NCVH 2015

Screening Athletes for Cardiovascular Risks
Sudden Cardiac Death
First, do no harm....... 

- “I wouldn’t ever set out to hurt anyone deliberately unless it was, you know, important – like a league game or something “ 

- Dick Butkus
HANK GATHERS
1967 - 1990
Outline

• Epidemiology
• Etiology of Sudden Cardiac Death
• Pre-participation Physicals
• 26th Bethesda Conference Guidelines for Athletic Participation
Quick abbreviations

- ARVD = arrhythmogenic right ventricular dysplasia
- AS = aortic stenosis
- CAA = coronary artery anomaly
- DC = dilated cardiomyopathy
- HB = heart block
- LQTS = long QT syndrome
- MC = myocarditis
- MVP = mitral valve prolapse
- NMS = neurally mediated syncope
- TCA = tunneled coronary artery
- VP = ventricular preexcitation
THE YOUNG ATHLETE
HOW MANY ARE AT RISK?

• 44 Million youths participate in “sports programs”
• 3.5 Million high school athletes
• 500,000 college athletes in the US
• 10,000 “pro-athletes” in the US
THE YOUNG ATHLETE
THE CAUSES

• Structural Heart Disease
  • Hypertrophic Cardiomyopathy
  • Anomalous Origin of the Coronary Arteries
  • Arrhythmogenic Right Ventricular Cardiomyopathy
  • Myocarditis/Cardiomyopathy
  • Valvular Disease

• The “Channelopathies”

• Marfan Syndrome
THE ADULT ATHLETE
THE CAUSES

• Coronary Artery Disease
• Valvular Heart Disease
• Cardiomyopathy
• “Young Athlete” Disease
THE YOUNG ATHLETE and SUDDEN CARDIAC DEATH

• Rare events
• Without warning, hence SUDDEN
• Devastating
• Occur in healthy individuals
• Attract attention
• Media Bias
THE ADULT ATHLETE

• Harder to define the numbers and risk
  • Heart disease is common among adults
  • Exercise programs vary
  • No organized reporting program
• Marathoners: <1/100,000 on race day
• Tri-athletes: 1.5/100,000 on race day
• Recreational runners: 1/10,000/year or 1/396,000 hours of running
• Nordic Skiers: 1/607,000 hours
• **Individuals with disease are 2-3X more likely to have an event during exertion.**
Causes of death in the US population aged 1 to 21 years.

Mark S. Link, and N.A. Mark Estes III Circulation.
2012;125:2511-2516
Incidence and Causes of Sudden Death in U.S. College Athletes

Figure Legend:

Causes of Sudden Death in NCAA Athletes

Left: All causes. Right: Confirmed CV causes in 47 athletes. *Collapse was virtually instantaneous during physical activity, suggesting underlying CV disease was responsible, but in the absence of a firm post-mortem diagnosis. Not included here are 16 athletes in whom the cause of death remains unresolved. AMI = acute myocardial infarction; ARVC = arrhythmogenic right ventricular cardiomyopathy; CAD = coronary artery disease; CM = cardiomyopathy; CV = cardiovascular; HCM = hypertrophic cardiomyopathy; LQTS = long QT syndrome; MVP = (myxomatous) mitral valve prolapse; NCAA = National Collegiate Athletic Association; SCT = sickle cell trait.

Epidemiology

• College and Professional Athletes
  – 500,000 participants each year

• Competitive Athletics:
  – Several million high school students participate in competitive athletics each year in the United States

• ‘Other’ Organized Sports Participation
  – 25 million children and young adults
Epidemiology

• Incidence of Sudden Cardiac Death:
  – Organized High School/College Athletes
    • 1:134,000/Year (Male) (7.47:million/Year)
    • 1:750,000/Year (Female) (1.33/million/Year)
  – Air Force Recruits
    • 1:735,000/Year
  – Marathon Runners
    • 1:50,000 Race Finishers (Mean Age 37yo)

