

**Research Article**

## **CLINICAL RESEARCH ON CARDIOVASCULAR ALTERATION ON 86'S PROFESSIONAL ATHLETES IN IRAN**

**Lotfali Pourkazemi<sup>1\*</sup> and Roghieh Razeghi Jadid<sup>2</sup>**

<sup>1</sup>*Sports Medicine Federation of Iran*

<sup>2</sup>*Department of Herbal Sciences, Tonekabon Branch, Islamic Azad University*

*\*Author for Correspondence*

### **ABSTRACT**

Over the past 4 decades, numerous scientific reports have examined the relationships between physical activity, physical fitness, and cardiovascular health. The present study aimed to research on cardiovascular alterations on 86's professional athletes in Iran. 86 elite athletes with more than 5 years background of professional sport and medal winner in international, Asian and Olympic Games were entered the study. Sampling model was cluster form from marathon runners, long-distance runners and also professional climbers. Clinical visit: Bradycardia (94%), systolic murmur (55%), S3 and S4 sounds (27%), increasing of pulse amplitude (90%). Furthermore, there was a significant correlation between cardiac dilation and wall thickening and stroke volume, which can affect strongly on athletes records and also his sport living time. Accordingly, Regular bases of Exercise can make Athletes heart strong and safe. Additionally, severe training for getting just a medal during less than 3 years can damage heart function and can make more hurt for heart, even sudden death.

**Keywords:** *Cardiovascular Alteration, Professional Athletes, Runners, Climbers*

### **INTRODUCTION**

Over the past 4 decades, numerous scientific reports have examined the relationships between physical activity, physical fitness, and cardiovascular health. All types of human movement, no matter what the mode, duration, intensity, or pattern, require an expenditure of energy above resting values. Much of this energy will be provided through the use of oxygen. In order to supply the working muscles with the needed oxygen, the cardiovascular and respiratory systems must work together. Different types of exercise impose various loads on the cardiovascular system. Isotonic (dynamic) exercise is defined as muscle contraction of large muscle groups resulting in movement. Isometric (static) exercise is defined as a constant muscle contraction of smaller muscle groups without movement. A third type of exercise is resistance exercise. This is a combination of isometric and isotonic exercise by use of muscle contraction with movement. Most activities usually combine all types of exercise.

During acute isotonic exercise, the total peripheral vascular resistance falls as a result of the marked vasodilatation of the vessels in exercising muscles, which overcomes the vasoconstriction of the splanchnic and renal vessels. As a result, after load decreases and the increased cardiac output is redistributed, mainly to the active muscles.

Regular participation in intensive physical exercise is associated with central and peripheral cardiovascular adaptations that facilitate the generation of a large and sustained cardiac output and enhance the extraction of oxygen from exercising muscle for aerobic glycolysis, respectively. An increase in cardiac size is fundamental to the ability to generate a large stroke volume. Over the past three decades, the athlete's heart has been the subject of several echocardiographic studies involving many thousands of athletes (Pluim *et al.*, 2000; Pelliccia *et al.*, 1991; Sharma *et al.*, 2002; Spirito *et al.*, 1994; Morganroth *et al.*, 1975).

Most studies have been cross sectional in design and focused on Caucasian athletes aged 18–35 years. These studies provide insight into the magnitude and determinants of cardiac size in athletes and are invaluable in aiding the differentiation of physiological left ventricular hypertrophy (LVH) (athlete's heart) from hypertrophic cardiomyopathy (HCM), the leading cause of exercise related sudden cardiac death in young athletes (Maron, 2003).

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Athletic training is associated with statistically significant increases in cardiac dimensions compared with sedentary individuals. A meta-analysis of almost 1000 M-mode echocardiographic studies in highly trained male athletes showed that athletes exhibited a 15–20% increase in septal and left ventricular posterior wall thickness, respectively (Pluim *et al.*, 2000). In terms of absolute values, however, the mean LVWT in athletes was between 10 and 11 mm and fell within the normally accepted range for sedentary individuals.

