

Impact of Sport and High Intensity Training on Global and Regional Myocardial Deformation in Elite High School Athletes



Rebecca K Lindquist, Angela R Miller, Chelsea L Reece, Jennifer A Johnson, Jonathan N Johnson, Sharon L Mulvagh, Patrick W O'Leary, Frank Cetta, Benjamin W Eidem, Mayo Clinic, Rochester, MN

ABSTRACT

Background: Cardiac adaptation to high intensity athletic training is characterized by increases in left ventricular (LV) chamber dimension, wall thickness & mass. The physiologic impact of training on myocardial deformation in highly trained elite high school athletes has not been extensively reported. The purpose of this study was to assess the impact of the type of sport participation & long-term training on parameters of LV systolic & diastolic function including newer modalities of tissue Doppler and myocardial deformation in highly trained high school athletes compared to sedentary controls.

Methods: Standard two-dimensional (2D), spectral Doppler, & tissue Doppler were prospectively performed utilizing a GE Vivid 7 system to evaluate LV systolic and diastolic function in 78 elite high school athletes (45 males) compared to healthy controls. Longitudinal 2D strain was performed to evaluate 17 regional (apical, mid, and basal myocardial segments) and global longitudinal LV myocardial strain. The impact of the type of sport and training (figure skating, ice hockey, soccer) on LV systolic and diastolic function were assessed.

Results: Data are summarized in tables 1-4. Heart rate, LV dimensions, and LV mass were significantly different in athletes compared to healthy controls (p<0.001). Traditional measures of LV systolic and diastolic function as well as tissue Doppler and global longitudinal 2D strain were not different between groups. However, regional differences in strain were identified, with apical deformation increased relative to basal function in athletes compared to controls (p<0.001). Similar regional changes were seen in all athletes, the most prominent differences in skaters and hockey players.

Conclusions: While global longitudinal myocardial velocity and deformation were not significantly different in athletes compared to healthy controls, regionally increased apical deformation compared to basal function was consistently demonstrated in all athletic groups. Ongoing assessment of radial and circumferential deformation as well as LV torsion may add novel insights into whether this represents a compensatory adaptation or augmented contractile reserve with training in athletes. This information may also assist in distinguishing athletic from myopathic increases in LV mass.

METHODS

Standard two-dimensional (2D), spectral Doppler & tissue Doppler echocardiography exams were prospectively performed in 78 elite high school athletes (45 males) and compared with healthy controls. A GE Vivid 7 system was utilized to evaluate LV systolic and diastolic function.

Traditional echo parameters measured were LV mass, ejection fraction, mitral inflow and TDI. Longitudinal 2D strain was performed to evaluate 17 regional (apical, mid, and basal myocardial segments) and global longitudinal LV myocardial strain. The impact of the type of sport and training (figure skating, ice hockey, soccer) on LV systolic and diastolic function were assessed.

BACKGROUND

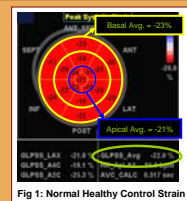
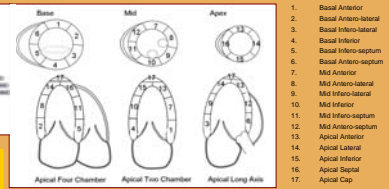
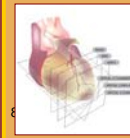


Fig 1: Normal Healthy Control Strain

RESULTS

Table 1: Demographics

	Athletes	Controls	P-value
N	78	25	-
Height (cm)	170 ± 10	165 ± 13	NS
Weight (kg)	66 ± 11	63 ± 14	NS
BSA (m ²)	1.8 ± 0.2	1.7 ± 0.2	NS
HR (BPM)	63 ± 11	71 ± 13	p<0.01
BP _{systolic} (mmHg)	124 ± 11	122 ± 11	NS
BP _{diastolic} (mmHg)	67 ± 9	71 ± 10	NS

Table 2: Traditional Echo Measurements

	Athletes	Controls	P-Value
LV SWT _{diast} (mm)	0.9 ± .01	0.8 ± 0.1	NS
LV PWT _{diast} (mm)	1.3 ± 0.2	1.1 ± 0.1	p<0.01
LV Mass (gm/m ²)	147 ± 34	131 ± 38	p<0.001
LV Mass Index	35 ± 7	31 ± 7	p<0.01
LV EDD (cm)	5.1 ± 0.4	4.8 ± 0.5	p<0.01
LV ESD (cm)	3.2 ± 0.4	3.0 ± 0.3	p<0.3
LV EF (%)	61 ± 6	60 ± 5	NS
MV E-velocity (m/sec)	1.0 ± 0.2	1.0 ± 0.2	NS
MV A-velocity (m/sec)	0.4 ± 0.1	0.4 ± 0.1	NS
MV E/A	2.5 ± 0.7	2.4 ± 0.6	NS
MV V _p	83 ± 15	86 ± 17	NS