• In brief, ~ 300 deaths/year.
• But the media attention and legal implications, make these events standout.
Etiology

1) HCM – 36%
2) Coronary Anomalies 17%
3) Increased Cardiac Mass (possible HCM) 10%
4) Ruptured Aorta/Dissect 5%
5) Tunneled LAD 5%
6) Aortic Stenosis 5%
7) Myocarditis 3%
8) Dilated CM 3%
9) Idiopathic Myocardial scarring 3%
10) Arrhythmogenic RV dysplasia 3%

• OTHERS...
  • MVP
  • CAD
  • ASD
  • Brugada Syndrome
  • Commotio Cordis
  • Complete heart block
  • QT prolongation syndrome
  • Ebstein’s anomaly
  • Marfan’s Syndrome
  • Wolff-Parkinson White Syndrome – WPW
  • Ruptured AVM
  • SAH
THE YOUNG ATHLETE and SUDDEN CARDIAC DEATH

• The “Underlying Substrate”: Many of these conditions predispose to lethal arrhythmia

• There can be changes in the athlete’s heart that may increase the risk
  • Hypertrophy (the “muscular heart”)
  • LV and RV dilation (the “enlarged heart”)
  • Increased demand and “adrenalin”
MECHANISM OF SUDDEN DEATH
Ventricular Tachycardia and Ventricular Fibrillation

Normal EKG

Ventricular Tachycardia

Polymorphic Ventricular Tachycardia
Ventricular Fibrillation
HYPERTROPHIC CARDIOMYOPATHY
HYPERTROPHIC CARDIOMYOPATHY

• Affects 1 in 500 individuals
• Genetically determined
  • Sporadic or inherited
  • At least 11 genes, 1400 mutations
• Accounts for 35 – 40% of athletic deaths
• Can be symptomatic/detectable before SCD
• Increased risk with increasing age
• Ventricular arrhythmia is primary cause of death
## Risk Factors for Sudden Cardiac Death in Hypertrophic Cardiomyopathy

<table>
<thead>
<tr>
<th>Major Risk Factors</th>
<th>Possible in Individual Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac arrest (VF)</td>
<td>AF</td>
</tr>
<tr>
<td>Spontaneous sustained VT</td>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td>Family history of premature sudden death</td>
<td>LV outflow obstruction</td>
</tr>
<tr>
<td>Unexplained syncope</td>
<td>High-risk mutation</td>
</tr>
<tr>
<td><strong>LV thickness greater than or equal to 30 min</strong></td>
<td>Intense (competitive) physical exertion</td>
</tr>
<tr>
<td>Abnormal exercise BP</td>
<td></td>
</tr>
<tr>
<td>Nonsustained spontaneous VT</td>
<td></td>
</tr>
</tbody>
</table>

Modified with permission from Maron BJ et al. J Am Coll Cardiol 2003;42:1687-713.
HYPERTROPHIC CARDIOMYOPATHY

- Treatment: Medical
- Treatment: Implantable Defibrillator

“Disqualified” from participation in all but low effort sports (bowling or curling if at Univ of Arkansas) regardless of symptoms, phenotype, treatment.
ECG of HOCM patient
ANOMALOUS ORIGIN OF THE CORONARY ARTERIES

• 15 – 20% of sudden death in young athletes
• In one review of 78 cases of CAA who died of sudden death, 62% of those were asymptomatic
• S/S: Only ~ 1/3 of pts have any symptoms of exertional syncope (<25yo) or exertional cp (25-50yo)
• Exam: usually normal
• Testing:
  • EKG: usually normal or Q-waves showing infarction
  • Cardiac CTA
• Treatment: Medical or Surgical
• May be “cleared” to participate if corrected
Anatomy

NORMAL

Aorta

L. Circ.

L.A.D.

R.C.A.

Pul. A.

ANOMALOUS ORIGIN LEFT

Aorta

L. Circ.

L.A.D.

R.C.A.

Pul. A.

