ECG Advanced Cardiac Screening of Athletes

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DISCLOSURE: Neither Dr. Cole nor any member of his immediate family has a financial relationship or interest with any proprietary entity producing health care goods or services. The content of his material(s)/presentation(s) in this CME activity will not include discussion of unapproved or investigational uses of products or devices.

LEARNING OBJECTIVES:

At the conclusion of this presentation, the participant should be able to:

1. Discuss past, present and future of cardiac screening of athletes.
2. Discuss risk and benefits of adding ECG screening to our current strategies
3. Discuss current ECG guidelines for athletes.
Advanced Cardiac Screening in Athletes

Eric L. Cole, MD FAAFP
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Overview

- We will briefly discuss past, present, and future of cardiac screening
- Discuss the risks and benefits of adding ECG screening to our current strategies
- Discuss how to implement an ECG screening effectively
- Discuss the current ECG guidelines for athletes
PPE goals

- Primary Objective is prevention of sports related death in athletes
  - The current questions were developed by expert opinion and have never been validated scientifically
  - Limited evidence supporting the effectiveness of our current PPE screening
- Death due to intrinsic cardiac condition remains the leading cause of mortality during sport
12 lead ECG

- No evidence currently that adding to current PPE protocol will prevent SCA/SCD

- However the standard PPE alone has a low sensitivity for detecting conditions associated with SCA/D – but newer data suggests that when PROPERLY INTERPRETED ECGS ALONG WITH AVAILABLE SKILLED CARDIOLOGY SERVICES, screening is improved if the measurable endpoint is detecting conditions that are associated with SCA/D
Current thoughts

- The AHA currently does not support mandatory ECG screening, but has supported ECG screening programs when they are well-designed and properly implemented with adequate cardiology resources.

- Uncertain how many NCAA institutions offer advanced cardiac screening beyond standard PPE
  - Gaps are likely based as much on infrastructure support and available expertise as it is philosophical and medical variation
Current trends

- The opinion now of many (including several Sports Medicine leaders) is that we can not effectively screen athletes with H&P alone.

- It is thought that ECG screening can lead to EARLY detection of cardiac conditions associated with SCA/D.

- But there are certain components that are required:
  - Apply modern ECG standards
  - Dedicated cardiology back up
Risk of SCD

- Although regular exercise is beneficial for prevention of CVD, athletes involved in competitive sports are at least 2.8X more likely to die from SCD than their sedentary counterparts.
  - Not that exercise is the cause, but that it is the trigger for those that harbor undiagnosed cardiac pathology to have a serious ventricular arrhythmia.
Incidence

• Remains controversial
  • Due to reporting methodology, reliability of case identification, and accuracy of population denominators
  • Current reasonable estimate is 1:50,000
    • Males > females
    • African descent > others
    • African males at greatest risk
Rationale

- Exercise is considered the trigger for SCD with underlying cardiac disorders
- Early detection can then lead to mitigation of this risk through activity restriction and medical interventions
- AHA and ESC do agree “that compelling justification exists for cardiovascular pre-participation screening on medical, ethical, and legal grounds”
Adding ECG effective?

- 5x more SN than history and 10X than physical
- 47,137 athletes and 15 studies reviewed
- SN/SP ECG 94/93%
  - History 20/94%
  - Physical 9/97%
- False-positive rate of ECG 6%
- False positive rate of history 8% and PE 10%
- Conclusion – ECG is the most effective strategy for screening for CVD in athletes.
<table>
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<th>Syndrome</th>
<th>ECG</th>
<th>Hx + PE</th>
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<tr>
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<td>≤90%</td>
<td>&lt;10%</td>
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<td>ARVC</td>
<td>60–80%</td>
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<td>Pre-excitation syndrome (WPW)</td>
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<tr>
<td>Congenital coronary artery anomalies</td>
<td>&lt;10%</td>
<td>&lt;10%</td>
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Impact of Screening

- Not a lot of Data
- The Italian experience; Corrado et al. did show an 89% reduction in sudden death; 3.6 per 100,000 person years to 0.4 per 100,000 person years over a 26 year period.
- Death from cardiomyopathies (HCM and ARVC) was greatly reduced
- This type of study has yet to be reproduced
Limitations to Screening