Subsequent two-dimensional echocardiographic studies in large cohorts of highly trained athletes have shown that the vast majority has an LVWT  $\leq 12$  mm and would not normally be considered to have LVH. However, a small minority of athletes exhibit substantial increases in the magnitude of LVWT measurements that overlap with those observed in patients with morphologically mild HCM. In an Italian study of 947 Italian Olympian athletes, 1.7% had an LVWT exceeding 12 mm (Spirito *et al.*, 1994). A more recent study of 3000 highly trained British athletes revealed that 1.5% of athletes exhibited an LVWT  $>12$  mm (Basavarajaiah, 2008) (Figure 1) The maximal value for LVWT in both studies was 16 mm suggesting that an athlete with a maximal LVWT  $>16$  mm may be considered to have pathological LVH, although there have been isolated reports of LVH of up to 19 mm (Nagashima *et al.*, 2003; Rodriguez *et al.*, 1995) in some ultra-endurance athletes.

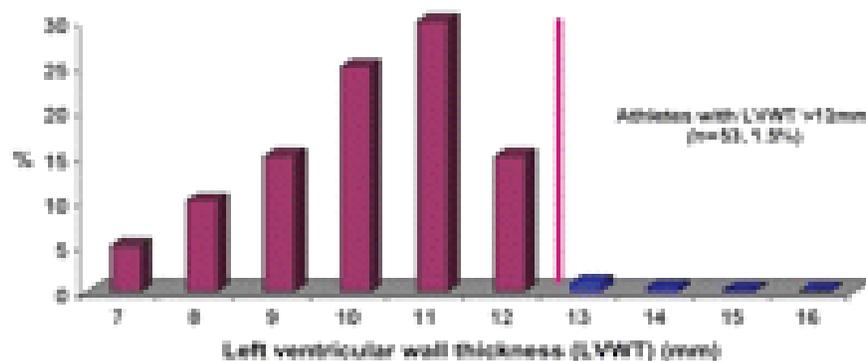


Figure 1

Distribution of left ventricular wall thickness in 3500 highly trained athletes demonstrating that  $\sim 2\%$  athletes exhibit a left ventricular wall thickness  $>12$  mm. Reproduced from Basavarajaiah *et al.*, (2008) with permission from the American College of Physicians.

Morgenroth and colleagues utilized echocardiography to assess left ventricular end-diastolic volume and mass increased in isotonic athletes (athletes performing work with chronic volume demands) as compared to controls, while isometric athletes (athletes performing work that required chronic pressure demand) had increased left ventricular mass but normal end-diastolic volume.

An average wall thickness was greater in isometric athletes (Morganroth, 1975).

The work of Sugishita and colleagues should be reviewed to compare and contrast the normal changes that occur in the athlete's heart in response to training from the abnormal changes that occur in response to pathological conditions in the heart.

They assessed cardiac morphology echocardiographically in 31 runners and 17 judo athletes and compared this data with that obtained from 15 patients with aortic regurgitation, 13 patients with hypertension, 14 patients with dilated cardiomyopathy, 11 patients with hypertrophic cardiomyopathy, and 25 controls. They found that the ratio of left ventricular radius to wall thickness was normal in runners but increased in patients with aortic regurgitation and dilated cardiomyopathy. Therefore, the changes that occurred in response to the chronic volume demands placed upon the long-distance runners' hearts could clearly be distinguished from the abnormal changes in those patients with pathological volume overload states (Sugishita, 1983).

Common EKG changes in athletes are following items:

- Sinus bradycardia

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- Sinus arrhythmia
- First degree AV-block
- Second degree Wenckebach AV-block
- incomplete RBBB (QRS duration <0.12 s)
- early repolarization, and isolated QRS voltage criteria for LVH

Less common EKG changes in athletes are following items:

- wpw syndrome
- Atrial Fibrillation
- Atrial Flutter
- Junctional rhythm
- Wandering atrial pacemaker
- J point elevation
- Sinus pusses
- Incomplete RBBB (Right bundle branch block pattern)
- Prominent U waves
- Biphasic T waves
- ST depression
- Isolated T-wave inversion
- Ventricular and atrial ectopi
- T-wave isoelectric

**Objectives**

The athlete may be subject to diagnostic evaluations including a stress test, ambulatory monitoring, or invasive tests such as electrophysiological evaluation or cardiac catheterization based on such ECG abnormalities as ventricular hypertrophy, repolarization abnormalities, or bradycardias, which are physiological rather than pathologic. Also, the predominant adaptations include increased left ventricular end-diastolic cavity dimension, increased left ventricular wall thickness, improved diastolic filling, and decreased heart rate. Thus the present study aimed to research on cardiovascular alterations on 86’s professional athletes in Iran.