ANOMALOUS ORIGIN OF RIGHT CORONARY ARTERY FROM LEFT SINUS OF VALSALVA
ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

• Arrhythmogenic Right Ventricular Dysplasia, also known as arrhythmogenic right ventricular cardiomyopathy, is characterized by replacement of the right ventricular muscle by fatty and fibrous tissue.

• Arrhythmias of right ventricular origin that range from isolated premature ventricular beats to nonsustained or sustained VT and ventricular fibrillation.
ARRYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY
ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY

- Global or regional right ventricular dysfunction, and late evolution to right or biventricular heart failure.
- Incomplete or complete RBBB
- Inverted T waves in the anterior precordial leads
- Localized prolongation of the QRS complex in leads V1 and V2
- Epsilon waves visible as sharp discrete deflections at the terminal portion of the QRS complex in the anterior precordial leads
- Use QRS width in Lead I which is always <120ms
- Lead III R>S
- S wave upstroke in V1 - V3 >55ms was found in 95 percent of ARVD
ARRHYTHMOGENIC RIGHT VENTRICLE CARDIOMYOPATHY

- Prevalence: 1/1000 – 2000
- Genetic, 30% inherited.
- 5% of sudden death in young athletes
- Can be symptomatic: palpitations, fainting
- Treatment: medical, ICD
- Disqualified from competitive sports
Epsilon wave (arrowhead) in a patient with arrhythmogenic right ventricular dysplasia.

Arrhythmogenic RV Cardiomyopathy
12-lead ECG showing Epsilon wave
Arrhythmogenic RV Cardiomyopathy
(RV conduction delay, inverted T-waves V1-V5)
Sustained Monomorphic VT
49-year-old man with ARVC
Note the thin left ventricular wall (LVW), dilated LV chamber, and depiction of decreased forward blood flow with DCM.
• 5 -10% of sudden cardiac arrests in young athletes
• Causes: “viral”, inherited/genetic, idiopathic
• Can be symptomatic: shortness of breath, palpitations, fatigue/weakness, fainting, chest discomfort
• Treatment: Medical, time, ICD, transplant
• Disqualified from most competitive sports. May return if recover. ICD = no contact sports
MARFAN SYNDROME

- Connective tissue disorder
- Genetic
  - 25% sporadic
  - Autosomal Dominant
- 1/3000 – 5000
In Marfan syndrome, the enlarged aorta can eventually lead to tears or an aneurysm.
COMMOTIO CORDIS
“Agitation of the heart”

- Vulnerable moment
- High force, specific area
- Baseball, hockey, karate
- Kids more vulnerable
- *20% survival*
- Boys > girls
- Prevention key
  - Training to avoid impact
  - ? vests
INHERITED ARRHYTHMIA
and
SUDDEN CARDIAC ARREST
The “Channelopathies”
THE CHANNELOPATHIES AND SUDDEN CARDIAC ARREST

• Long QT Syndrome
• Brugada Syndrome
• Catecholaminergic Polymorphic Ventricular Tachycardia
• Short QT
WHAT IS A CHANNEL?
THE CHANNELOPATHIES AND SUDDEN CARDIAC DEATH

• Inherited/genetic conditions
• Lead to Ventricular Tachycardia and Ventricular Fibrillation
• Evident (variably/intermittently) on EKG
• Cause of Sudden Cardiac Death in both athletes and non-athletes. Exercise does increase the risk in many of these conditions
THE CHANNELOPATHIES
BRUGADA SYNDROME

• Genetic
  • Genetic testing variable
  • Na+ channel

• EKG variable
  • Provocative testing

• Multiple types
• Male > Female
• Avg age at DX: 41
• Fever/hyperthermia trigger
• Night time trigger
• Treatment: ICD, limited medications
• Caution advised for competitive sports with no history of events
• With history of events or ICD low level sports only
Brugada Syndrome
(Typical ST-T abnormality V1-V2)
THE CHANNELopathies
THE LONG QT SYNDROME

Each section of an electrocardiogram (ECG or EKG) is referred to by a letter name: Q, R, S, and T.