- Cost
- False Positive Screens
- False Negative Screens
  - CAD and other undetected anomalies
- Lack of properly trained clinicians
- Legal Considerations
If the decision is made to screen with ECG

- Pre-screening planning and coordination
  - Getting buy in from all involved
    - Team Physicians, administrators, coaches, trainers, Cardiology consultants
  - Decision must me made as to who to screen
    - All athletes vs. Highest risk groups
    - Must be agreed upon from the beginning
  - ECG standards must be agreed on
  - Avenues for prompt secondary testing
  - Training --- lead placement and interpretation
ECG interpretation

- Free modules on BMJ Learning
- Learning.bmj.com/ECGathletes
- Criteria specific ECG machines
- Refined criteria > Seattle criteria > ESC criteria
- Commitment to evolve quality standards and continuous reassessment as improvements are made
  - Best Practices
Cost

- Equipment
  - ECG machine, leads, paper
  - ECG overread
  - Cardiology services and additional testing
  - At this point should be considered preventative services
  - Secondary Costs would be more diagnostic services
Integrating ECG Screening

- Standardized history and physical examination + ECG
- Models
  - ECG station during PPE
  - ECG after examination
  - ECG off-site
  - Private Physician office
- ECG overread
  - PCP/ Sports med point of care / cardiology remotely
Secondary Testing

- For the Athletes that have positive screen – history, physical, or ECG must have adequate cardiology oversight

- Part of the planning should identify a predetermined avenue for efficient testing and consultation
  - Echo or stress echo
  - Cardiac MRI
  - Cardiac Catheterization
Vulnerable Athlete Response Plan

- Asif; J Electrocardiol 2015
- In pre-planning phase identify support mechanisms
- Provide ongoing support
- Involve a multi-disciplinary team
  - Team or personal PCP, cardiologist, psychologist, counselor, ATCs, and genetic counselors
PPE

- AHA 14 element recommendation
- PPE monograph 4th edition
- European Society of cardiology
- International Olympic Committee
- FIFA
**Personal history**
1. Chest pain/discomfort/tightness/pressure related to exertion
2. Unexplained syncope/near-syncope
3. Excessive and unexplained dyspnoea/fatigue or palpitations, associated with exercise
4. Prior recognition of a heart murmur
5. Elevated systemic blood pressure
6. Prior restriction from participation in sports
7. Prior testing for the heart, ordered by a physician

**Family history**
8. Premature death (sudden and unexpected or otherwise) before 50 years of age attributable to heart disease in one or more relatives
9. Disability from heart disease in a close relative <50 years of age
10. Hypertrophic or dilated cardiomyopathy, long QT syndrome or other ion channelopathies, Marfan syndrome or clinically significant arrhythmias; specific knowledge of genetic cardiac conditions in family members

**Physical examination**
11. Heart murmur
12. Femoral pulses to exclude aortic coarctation
13. Physical stigmata of Marfan syndrome
14. Brachial artery blood pressure (sitting position)

Table 9.1 The 14-element AHA recommendations for pre-participation cardiovascular screening of competitive athletes

*a Parental verification is recommended for high-school and middle-school athletes

*b Judged not to be of neurocardiogenic (vasovagal) origin; of particular concern when occurring during or after physical exertion

*c Refers to heart murmurs judged likely to be organic and unlikely to be innocent; auscultation should be performed with the patient in both the supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction

*d Preferably taken in both arms
Normal ECG Findings in Athletes
Normal Findings

- Sinus Bradycardia
  - Due to increased vagal tone in a well-trained athlete, especially endurance athletes, in the absence of symptoms, a heart rate >30 should be considered normal

- Sinus Arrhythmia
  - HR increases during inspiration and decreases during expiration

- First degree Atrioventricular Block
Normal Findings

- Second degree AV block (Mobitz type 1 or
  - Wenckebach phenomenon)
- Incomplete RBBB
- Junctional Escape Rhythm
- Ectopic Atrial Rhythm
- QRS voltage criteria for LVH and RVH
- Early Repolarization
Normal Findings

