





**Exercise and Arrhythmia: Is there too much of a good thing?** 

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**Benefits of Exercise**

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- A physically active lifestyle leads to a 42-44% reduction in the development of CV diseases (primary prevention).
- Exercise reduces CV mortality and hospital admission in those who have CV diseases (secondary prevention).
- Exercise helps control HTN, diabetes, PAD, CAD, hyperlipidemia, etc

**Exercise is a form of Ischemic Preconditioning**

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- Ischemic preconditioning:
  - brief periods of ischemia before prolonged coronary artery occlusion reduce subsequent infarct size (as much as 75% in animals) and/or the risk of developing VT/VF
- Because of the absence of physiological adaption and improving risk factor during such a short period of time, ischemic preconditioning is thought to be caused by favorable changes in neuro-hormonal and metabolic factors.
- Early protection disappears 2-4 hours after preconditioning
- Delayed protection appears 24 hours later and lasts 2-3 days

**Exercise is a form of Ischemic Preconditioning**

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- Exercise:
  - may produce local and systemic factors (adenosine, bradykinins, etc) that are also produced during ischemic preconditioning
  - may produce mild ischemia similar to ischemic preconditioning
  - exercise-induced angina is often significantly reduced on a second exercise effort (warm-up angina)
  - exercise-induced angina also shows biphasic protection

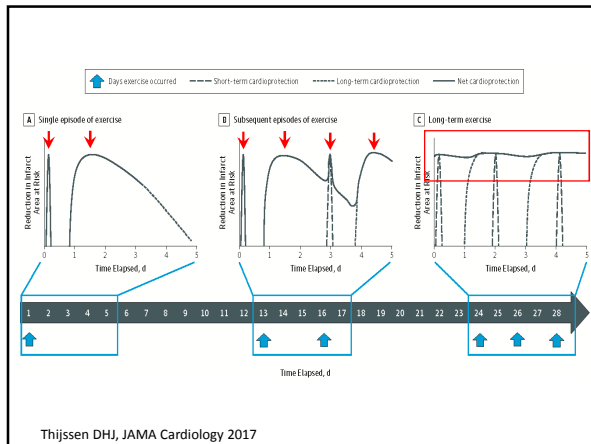
Lambiase PD, JACC 2003;41(7):1174

**Exercise is a form of Ischemic Preconditioning**

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- Greater physical activity level in the week before MI appear to be associated with lower in-hospital mortality and less fatal events.
- Greater physical activity level in the week before CABG appear to be associated with higher rates of survival
- Moderate intensity exercise (below lactate threshold) appears to be enough to release beneficial neuro-hormonal and metabolic factors

Abete P, JACC 2001;38(5):1357 Rengo G, Am Heart J, 2007;154(2):352



### Benefits of Exercise

- A minimum of 30 minutes of moderate-intensity physical activity (continuous or in 10-minute increments) is required on most (preferably all) days of the week to optimally reduce the risk of CAD
- Equivalent to roughly 1.5 miles/d of brisk walking (approximately 150 kcal/d for an average-sized person)
- No CV workup is needed for this level of exercise unless with known history of CV diseases.
- Lesser amounts of ongoing physical activity, such as 15 minutes/d or 90 minutes/wk, are not associated with a survival benefit

Fletcher GF, 2013, AHA Scientific Statement; Circulation 2013;128:p873

### Benefits of Exercise

see guideline of pre-exercise workup

Fletcher GF, 2013, AHA Scientific Statement; Circulation 2013;128:p873

### Benefits of Exercise

- Patients with known CV diseases:

see guideline of pre-exercise workup

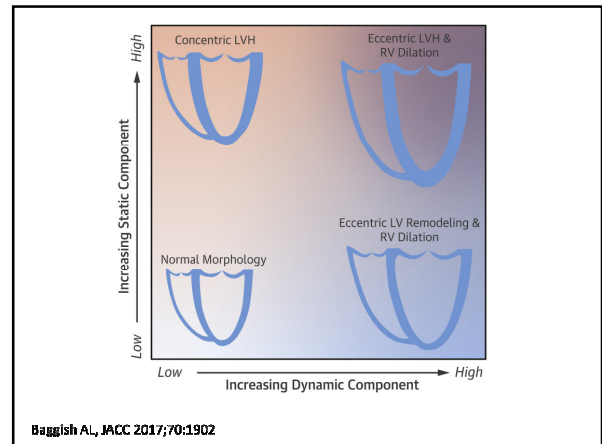
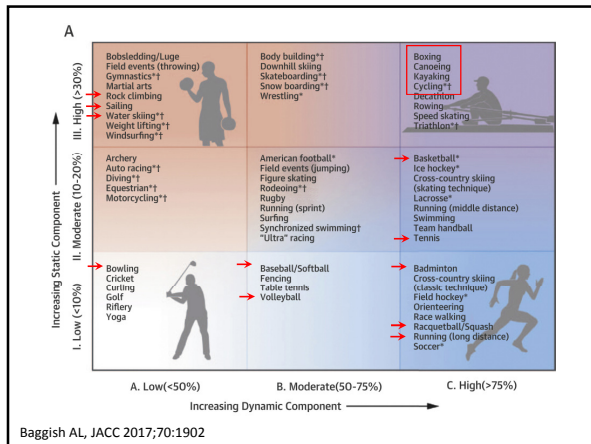
Fletcher GF, 2013, AHA Scientific Statement; Circulation 2013;128:p873

### Static and Dynamic Exercise

- **Static Exercise:**
  - short and forceful skeletal muscle contractions → activating afferent metabolic and mechanical signals → increase vascular resistance and BP
  - during acute bouts of static exercise: CV system need to maintain cardiac output in the presence of increased LV afterload
  - e.g. weight lifting, track and field throwing

### Static and Dynamic Exercise

- **Dynamic or endurance Exercise:**
  - repetitive (often rhythmic) contraction and relaxation of large skeletal muscle groups which requires increased oxidative metabolism
  - intensive of dynamic exercise can be measured by oxygen uptake (VO<sub>2</sub>)
  - during dynamic exercise, CV system needs to increase cardiac output to ensure adequate delivery of metabolic substrate to muscle
  - increase cardiac output by:
    - increase heart rate
    - increase stroke volume
    - reduce systemic vascular resistance



### Static and Dynamic Exercise

- Most sports involve both static and dynamic activities in different mix
- Dynamic exercise:
  - facing bi-atrial and bi-ventricular volume challenges → chamber dilatation (physiological dilatation of all 4 chamber)
  - LV EF may be slightly below normal at rest but normalizes during exercise
  - Stroke volume, not EF, is regulated by resting metabolism
- Static exercise:
  - facing pressure challenges → only LV thickening with minimal effects on the other 3 chambers
  - LVH (often concentric) rarely exceeds 13 mm

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**Do not choose a sport that can hurt you!!**

### Arthritis and Exercise

- During walking, knee joints may bear 2-3x of the body weight → every pound of weight loss → takes away 3 pound of impact on knees
- Impact also transmits to hips, back and neck
- During running, knee joints may bear 4-8x of the body weight → using running as the exercise to lose weight may be a bad idea.
- Shed 3000 calories → lose one pound of weight
  - ingest 150 calories less + burn 150 extra calories
  - lose a pound of weight every 10 days (20 minutes of exercise may burn 150 calories)



### Sudden Death in Athletes

- Coronary artery disease and/or anomaly is the number one killer
- Hypertrophic cardiomyopathy **may be** the number two killer
- Hereditary arrhythmias account for the rest SCD
  - Arrhythmogenic RV cardiomyopathy/dysplasia (ARVC/D)
  - Long QT, short QT
  - Brugada syndrome (pseudo-RBBB)
  - Catecholaminergic polymorphic VT (CPVT)
  - Early repolarization