Heart is full of blood at Q. Heart contracts at end of T.
LONG QT SYNDROME

• Not rare: 3000 – 4000 deaths/yr in children/adolescents
• Inherited/genetic
  • 12 types/genes, hundreds of different mutations
  • Variable “lethality”
  • AR associated with deafness
• Variable expression
• Acquired form
  • Medications/drugs
  • Electrolyte changes
• Increased risk of SCD with exercise, risk variable based on type
• SCD in athletes: not rare, numbers not clear
• EKG +, gene +, symptom +: Disqualified from competitive sports
Long QT Syndrome in a 16-year-old girl
QT=520 ms; Atrial Tachycardia with 2:1 AV conduction
ACQUIRED LONG QT

- Medications: [www.qtdrugs.org](http://www.qtdrugs.org)
  - Antiarrhythmics
  - Antibiotics: Levaquin, Zithromax (Z pack), erythromycin
  - Antidepressants: Tricyclics, Prozac, Celexa
  - Tamoxifen
  - diuretics
  - 140 other drugs
- Methadone
- Combinations of drugs
- Electrolytes: Low K+, Mg++, Ca++
- Genetic + Drugs, ? Unmasked congenital form
- Reversible
ACQUIRED LONG QT AND EXERCISE

• ? Drug + exercise interaction
• ? Electrolyte changes with exercise
  • Dehydration
  • Excessive “free water” intake
  • Losses with sweating
  • Diuretics
  • ? Greater risk with endurance events

• The Perfect Storm: Congenital substrate + drugs + exercise
THE CHANNELOPATHIES: CATECHOLAMINERGIC POLYMORPHIC VT
CPVT

- Genetic, at least 2 gene mutations
- Inherited
- Emotional and physical triggers. Symptoms: dizziness and syncope
- Usually presents in childhood and adolescence
- Treatment: Medical therapy, ICD + medical, Sympathectomy, Medical therapy for gene + asymptomatic.
- Generally recommend against competitive sports, ICD precludes contact sports
- Beta blockers
OTHER ARRHYTHMIA
WOLFF PARKINSON WHITE

- 1/400
- Often Incidental finding
- Can present with symptoms
- Often first diagnosed in adulthood
- Risk of V-fibrillation
- Risk stratify asymptomatic Pts
- Ablation
- OK to participate in competitive sports once treated
HOW DO WE FIND THESE - SCREENING YOUNG ATHLETES

• Recommendations vary widely internationally and within the US
• Recommendations vary widely based on level of participation
• Not clear if definitely reduces risk
  • Findings variable with time
  • Variable age of onset
• These are relatively rare diseases
SCREENING GOAL

- To identify those at risk
- Prevent injury and lethal events

TO ASSIST YOUNG ATHLETES AND THEIR FAMILIES IN MAKING RATIONAL DECISIONS REGARDING THE RISK OF ATHLETIC PARTICIPATION
Screening requirements

- In the US competitive athletes are screened by means of history and physical examination.
- Only Europe mandates a resting ECG.
- In 1982 the incidence of SCD in Italy was 4.2/100,000 athletes. In 2004 the incidence of SCD decreased markedly to 0.9/100,000. Due to Arrhythmogenic RV dysplasia.
Pre-Participation Physicals

• History
  – Screen for medications and drugs of abuse that can have potential cardiotoxic effects (Beta agonists, Theophylline, TCA’s, Macrolides, Pseudoephedriine, Phenylpropanolamine, Tobacco, Alcohol, Cocaine, Amphetamines, Ephedrine, and Anabolic Steroids)

• Questions
  – Have you ever passed out during or after exercise?
  – Have you ever been dizzy during or after exercise?
  – Have you ever had chest pain during or after exercise?
  – Do you get tired more quickly than your friends do during exercise?
  – Have you ever had racing of your heart or skipped heart beats?
THE PREPARTICIPATION EXAM