Table 3: Myocardial Velocity & Deformation Data

	Athlete	Controls	P-Value
TDI			
Septal E (cm/s)	0.15 ± 0.02	0.14 ± 0.02	NS
Septal A	0.06 ± 0.01	0.06 ± 0.01	NS
Septal S	0.09 ± 0.01	0.09 ± 0.01	NS
Mitral E	0.19 ± 0.03	0.20 ± 0.03	NS
Mitral A	0.06 ± 0.01	0.06 ± 0.01	NS
Mitral S	0.11 ± 0.02	0.11 ± 0.03	NS
Tricuspid E	0.16 ± 0.03	0.16 ± 0.03	NS
Tricuspid A	0.08 ± 0.02	0.09 ± 0.02	NS
Tricuspid S	0.14 ± 0.02	0.14 ± 0.02	NS
Strain			
Global 2D	-20.3 ± 1.8	-20.7 ± 1.9	NS
Apical Avg	-21.9 ± 3.4	-20.6 ± 2.8	p<0.01
Mid Avg	-20.1 ± 1.7	-20.4 ± 2.0	NS
Basal Avg	-19.4 ± 1.8	-21.1 ± 2.7	p<0.001

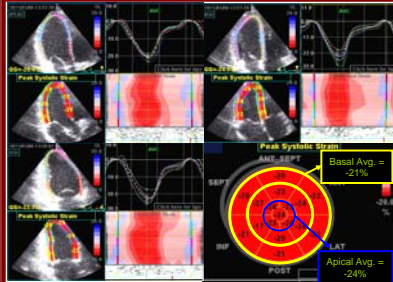


Fig 2: Figure Skater

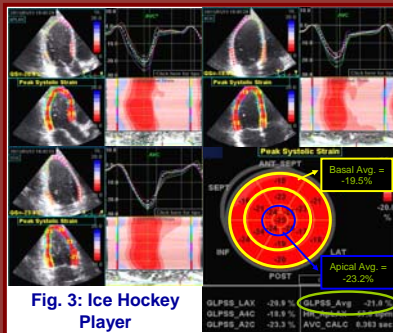


Fig 3: Ice Hockey Player

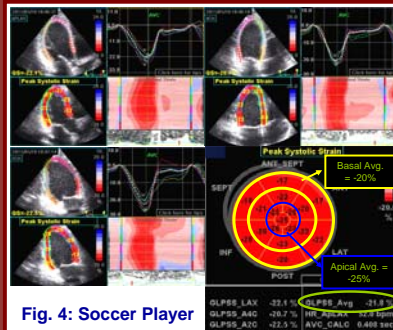


Fig 4: Soccer Player

Table 4: Regional Deformation Pattern in Athletes

	Figure Skaters	Ice Hockey	Soccer
Strain			
Global 2D	-21.5 ± 1.9	-20.4 ± 1.8	-20.0 ± 1.7
Apical	-23.2 ± 3.9	-22.5 ± 3.2	-21.1 ± 3.5
Mid	-21.3 ± 1.6	-20.1 ± 1.6	-19.9 ± 1.7
Basal	-20.5 ± 2.6	-19.1 ± 1.7	-19.7 ± 1.8

DISCUSSION

Similarities between trained athletes and healthy control subjects:

- LV systolic function
LV EF: 61 ± 6% vs. 60 ± 5%
- LV diastolic function
MV E/A: 2.5 ± 0.7 vs. 2.4 ± 0.6
- TDI (see Table 3)
- Global longitudinal 2D strain
-20.3 ± 1.8% vs. -20.7 ± 1.9%

Significant differences between trained athletes and healthy subjects:

- Heart rate (63 ± 11 bpm vs. 71 ± 13 bpm)
- LVEDD (5.1 ± 0.4 cm vs. 4.8 ± 0.5 cm)
- LV mass (147 ± 34 gm/m² vs. 131 ± 38 gm/m²)
- Apical average strain (-21.9 ± 3.4% vs. -20.6 ± 2.8%)
- Basal average strain (-19.4 ± 1.8% vs. -21.1 ± 2.7%)

Similar regional changes were seen in all athletes, the most prominent differences in figure skaters and hockey players

CONCLUSIONS

1. Global average longitudinal myocardial velocity and deformation are comparable between athletes and healthy control groups.
2. Apical level deformation was significantly increased compared to the basal level in all athletic groups.
3. Further assessment can provide insight as to whether these findings represent a compensatory adaptation or augmented contractile reserve as a result of athletic training.
4. This study may also enable differentiation between increased LV mass due to athletic training from pathologic cardiomyopathies.

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