Corrado D, EHJ, 2010;31:243

### Physiological vs. Pathological ECG Changes

- Physiological changes due to training:
  - Sinus bradycardia, first degree AV block, early repolarization caused by physiological adaptation of cardiac autonomic nervous system
  - Pure voltage criteria for LVH (normal ST, T repolarization) due to increased LV thickness and chamber size (left axis deviation, **intraventricular conduction defects** are considered abnormal)
  - African American athletes have more prevalent and pronounced ECG changes e.g. voltage criteria for LVH and early repolarization
    - 20% black athletes exhibit LVH >12 mm (only 4% in white athletes)
    - 3% black athletes with LVH > 15 mm

Corrado D, EHJ 2010;31:243

### Physiological vs. Pathological ECG Changes

- High endurance training (e.g. cross-country skiing, cycling rowing/canoeing)
  - Great extent of physiological changes (e.g. bradycardia, increased QRS voltage) due to large cardiac output required for these types of exercise
  - In highly trained athletes, sinus bradycardia <30 bpm and asymptomatic sinus pause>2" are not uncommon.
    - Absence of symptoms e.g. dizziness or syncope
    - HR normalizes during exercise
    - Bradycardia reverses with training reduction

Corrado D, EHJ 2010;31:243

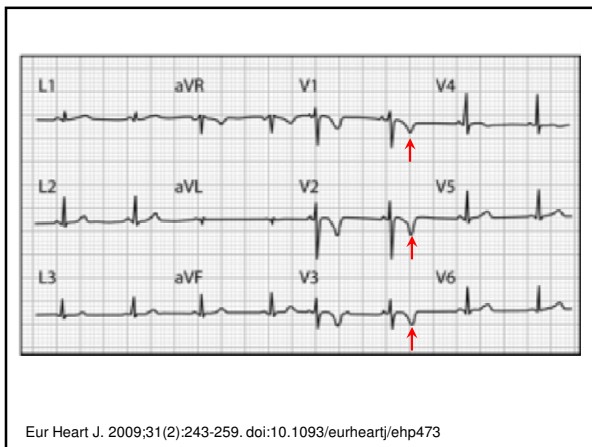
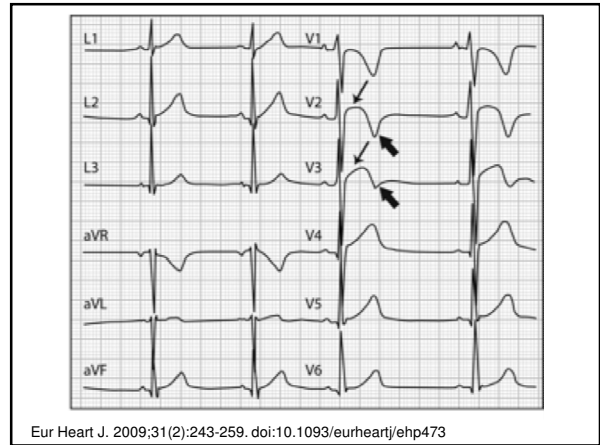
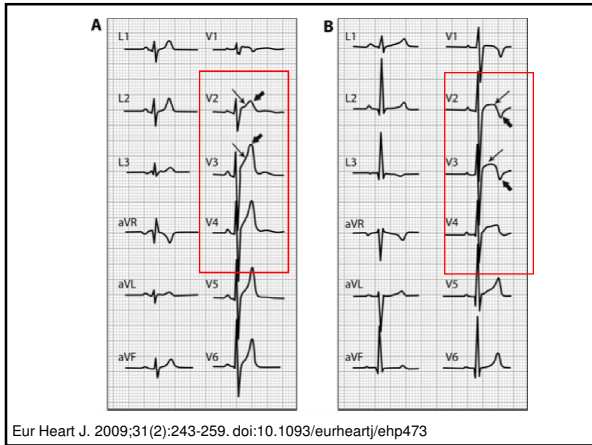
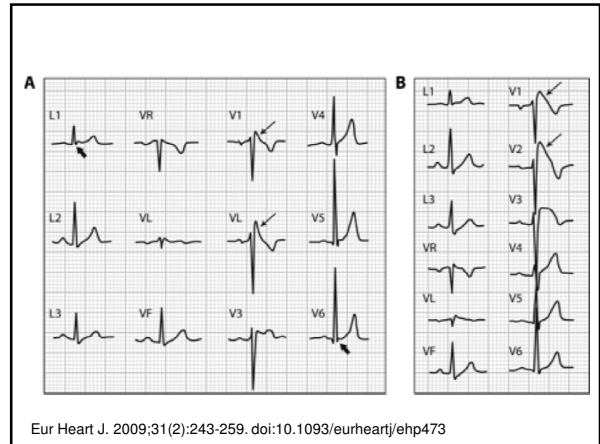
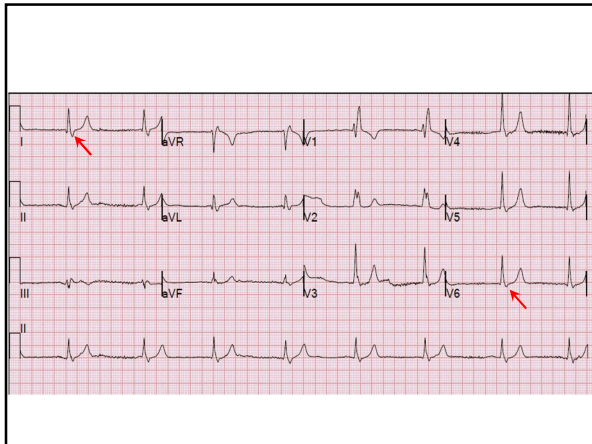
### Brugada Syndrome vs. RBB