• Review for symptoms
  • Dizziness or fainting, shortness of breath, palpitations, chest discomfort, can’t keep up

• Family History
  • Premature death
  • “Death under unusual circumstances”

• Physical exam
  • Murmurs, build, pulses
Pre-Participation Physicals

• Yes, more questions
  – Have you had high blood pressure or high cholesterol?
  – Have you ever been told you have a heart murmur?
  – Has any family member or relative died of heart problems or sudden death before age 50?
  – Have you had a severe viral infection within the last month (ie. Myocarditis or mononucleosis)
  – Has a physician ever denied or restricted your participation in sports for any heart problems?
Flow Chart of the Italian Protocol of Cardiovascular Pre-Participation Screening

Young competitive athletes are defined as individuals 12 to 35 years of age who are engaged in a regular fashion in exercise training as well as participating in official athletic competitions. First-line examination includes family history, physical examination, and 12-lead electrocardiography (ECG); additional tests are requested only for subjects who have positive findings at the initial evaluation. Angio/EMB = contrast angiography/endomyocardial biopsy; EPS = electrophysiologic study with programmed ventricular stimulation; MRI = magnetic resonance imaging. Reprinted, with permission, from Corrado et al. (3).
SO, WHAT ABOUT EKGs

- Not recommended routinely in US
- Required in Europe
- Controversial
  - Not clear it helps
  - Athletes often have EKG changes that are “normal”
  - False negatives, False positives....unnecessary exclusions
  - Cost of EKGs, Cost of additional testing, Cost of disqualifying athletes
  - Estimated $80,000 to find one case
• EKG’s

  – Findings in Athletes considered WNL
    • Sinus Bradycardia – as low as 30-40 bpm
    • Various A/V blocks occur in up to 33% of athletes
      – First Degree (PR>0.2) – Most Common
      – Second Degree (Mobitz-1 or Wenkeback)
    • Increased R or S wave voltage without Left axis deviation, QRS prolongation, or LAE
    • U-waves with up-sloping ST segments and normal T waves
    • Incomplete RBBB
### EKG’s

<table>
<thead>
<tr>
<th>Electrocardiogram</th>
<th>Athletes</th>
<th>Controls</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus bradycardia</td>
<td>80%</td>
<td>19%</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>Sinus arrhythmia</td>
<td>52%</td>
<td>9%</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>PR interval</td>
<td>153 msec</td>
<td>140 msec</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>QRS interval</td>
<td>92 msec</td>
<td>89 msec</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>QT interval</td>
<td>391 msec</td>
<td>379 msec</td>
<td>$P = 0.002$</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>45%</td>
<td>10%</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>Left atrial enlargement</td>
<td>14%</td>
<td>1.2%</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>Right atrial enlargement</td>
<td>16%</td>
<td>2%</td>
<td>$P &lt; 0.0001$</td>
</tr>
<tr>
<td>Minor T-wave inversion</td>
<td>4%</td>
<td>4%</td>
<td>NS</td>
</tr>
<tr>
<td>ST elevation</td>
<td>43%</td>
<td>24%</td>
<td>$P &lt; 0.0001$</td>
</tr>
</tbody>
</table>

Adapted with permission.$^{17}$
Common ECG “Abnormalities” in Athletes

Classification of ECG Abnormalities in the Athlete

Common electrocardiography (ECG) abnormalities: up to 80% of trained athletes exhibit ECG changes such as sinus bradycardia, first degree atrioventricular (AV) block, early repolarization, incomplete right bundle branch block (RBBB) and pure increase of QRS voltages (Group 1). Such common ECG changes are the consequence of the physiologic cardiovascular adaptation to sustained physical exertion and do not reflect the presence of an underlying cardiovascular disease. Therefore, they are not associated with an increase of cardiovascular risk and allow eligibility to competitive sports without additional evaluation. Uncommon ECG abnormalities: this subset includes uncommon ECG patterns (<5%) such as ST-segment and T-wave repolarization abnormalities, pathological Q waves, intraventricular conduction defects, and ventricular arrhythmias (Group 2). These ECG abnormalities are unrelated to athletic conditioning and should be regarded as an expression of possible underlying cardiovascular disorders, notably cardiomyopathies and cardiac ion channel diseases, and thus associated with an inherent increased risk of sudden arrhythmic death. Modified from Corrado et al. (22). LBBB = left bundle branch block.

