) in athletes vs control, respectively.[10]

Compared to controls, in our study the QRS showed no significant statistical difference (101.15 ± 3.661 vs 100.52 ± 5.867, $P = 0.465$) in athletes I vs control, respectively. QRS showed no significant statistical difference (102.74 ± 6.561 vs 100.25 ± 5.867, $P = 0.125$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, QRS showed no significant statistical difference (101.15 ± 3.66 vs 102.74 ± 6.561, $P = 0.2771$) in athletes I vs athletes II, respectively.

This comes in disagreement with S Sharma, 2002 who showed in a study done on 1000 junior athletes in the same age and sex of our study group that the QRS duration was more prolonged in athletes than nonathletes (92 vs 89 ms, $P < 0.0001$), respectively.[10]

This difference between our study and that of S Sharma may be due to the smaller number of our study group (50 patients) in comparison to this study that comprised 1000 junior athletes.[10]

Tanji[11] said that in young endurance athletes, the electrocardiogram may show several abnormalities such as an increase in QRS voltage, that can make it difficult to convince oneself that there is no hypertensive cardiac involvement. In some cases, an echocardiogram with a Doppler evaluation of diastolic function can help in evaluating young athletes with abnormal electrocardiograms for structural heart disease.

4.3. Regarding Echo parameters

Compared to controls, in our study the septal wall in diastole (SWd) was not significantly different (0.93 ± 0.124 vs 1.07 ± 0.179, $P > 0.05$) in athletes I vs control, respectively. SWd was significantly thicker (1.21 ± 0.232 vs 1.07 ± 0.179, $P = 0.012$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training, SWd Showed a significant increase (0.93 ± 0.124 vs 1.21 ± 0.232, $P < 0.001$)* in athletes I vs athletes II, respectively.

This comes in agreement with Piraye Kervancioglu who showed that football players were shown to have significantly higher values than the control group for SWd (0.98 ± 0.13 vs 0.85 ± 0.09, $P < 0.05$) in a study on a total of 40 male football players.[7]

Compared to Controls, in our study the posterior wall in diastole (PWd) was not significantly different (0.90 ± 0.106 vs 1.07 + 0.225, $P > 0.05$) in athletes I vs control, respectively. PWd showed no significant statistical difference (1 ± 0.20 vs 1.07 ± 0.225, $P = 0.202$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, PWd showed significant increase (0.90 ± 0.106 vs 1 ± 0.20, p0.008) in athletes I vs athletes II, respectively.

This came in disagreement with Piraye Kervancioglu who showed that there was no significant difference between the PWd of the football players and control group (0.85 ± 0.09 vs 0.81 ± 0.08, $P = 0.91$).[7]

Compared to controls, in our study Ejection fraction EF showed a significant increase (60.94 ± 3.084 vs 54.14 ± 13.063, P 0.005) in athletes I vs control, respectively. EF remained significantly greater (60.48 ± 10.53 vs 54.14 ± 13.06, $P = 0.04$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, EF was not significantly different (60.94 ± 3.08 vs 60.48 ± 10.53, $P = 0.85$) in athletes I vs athletes II, respectively.

This comes in agreement with Piraye Kervancioglu who showed that football players were shown to have significantly higher values than the control group for EF (69.42 ± 4.50 vs 66.28 ± 4.54, $P < 0.05$).[7]

Compared to Controls, in our study LVEDd showed no significant statistical difference (4.79 ± 0.507 vs 4.69 ± 0.635,

$P = 0.461$) in athletes I vs control, respectively. LVEDd showed no significant statistical difference (4.79 ± 1 vs 4.69 ± 0.635, $P = 0.638$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, LVEDd showed no significant statistical difference (4.79 ± 0.507 vs 4.79 ± 1, $P = 0.987$) in athletes I vs athletes II, respectively.

This came in disagreement with Piraye Kervancioglu who showed that there was a significantly higher values in LVEDd in football players than control (5.37 ± 0.38 vs 4.63 ± 0.32, $P > 0.05$) which accounts in a large part for the elevated left ventricular mass LVM in athletes.[7]

Compared to controls, in our study the left ventricular end systole diameter LVESd showed no significant statistical difference (3.48 ± 0.19 vs 3.48 ± 0.19, $P = 0.483$) in athletes I vs control, respectively. LVESd showed no significant statistical difference (3.19 ± 0.679 vs 3.48 ± 0.19, $P = 0.142$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, LVESd showed a significant decrease (3.48 ± 0.19 vs 3.19 ± 0.679, $P = 0.016$) in athletes I vs athletes II, respectively.

This comes in disagreement with Eric Abergel who showed in a study done on 286 cyclists and 52 matched sedentary volunteers (39.7 ± 4.1 vs 31.4 ± 4.1 $P < 0.05$) in athletes vs control, respectively.[4]

Compared to controls, in our study the LA show a significant increase (3.28 ± 0.392 vs 2.58 ± 1.321, $P = 0.005$) in athletes I vs control, respectively. LA showed a significant increase (3.62 ± 0.423 vs 2.58 ± 1.321, $P = 0.001$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, LA showed a significant increase (3.28 ± 0.392 vs 3.62 ± 0.423 $P = 0.001$) in athletes I vs athletes II, respectively.