**MATERIALS AND METHODS**

86 elite athletes with more than 5 years background of professional sport and medal winner in international, Asian and Olympic Games were entered the study. Sampling model was cluster form from marathon runners, long-distance runners and also professional climbers.

The data were gathered two times during two years through following three ways:

- 1) Clinical visits
- 2) Echocardiography
- 3) Electrocardiographs (EKG)

**RESULTS AND DISCUSSION**

**Results**

**Table 1: Clinical Visit in Athletes**

<b>Bradycardy</b>	<b>Long distance runners (23 to 50 beats/minute) 83%</b>	<b>Other athletes (50 to 60 beats/minute) 96%</b>
<b>Systolic murmur</b>	dynamic athletes 70 to 80%	static athletes 30 to 40%
<b>Heart sounds</b>	S <sub>3</sub> sound 40 to 60%	S <sub>4</sub> sound 5 to 10%
<b>Pulse amplitude</b>	Increased 80 to 100%	

1: Bradycardy = Heart rate less than 60 beats per minute  
 2: Ejection systolic Murmur Grade 1-2 in the left sternal border

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Clinical visit: Bradycardia (94%), systolic murmur (55%), S3 and S4 sounds (27%), increasing of pulse amplitude (90%).

Echocardiographs: LVH (72%), LVID (58%), RVH (50%), and cardiac Walls changes (97%).

Electrocardiographs: ST changes (66%), T changes (83%), QRS changes (70%), P changes (59%).

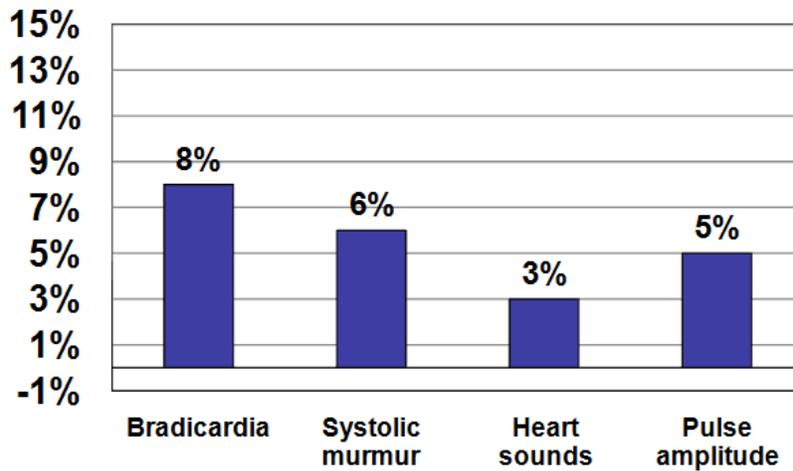
**Table 2: Echocardiography in Athletes**

<sup>1</sup> LVH	Dynamic athletes 80 to 90%	Static athletes 50 to 60%
<sup>2</sup> LVID	men 45 mm to 72 mm	Women 37 mm to 67 mm
<sup>3</sup> RVH	Dynamic athletes 55 to 75%	Static athletes 35 to 47%
Cardiac wall changes	Increased interventricular septum thickness with average 24% greater than normal in 95% to 100%	Increased posterior free wall thickness 95 to 100%

1: LVH= left ventricular Hypertrophy

2: LVID= left ventricular internal Dimension

3: RVH= Right ventricular Hypertrophy



**Figure 2: Percentage of higher clinical visit parameters changes in Long-distance athletes and climbers in comparison with other athletes**