Figure Legend:

Classification of ECG Abnormalities in the Athlete

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Mandatory Electrocardiographic Screening of Athletes to Reduce their Risk for Sudden Death: Proven Fact or Wishful Thinking?

Arie Steinvil, Tamar Chundadze, David Zeltser, Ori Rogowski, Amir Halkin, Yair Galily, Haim Perluk, and Sami Viskin.

Tel-Aviv Sourasky Medical Center and Sackler School of Medicine, Tel Aviv University; Zinman College of Physical Education & Sports Sciences at the Wingate Institute; and Mediton Medical Center, Tel-Aviv, Israel.
Results

- Identified 24 events of sudden death or cardiac arrest in athletes.
- All of them were males, aged 12-44 (23.9 ± 8.8) years and the majority were football (soccer) players.
- Of these 24 events, 11 events occurred during the 12 years preceding the 1997 sports legislation mandating pre-participation screening and 13 occurred during the 12 years that followed.
Annual incidence of sudden cardiac death expressed per 100,000 person-years in the 3 studies evaluating the effects of screening on the mortality of athletes over time.

The Italian study\(^2\) (pink) concluded that ECG screening (started in 1982) significantly reduced the incidence of sudden death by comparing sudden death rates in the 2-years pre-screening period (Point A to B) to the post-screening period (B to F). The present study (green) compared the 12 years before screening (C to E) to the 12 years after the onset of mandatory ECG screening (E to G). *Had we limited our comparison of the post-screening period to the 2-year period preceding the enforcement of screening in Israel (D to E vs. E to G) (as performed in the Italian study) we would have erroneously concluded that screening saved lives of athletes.* A Minnesota study\(^3\) (yellow) shows a low mortality rate in a population of athletes not undergoing systematic ECG screening.

\(^2\)Corrado, *JAMA* 2006;296:1593
\(^3\)Maron, *Am J Cardiol* 2009;104:276.
1. In this retrospective study, mandatory screening of athletes with resting electrocardiogram and exercise effects had no apparent effect on the yearly cardiac-arrest event-rates among competitive athletes.

2. Importantly, had we limited our comparison of the post-screening period to the 2-year period preceding the enforcement of screening in Israel (as performed in the Italian study) we would have erroneously concluded that screening saved lives of athletes.
LOWERING RISK IN THE YOUNG ATHLETE

- Preparticipation Exam
- Parental involvement in children and adolescents
- Coaches/trainer/athlete awareness
- Symptom awareness
- Workout/practice design
- Hydration/electrolyte replacement
- AEDs in close proximity when feasible and AED training
- CPR training of coaches/trainers/athletes
WHAT ABOUT US - THE ADULT ATHLETE

CARDIOVASCULAR DISEASE IS THE PRIMARY CAUSE OF SUDDEN CARDIAC DEATH IN ADULT ATHLETES
THE ADULT ATHLETE

• Primary Cause: Coronary Artery Disease
• Cardiomyopathy
• Vascular Disease
• Arrhythmia
• Valvular Heart Disease
THE ADULT ATHLETE

The adult athlete can still have almost any of the conditions of the young athlete.
CORONARY ARTERY DISEASE

STILL NUMBER ONE!
WHAT IS THE RISK?