Eur Heart J. 2009;31(2):243-259. doi:10.1093/eurheartj/ehp473

### Typical Right Bundle Branch Block

Total QRS complex prolonged (≥0.12 second). Terminal broad S wave in lead I. RSR' complex in lead V1.

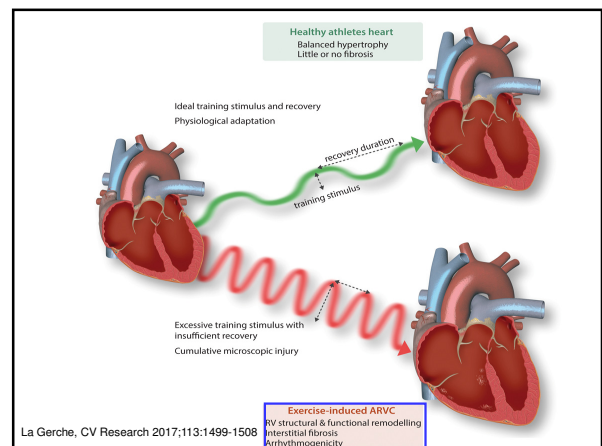
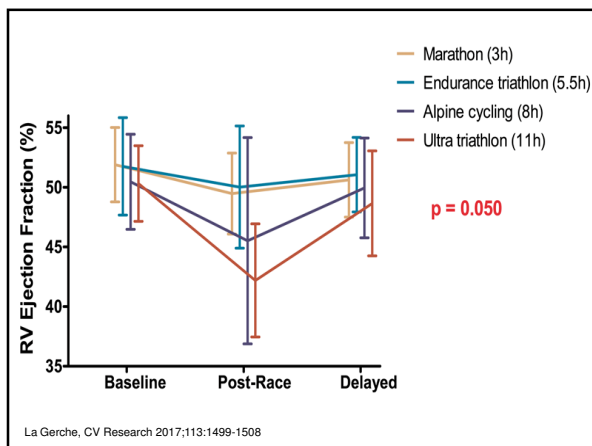
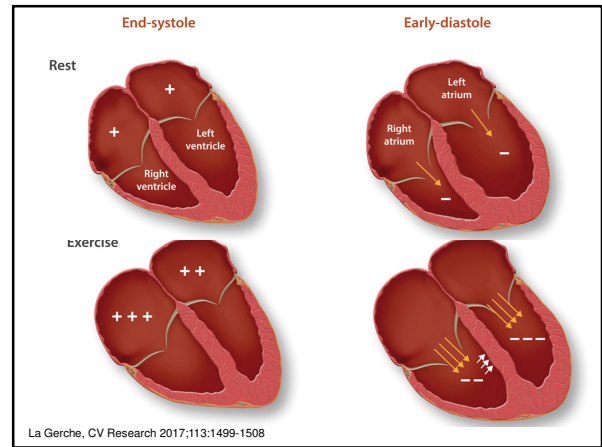
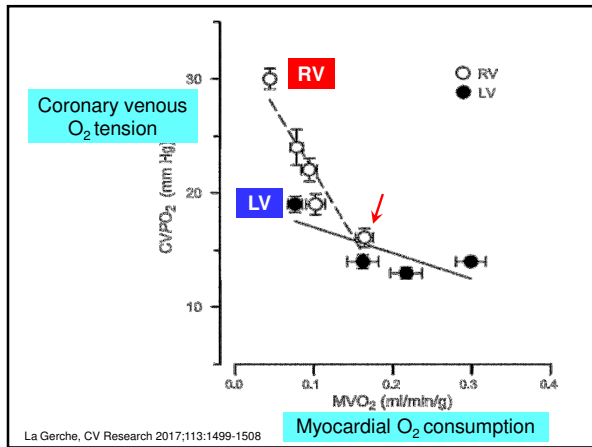
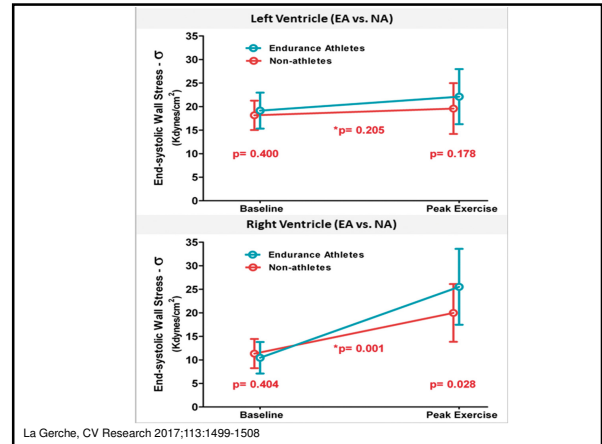
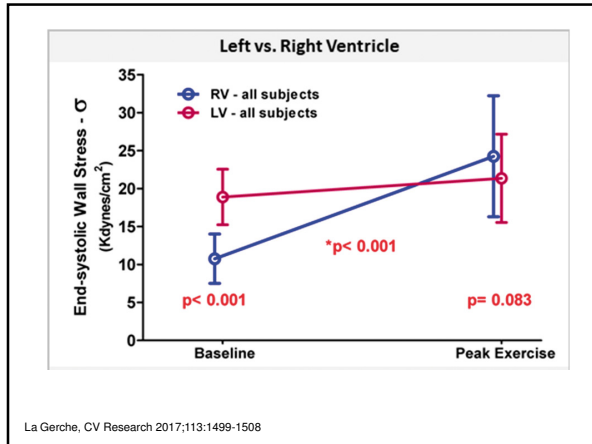




### Differential Impact of Exercise on RV and LV

- Ventricular wall stress determines myocardial O<sub>2</sub> consumption
- During exercise, RV wall stress can increase by as much as 170% as compared with 20-25% increase in LV wall stress
- After an endurance race: a decrease in the RV EF but not the LV EF for nearly a week after the race.
- Endurance athletes also achieve much higher peak pulmonary pressure, leading to higher RV load and stroke work during exercise

Calkins H, Trends in CV Medicine, 2015(25):181



### Exercise-induced ARVD/C

- Disproportional increase in wall stress of RV during exercise causes acute RV dysfunction as a result of micro-trauma
- Repeated RV micro-trauma may result in chronic RV structural and functional changes in *some* endurance athletes.
- LV function is unaffected due to differential responses to physical activity
- Gene-elusive ARVD/C patients are truly gene-elusive?

Calkins H, Trends in CV Medicine, 2015(25):181

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