**Table 3: Electrocardiographs (EKG)**

<sup>1</sup> ST- changes	ST- segment elevation with high frequency of J-point elevation 66%	St segment depression with J-point 5%
<sup>2</sup> T- changes	T-Tall and peaked 83%	T-inverted 35%
<sup>3</sup> QRS- changes	With right axis deviation (>45°) 70%	With left axis deviation(<0°) 6%
<sup>4</sup> P-changes	Increased P-wave amplitude 59%	Notched P-wave 21%

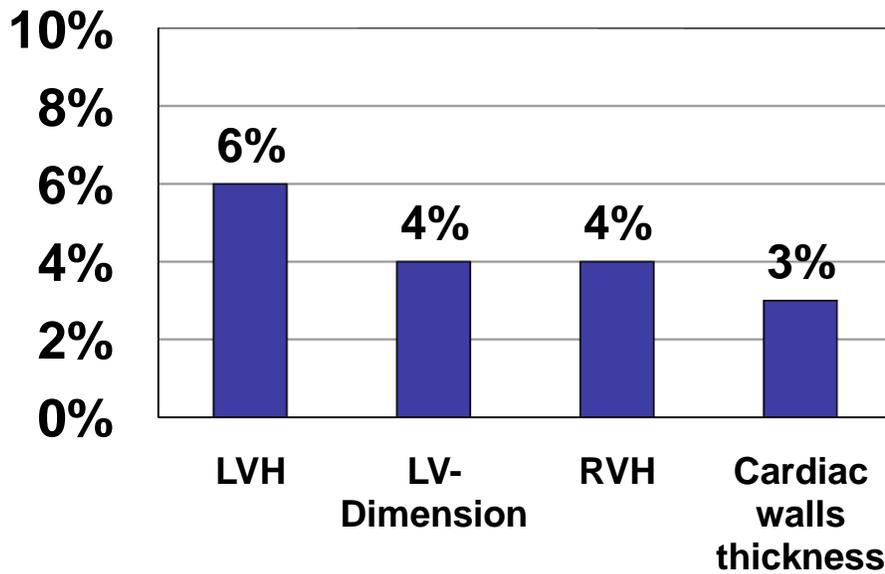
1: ST-segment elevation due to early repolarization in inferior and precordial leads.

2: T-Tall and peaked due to early repolarization

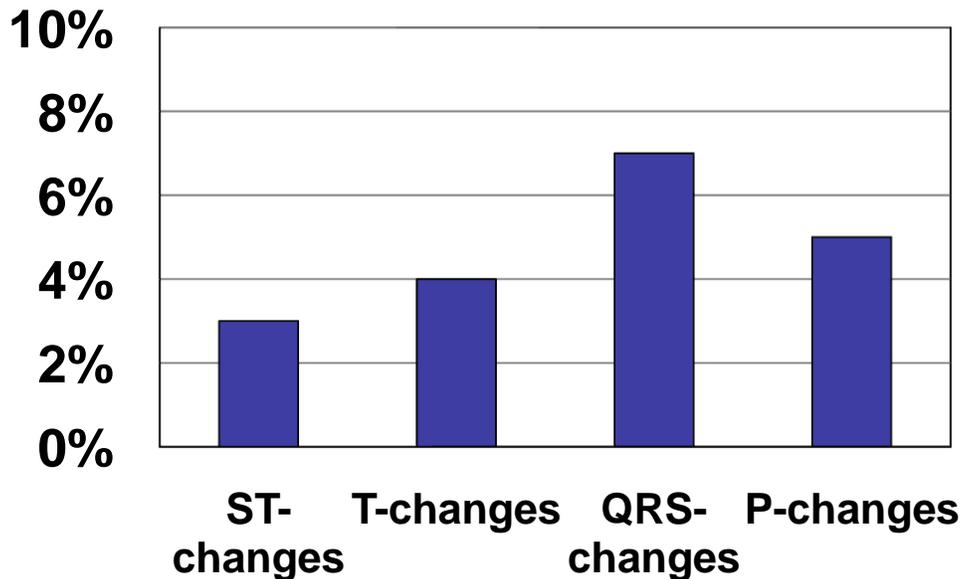
3: normal QRS frontal axis is between 0o and 90o

4: P-wave changes due to atrial hypertrophy

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**Figure 3: Percentage of higher echocardiography parameters changes in Long-distance athletes and climbers in comparison with other athletes**



**Figure 4: Percentage of higher EKG parameters changes in Long-distance athletes and climbers in comparison with other athletes**

**Discussion**

The results in this research shows that if the duration of exercise in years be longer and in a regular bases, there are a significant correlation was found between cardiac dilation and wall thickening and stroke volume, that can affect strongly on athletes records and also his sport living time.