• 800,000 Heart attacks/year
• 400,000 Sudden Cardiac Death
• **Sudden Death:** First symptom in 50%
• 2 – 3 X as likely to suffer a cardiac event during exercise in those with disease
• Numbers during exercise unknown
  • Marathon Risk: 1/50,000 – 100,000/race
  • 2012: 550,000 finished a marathon
  • 2011: 500,000 started their first marathon
• Nobody is keeping track outside of organized events
• Odds are there are many cardiac events during unorganized exercise that are not reported
DETERMINANTS OF RISK

1. Probability of Cardiac Disease

2. Intensity and Duration of Exercise

RISK INCREASES WITH INCREASED RISK OF UNDERLYING CVD, INTENSITY, DURATION
DETERMINING YOUR RISK

• AGE
• CVD RISK FACTORS
• HISTORY OF EXERCISE
• SYMPTOMS
• FAMILY HISTORY OF SUDDEN DEATH, FAINTING, ARRHYTHMIA, DEATH UNDER UNUSUAL CIRCUMSTANCES
• CORONARY ARTERY CALCIUM SCORING
WHO NEEDS SCREENING

• Everyone over 20 years old should know their risk factors for CVD and periodically re-evaluate them.
• Everyone should discuss with their physician their exercise routine (Physicians rarely ask).
• A “baseline” history and exam is indicated as part of the risk evaluation for exercise.
• It is reasonable for adults to have at least one baseline EKG.
RISK LEVEL

• **Low Risk**: man < 45, woman < 55, no CVD risk factors, no symptoms, no worrisome history

• **Moderate Risk**: man > 45, woman > 55, 1 or 2 CVD risk factors (not DM)

• **High Risk**: History or Symptoms of CVD, DM, age > 65, > 2 CVD risk factors
WHO NEEDS PRE-EXERCISE TESTING

INENSITY

LOW

LOW

MOD

MORDERATE

HIGH

RISK LEVEL
American Heart Assoc. Guidelines:

exercise ECG screening test

men > 40-45 years of age

women > 50-55 years of age (or postmenopausal)

with 1 independent coronary risk factor

hypercholesterolemia

or dyslipidemia including low HDL

systemic hypertension

current or recent cigarette smoking

diabetes mellitus

a history of myocardial infarction or SCD in a first-degree relative aged < 60 years.
### 26th Bethesda Conference Guidelines for Athletic Participation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertrophic cardiomyopathy</td>
<td>Exclusion from most competitive/noncompetitive sports, with possible exception of low-intensity sports, regardless of medical treatment, absence of symptoms, or implantation of defibrillator.</td>
</tr>
<tr>
<td>Coronary artery abnormalities</td>
<td>Exclusion from all competitive sports. Participation may be considered 6 months after surgical correction and after exercise stress testing.</td>
</tr>
<tr>
<td>ARVD</td>
<td>Exclusion from all competitive sports.</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>Exclusion if history of syncope associated with arrhythmia, family history of mitral valve prolapse and sudden death, documented arrhythmia, or moderate to severe mitral regurgitation.</td>
</tr>
<tr>
<td>Ebstein anomaly</td>
<td>Severe disease precludes participation in all sports. After surgical repair, low-intensity sports are permitted if tricuspid regurgitation is absent or mild, heart size is normal, and no arrhythmias are present on Holter monitoring and stress testing.</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>Exclusion from contact sports. Patients with aortic regurgitation and marked dilation of aorta are excluded from all competitive sports. Others may participate in low-intensity sports, with biannual echocardiography.</td>
</tr>
<tr>
<td>Long QT syndrome</td>
<td>Exclusion from all competitive sports.</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Athletes with history of myocarditis in previous 6 months are excluded from all competitive sports.</td>
</tr>
<tr>
<td>Wolff-Parkinson-White syndrome</td>
<td>Patients with normal exercise testing ± electrophysiologic study may be eligible for participation in all sports.</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>Individual risk assessment based upon ejection fraction, exercise tolerance, presence of inducible ischemia or arrhythmias, and presence of hemodynamically significant coronary stenoses on angiography.</td>
</tr>
</tbody>
</table>
AND THAT’S ALL I HAVE TO SAY ABOUT THAT.