- Convex (domed) ST segment elevation combined with T wave inversion in leads V1-V4 in black/African athletes
- T wave inversion in V1-V3 in young adolescent athletes < 16 years old
- LVH and RVH by voltage criteria in isolation are considered part of the normal athlete heart
29 year old asymptomatic soccer player demonstrating sinus rhythm, early repolarisation with ST elevation (arrows) and peaked T waves. These are common findings in training.
Figure 2. ECG shows marked sinus bradycardia (41 bpm) and sinus arrhythmia.
Figure 5. ECG shows an ectopic atrial rhythm. The atrial rate is 63 beats per minute and the P wave morphology is negative in leads II, III, and aVF (arrows), also known as a low atrial rhythm.
Figure 4. A 28 year old asymptomatic white handball player demonstrating a junctional escape rhythm. Note the constant RR interval between beats.
Figure 6. ECG shows 1st degree AV block (PR interval >200 ms). The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex.
Figure 7. ECG shows Mobitz Type I (Wenckebach) 2nd degree AV block demonstrated by progressively longer PR intervals until there is a non-conducted P wave (arrows) and no QRS complex. Note the first PR interval after the dropped beat is shorter than the last conducted PR interval prior to the dropped beat.
Figure 8. ECG showing incomplete right bundle branch block with an R’ wave in V1 (arrow) and a QRS duration of 110 ms.
Figure 11. ECG from a 29 year old asymptomatic soccer player demonstrating early repolarisation (J point and ST elevation) in I, II, aVF, V2-V6 (arrows), and tall, peaked T waves (circles). These are common, training related findings in athletes and do not require more evaluation.
Figure 13. ECG from a 19 year old asymptomatic soccer player demonstrating voltage criteria for LVH (S-V1 + R-V5 > 35 mm). Note the absence of left atrial enlargement, left axis deviation, ST depression, T wave inversion, or pathologic Q waves. Increased QRS amplitude without other ECG abnormalities is a common finding in trained athletes and does not require additional testing.
Figure 7. ECG from a 17 year old asymptomatic black/Afro-Caribbean soccer player demonstrating "domed" ST elevation followed by T wave inversion in leads V1-V4 (circles). This is a normal repolarisation pattern in black/Afro-Caribbean athletes.
Figure 17. Panel A shows normal variant repolarisation changes in a black/Afro-Caribbean athlete characterised by domed ST segment elevation and T wave inversion in V1-V4. Panel B shows pathologic T wave inversion in V1-V3. Note the isoelectric ST segment. The absence of ST segment elevation prior to T wave inversion makes this ECG abnormal. Additional testing is required to rule out ARVC.
Figure 18. Panel A shows normal variant repolarisation changes in a black/Afro-Caribbean athlete characterised by domed ST segment elevation and T wave inversion in V1-V4. Panel B shows a dowsloping ST segment elevation followed by T wave inversion in V1-V2 suggestive of a Brugada pattern ECG. Note the high take off and absence of upward convexity ("dome" shape) of the ST segment distinguishing this from the repolarisation variant in black/Afro-Caribbean athletes.
Abnormal Findings

Cardiomyopathy
Abnormal Findings

- Cardiomyopathy
  - Account for the majority of sudden deaths
  - 98% of HCM have abnormal ECG
  - 80% of ARVC have abnormal ECG
  - Demographic differences must be understood
T Wave Inversion

- Not all pathological – III, AVR, and V1 ok
- Is the most common ECG feature of myocardial disease – HCM, ARVC, DCM, LVNC, myocarditis
- Anterior TWI = ARVC
- Inferolateral TWI = HCM
- Careful with juvenile ECG pattern though (V1-V3)
  - 10-15% up to 12 and 2.5% 14-15
Anterior T wave inversion

- ATWI beyond V2 in caucasian older than 15 is rare and should prompt w/u of ARVC

- In African Athletes T wave inversion in V1-V4 that is preceded by J point elevation and convex dome shaped ST segment elevation is ok

- However ATWI preceded by isoelectric or depressed ST segment in athletes > 16 yoa requires investigation for ARVC
Anterior TWI