The comparison of this search with previous studies shows that:

In professional athletes specially in the long-distance runners and climbers there are in average 3 to %10 more changes in Echo, EKG and Clinical Visit findings, in compare with other athletes. It seems that the

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time of training & type of sport are two most significant causes for these changes. Dynamic athletes have more changes than others. A significant correlation was found between types of sport, years of training, age, gender, body surface area and gene with heart functional and anatomical changes, which can be strongly effective on athletes' records and his sport living time.

### Conclusion

- 1) Regular bases of Exercise can make Athletes heart strong and safe.
- 2) Severe training for getting just a medal during less than 3 years can damage heart function and can make more hurt for heart, even sudden death.
- 3) Based on these result we recommend athletes to pay attention to the following advises:
  - Step by step training from younger age is the best way for athletes to make better heart.
  - Long term training can make better adaptation for heart walls and cavity
  - Stopping of exercise for more than 3 months can affect heart muscle to lose its benefits for sport survival.
  - Unusual training and without sport Medicine educations can result sudden death in athletes heart or can make new heart abnormalities.

### REFERENCES

- Basavarajaiah S, Wilson M, Whyte G, Shah A, McKenna W and Sharma S (2008).** Prevalence of hypertrophic cardiomyopathy in highly trained athletes: relevance to pre-participation screening. *Journal of the American College of Cardiology* **51** 1033-9.
- Maron BJ (2003).** Sudden death in young athletes. *New England Journal of Medicine* **349** 1064-75.
- Morganroth J, Maron BJ, Henry WL and Epstein SE (1975).** *Annals of Internal Medicine* **82** 521–524.
- Morganroth J, Maron BJ, Henry WL and Epstein SE (1975).** Comparative left ventricular dimensions in trained athletes. *Annals of Internal Medicine* **82** 521-4.
- Nagashima J, Musha H, Takada H and Murayama M (2003).** New upper limit of physiologic cardiac hypertrophy in Japanese participants in the 100-km ultra marathon. *Journal of the American College of Cardiology* **42** 1617-23.
- Pelliccia A, Maron BJ, Spataro A, Proschan MA and Spirito P (1991).** The upper limit of physiologic cardiac hypertrophy in highly trained elite athletes. *New England Journal of Medicine* **324** 295-301.
- Pluim BM, Zwinderman AH, van der Laarse A and van der Wall EE (2000).** The athlete's heart: a meta-analysis of cardiac structure and function. *Circulation* **101** 336-44.
- Rodriguez Reguero JJICG, Lopez de la Iglesia J, Terrados N, Gonzalez V, Cortina R and Cortina A (1995).** Prevalence and upper limit of cardiac hypertrophy in professional cyclists. *European Journal of Applied Physiology and Occupational Physiology* **70** 375-8.
- Sharma S, Maron BJ, Whyte G, Firoozi S, Elliott PM and McKenna WJ (2002).** Physiologic limits of left ventricular hypertrophy in elite junior athletes: relevance to differential diagnosis of athlete's heart and hypertrophic cardiomyopathy. *Journal of the American College of Cardiology* **40** 1431-6.
- Spirito P, Pelliccia A, Proschan MA, Granata M, Spataro A and Bellone P et al., (1994).** Morphology of the 'athlete's heart' assessed by echocardiography in 947 elite athletes representing 27 sports. *American Journal of Cardiology* **74** 802-6.
- Sugishita Y, Koseki S, Matsuda M, Tamura T, Yamaguchi I and Ito I (1983).** Dissociation between regional myocardial dysfunction and ECG changes during myocardial ischemia induced by exercise in patients with angina pectoris. *American Heart Journal* **106** 1-8.