This comes in agreement with S Sharma 2002 who showed a significant LA enlargement in athletes vs control in a study on 1000 athletes vs 300 control where 14% of athletes had LA enlargement vs 1.2% in control $P < 0.05$.[10]

Compared to controls, in our study the right ventricle RV showed no significant statistical difference (2.09 ± 0.237 vs 2.27 ± 0.492, $P = 0.073$) in athletes I vs control, respectively. RV showed no significant statistical difference (2.37 ± 0.565 vs 2.27 ± 0.492, $P = 0.431$) in athletes II vs control, respectively. *Compared to themselves after one year of professional training*, RV was significantly increased (2.09 ± 0.237 vs 2.37 ± 0.565, $P = 0.01$) in athletes I vs athletes II, respectively.

This comes in agreement with Günther Schneider who showed that endurance athletes have increased RV dimensions in a study done on 21 male endurance athletes and 21 pair-matched untrained control subjects ($P < 0.05$).[12]

On the other hand, this comes in disagreement with a study done by M.K. Erol and Sule Karakelleoglu,[13] the study population consisted of 36 elite highly-trained male athletes and 16 age-matched healthy sedentary controls. All right ventricular systolic and diastolic echocardiographic parameters were similar in athletes with and without left ventricular hypertrophy ($P > 0.05$). The results of this study indicate that right ventricular systolic and diastolic functions do not deteriorate in the athlete's heart despite significant chamber dilatation. They suggest that these changes are a normal physiologic adaptation to prolonged training.

Turpeinen[14] also showed that exercise training causes a number of well known physiological changes in the heart: an increase in LVEDD and left ventricular wall thicknesses (IVS and LVPW), stroke volume is increased and heart rate is decreased during resting conditions.

5. Conclusions

Professional football playing in young males results in significant cardiovascular changes compared to their control of sedentary nonathletic medical students of similar age.

Compared to controls, clinical parameters in athletes showed a significant decrease in systolic blood pressure SBP and heart rate HR, ECG parameters showed significant prolongation in RR interval and QTc interval, and Echocardiographic parameters showed a significant increase in Left atrium diameter LA, Septal wall in diastole SWD, and ejection fraction EF.

Compared to themselves after one year of professional football playing by young males, ECG parameters showed a continuing significant prolongation in QTc, RR interval, and Echocardiographic parameters Septal wall in diastole SWD, Left atrium diameter LA, Right ventricle dimension RV, Posterior wall in diastole PWD and decrease in Left ventricular end systolic diameter LVESD compared to themselves one year earlier.

The international concern of Sudden cardiac death among professional young athletes may be attributed to Structural heart changes and ECG abnormalities acquired with professional training.

References

1. Maron BJ, Mitten MJ, Green Burnett C. Criminal consequences of commotio cordis. *Am J Cardiol* 2002;**89**:210–3.
2. Maron BJ, McKenna WJ, Danielson GK, et al. American College of Cardiology/European Society of Cardiology clinical expert consensus document on hypertrophic cardiomyopathy: a report of the American College of Cardiology Foundation Task Force on Clinical Expert Consensus Documents and the European Society of Cardiology Committee for Practice Guidelines. *J Am Coll Cardiol* 2003;**42**:1687–713.
3. Massimiliano Bianco, et al. 836 Journal of Cardiovascular Medicine 2008;9(8). Screening for coronary artery anomalies in 1360 elite competitive athletes.
4. Abergel Eric, Chatellier Gilles, Hagege Albert A. Clinical research: exercise, diet, and the heart serial left ventricular adaptations in world-class professional cyclists Implications for disease screening and follow-up. *J Am Colloid Cardiol* 2004;**44**:144–9, 10.1016/j.jacc.2004.02.057.
5. Hernelahti, Miika; Kujala, Urho M.; Kaprio, Jaakko; Karjalainen, Jouko; Sarna, Seppo. Hypertension in master endurance athletes. J Hypertension 1998;16(11):1573–77.
6. Martinelli1 FS, Chacon-Mikahil MPT, Martins LEB, et al. Heart rate variability in athletes and nonathletes at rest and during head-up tilt. Braz J Med Biol Res, 2005.
7. Piraye Kervancioglu, Savas Hatipoglu E. Cardiol J 2007;14(1):37–43.
8. Langdeau J-B, Blier L, Turcotte H, et al. Boulet. Electrocardiographic findings in athletes. The prevalence of LVH and conduction defects. *Can J Cardiol* 2001;**17**(6):655–9.
9. Hedelin R, Bjerle P, Henriksson-Larsen K. Heart rate variability in athletes: relationship with central and peripheral performance. *Med Sci Sports Exer* 2001;**33**:1394–8.
10. Sharma S, Maron BJ, Whyte G, et al. Physiologic limits of left ventricular hypertrophy in elite junior athletes. Relevance to

differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2002;**40**:1431–6.
11. Tanji JL. Exercise and the hypertensive athlete. *Clin Sports Med* 1992;**11**(2):291–302.
12. Günther Schneider et al.. Athlete's heart: Right and left ventricular mass and function in male endurance athletes and untrained individuals determined by magnetic resonance imaging. *J Am College Cardiol* 2002;**40**(10):1856–63.
13. Erol MK, Sule Karakelleoglu. Department of Cardiology, Medical School Hospital, Ataturk University, 25050 Erzurum. Assessment of right heart function in the athlete's heart. Heart Vessels 16(5):175-180.
14. Turpeinen AK, Kuikka JT, Vanninen E, et al. Athletic heart: a metabolic, anatomical, and functional study. *Med Sci Sports Exer* 1996;**28**:33–40.