Clinical Exercise Testing in the Athlete

- The athlete's heart
- Sudden cardiac death in athletes
- Screening athletes for cardiovascular disease

Historical Notes:

- Giovanni Lancisi (father of cardiology), 17th century, heart size has a role in ability to perform muscular work
- Sir William Osler (19th century) training is accompained by an increased capacity of the heart
- Beneke (1879) athletic activity results in growth of the left ventricle disproportionate to the diameter of the descending aorta—deleterious effect.
- Deutsch and Kauf (1927) took x-rays of hearts from thousands of athletes from 16 sports and confirmed they had larger hearts. This work dispelled the notion that vigorous athletic activity was deleterious to health

An Efficient Heart

- Large chamber size
- Slow heart rate
- Increased wall thickness

The Athlete's Heart: Structural Changes :

- Modest (10%) increase in left ventricular cavity
- Symmetrical increases in wall thickness
- 45-50% increase in left ventricular mass
  - (still within normal limits except for 9% of men and 7% of women)
- Normal indices of systolic function
- Normal or enhanced (during exercise) indices of diastolic filling

Structural Changes, Cont.

- Modest (24%) increase in right ventricular dimension
- Modest (16%) increase in atrial dimensions
- Regurgitation of at least 1 cardiac valve (91% athletes vs. 38% controls). (mitral and tricuspid valves)
  - Mechanism unknown but related to slower HR, increased SV, and more rapid filling

Coronary Arteries

- Resting cross sectional area of coronary arteries is similar between athletes and sedentary.
- Vasodilatory response to nitroglycerine was greater for runners
- Dilating capacity was positively correlated with aerobic capacity

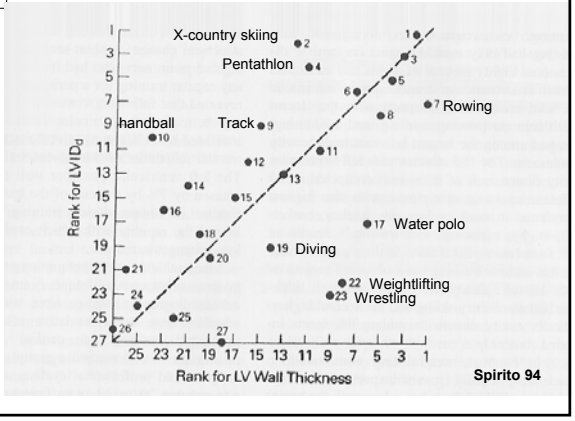
Are athletes naturally endowed with larger hearts or do their hearts change in response to exercise?

- Ehsani et. Al (1978)
  - **Competitive swimmers, 12 wk period of training and detraining.**
    - **Ventricular size increased with training**
    - **responses reversed during detraining**
  - **Must change in response to training**

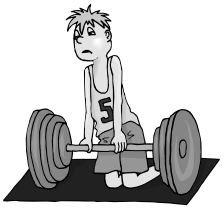
It is clear that the heart adapts to chronic overload

Impact of type of exercise

- **Morganroth et. al (1975). Cardiac changes are associated with the type of exercise training**
- **Spirito et. al (1994) studied elite athletes from 27 sports**
  - **While both isotonic and isometric athletes develop cavity dilation and increased wall thickness**
    - Ventricular dilation predominates slightly in the endurance athletes
    - Wall thickness predominates in the isometric athletes



Isotonic (endurance) sports favor Increases in Ventricular Volume



Isometric (static) sports favor Increases in Wall Thickness

Impact of intensity and duration

- **Exercise Intensity**
  - **> intensity, > effects on LV cavity and wall thickening**
  - **1 wk of vigorous swimming increased LVDV 11%**
- **Long duration training**
  - **Chronically trained professional cyclists (Nishimura et. al 1980)**
    - 20-29 and 30-30 yr olds, similar increases in LV size, wall thickness, and mass
    - 40-49 yr olds, greater wall thickness and mass
    - Many years of athletic training may have a cumulative effect

Effects of gender

- LVEDD and wall thickness 6 and 14% > in female athletes than female controls
- Female athl significantly smaller LVEDD, wall thickness, and LV mass (31%) than male athletes of similar age and body size in the same sport.
- LV hypertrophy is more common in female athletes (36-43%) than male athletes (17-22%)

Effects of age

- Children 6-14 yrs
  - no change in LVDD with training
  - May be due to less intense training or different cardiovascular effects in children
- Elderly
  - Proposal that older subjects have smaller cardiac responses to training is not supported by data

Effects of race

- Ekelund et. al (1990), black men had a higher SBP response to exercise than white men with similar resting BP.
- Will this impact the cardiac response to training?

Reversibility of Training Effects

- Ehsani et al (1978) 8%↓LVDD, 15% ↓ wall thickness, 27%↓ LVM when competitive runners cease training for 1 wk.
- Shapiro and Smith (1983) reversal of cardiac effects 6 wks after a 6 wk running program in sedentary subjects.
- Maron et al (1993) 15 to 33% changes in septal thickness in Olympic athletes in and out of season
- Hickson et al (1982) 10 wk cycling program followed by reduced training (67%) for 15 weeks. No loss of cardiac training effects

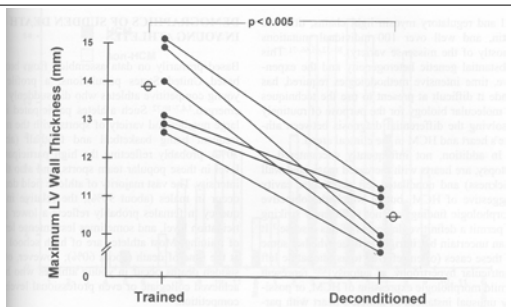


FIGURE 9.9. Changes in maximal left ventricular wall thickness associated with deconditioning in Olympic level rowers. Cardiac dimensions were obtained with two-dimensional echocardiography at peak training and subsequently after 6 to 34 weeks of deconditioning following the 1988 Olympic Games, with measurements made in a blinded fashion. Adapted with permission from Maron BJ, Pellicia A, Spartaro A, et al: Reduction in left ventricular wall thickness after deconditioning in highly trained Olympic athletes. Br Heart J 69:125–128, 1993.

Electrical Cardiac Changes in Athletes

- Alterations in rhythm
- Conduction
- Repolarization
- Precordial voltage

- Most effects are due to increased vagal tone or down-regulated sympathetic drive.

Arrhythmia	Gen pop (%)	Athletes (%)
Sinus bradycardia	23.7	50-85
Sinus arrhythmia	2.4-20	13.5-69
Wander. A pacemak	--	7.4-19
1st degree block	0.65	6-33
2nd degree block		
Mobitz I	0.003	0.125-10
Mobitz II	0.003	not reported
3rd degree block	0.0002	0.017
Junctional rhythm	0.06	0.031-7.0

Repolarization

- **ST segment elevation in precordial leads and peaked T waves**
  - Early repolarization, >10% of athletes
  - Due to ↓ resting sympathetic tone which reveals inherent asymmetry of repolarization.
- **J point depression and horizontal or up-sloping ST segment depression**
- **Peaked or Biphasic T waves in leads V1-V4**
- **T wave inversion in the lateral leads**
  - Differences in action potential duration of myocardial cells

Precordial Voltage

- **Increased voltages of QRS (8 to 76%)**
  - Left and right ventricular hypertrophy
- **Increased P wave voltage and notching**
  - Atrial hypertrophy and ↓sympathetic tone
- **QRS axis becomes more vertical and rotates toward the right**
  - Increased muscle mass in the apex of the right ventricle

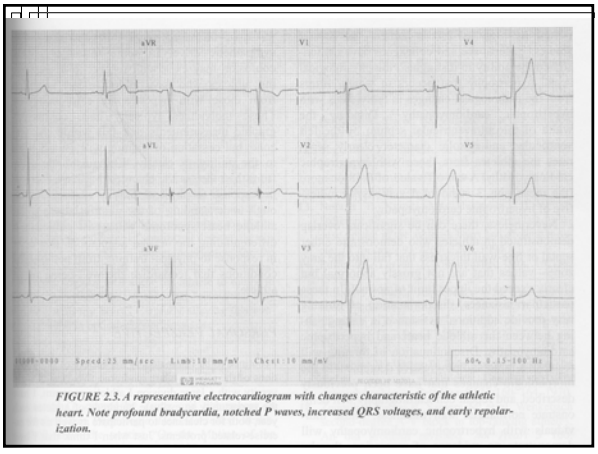


FIGURE 2.3. A representative electrocardiogram with changes characteristic of the athletic heart. Note profound bradycardia, notched P waves, increased QRS voltages, and early repolarization.

Sudden Cardiac Death in Athletes

Prevalence

- 1:200,000 per academic year for high school competitors
- 1:15,000 to 1:50,000 for healthy male athletes, joggers, and marathon racers

Causes of Sudden Cardiac Death

- Young athletes < 35 yrs = congenital cardiovascular diseases
  - Hypertrophic cardiomyopathy (36%)
- Adult and senior athletes
  - ischemic heart disease most common cause

Sudden Death due to Trauma

- Very rare: 70 cases
- Chest blows causing sudden death predominantly seen in baseball
- Velocity of the blow is not large enough to cause myocardial contusions (such as automobile accident causing MI)
- 70% commotio cordis (cardiac concussion) < 16 yrs (more pliable chest)

Causes of Commotio Cordis

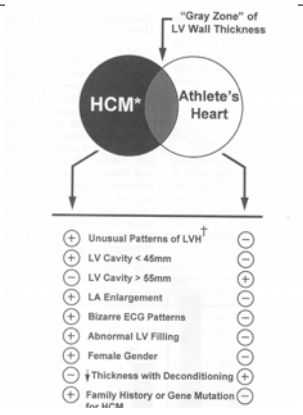
- Blow to the left chest
  - Cardiac cycle dependent (during repol)
  - Most have ventricular fibrillation
- Survival 10%, depends on timing of resuscitation (within 1 min)
- Reversal can result in complete recovery

Screening Athletes

- Morphologic changes in athletes are small compared to patients with primary myocardial disease or significant valvular disease
- Most common differential diagnosis is between athlete's heart and HCM

Echocardiographic Limits in Athletes

- Wall thickness (septal and posterior) < 1.3 cm
- LV end-diastolic cavity dimension ≤ 6.0 cm
- Left ventricular mass men ≤ 294 g
  women ≤ 198 g



Conclusions

- **Chronic exercise produces changes in the heart: morphological, electrical, functional**
- **Such changes are not dangerous, but can complicate diagnosis of cardiac disease**
- **By medical screening, it is possible to distinguish athlete's heart from cardiac disease (echo for HCM)**
- **Sudden death in athletes is rare and can be prevented by appropriate medical screening**