- Athletic Heart – J point elevation, ST elevation, or biphasic T waves

- ARVC – absence of J point elevation, depression of ST segment, low limb lead voltages, prolonged S wave upstroke, ventricular ectopy with LBBB morphology, and epsilon waves

  - Evaluation could include echo, CMRI, holter, exercise ECG, and signal average ECG
Figure 11  ECG from a 30-year-old patient with ARVC showing anterior TWI in V1-V3 preceded by a flat or downsloping ST segment without J-point elevation. PVCs are also present. ARVC, arrhythmogenic right ventricular cardiomyopathy; PVC, premature ventricular contraction.
Figure 6  Normal and abnormal patterns of TWI. (A) Anterior TWI in V1-V3 in a 12-year-old asymptomatic athlete without a family history of SCD considered a normal ‘juvenile’ pattern. (B) TWI in V1-V4 in a 17-year-old asymptomatic mixed race (Middle-Eastern/black) athlete without a family history of SCD. This is a normal repolarisation pattern in black athletes. (C) Biphasic TWI in V3 in a 31-year-old asymptomatic black athlete without a family history of SCD. Anterior biphasic T waves are considered normal in adolescents <16 years old and in adults when found in a single lead, most commonly V3. (D) Abnormal TWI in V1-V6 in an adult symptomatic former soccer player with genetically confirmed ARVC and a positive family history of SCD (brother died at 26 years of age). (E) An ECG demonstrating the type 1 Brugada pattern with high take-off ST elevation ≥2 mm with downsloping ST segment elevation followed by a negative symmetric T wave in V1-V2. (F) Inferolateral TWI in leads I, II, III, aVF, V2-V6 and ST segment depression in leads II, aVF, V4-V6 in a 31-year-old asymptomatic professional soccer referee. These markedly abnormal findings require a comprehensive evaluation to exclude cardiomyopathy. ARVC, arrhythmogenic right ventricular cardiomyopathy; SCD, sudden cardiac death; TWI, T wave inversion.
**Lateral or Inferolateral T wave inversion**

- Regardless of race, LTWI in two or more leads
  - I, AVL, V5, V6 - lateral
  - II, III, AVF – inferior

- Requires comprehensive evaluation
  - Echo, CMRI, Holter, Exercise ECG

- They can be deep (>2mm) and extend to the inferior leads, but even < 2mm inversion needs further investigation

- If the w/u is negative – follow them carefully!
Figure 3  ECG from a patient with HCM demonstrating QRS voltage criteria for LVH in association with deep TWI and ST segment depression predominantly in the lateral leads (I, aVL, V4–V6), voltage criteria for left atrial and right atrial enlargement and left axis deviation. HCM, hypertrophic cardiomyopathy; LVH, left ventricular hypertrophy; TWI, T wave inversion.
Lone Inferior TWI

- Uncertain, but proceed with echo and possibly CMRI
- Can be found in 6% of black athletes and 2% whites, but can also be found in cardiomyopathy, so until further data comes out, consider abnormal, and work up
- Flat or Biphasic T waves
  - Work up if the negative component is greater than 1 mm
ST segment depression

- Need high quality ECG to determine the isoelectric line
- Any ST depression (in excess of 0.05mv (0.5mm)), especially in leads I and AVL warrants further evaluation echo at minimum, CMRI
- Will be seen in up to 50% of patients with cardiomyopathies, especially HCM
- Rare (<0.5%) in healthy athletes
Pathological Q waves

- Can be difficult due to lack of consensus on definition
- >40ms; > 3mm depth; > 25% R wave in 2 contiguous leads
- Exclude leads III and AVR
  - Check lead placement and repeat ECG
- Echo to r/o CM; stress test if > 30 yoa
  - CMRI if associated with other abnormalities
Figure 14  ECG from an 18-year-old female swimmer demonstrating deep and wide pathological Q waves in V4-V6, I and aVL. Diagnostic testing revealed hypertrophic cardiomyopathy.
LBBB

- Not common in young healthy athletes (1/1000), but is commonly seen in cardiomyopathies, especially DCM, and ischemic heart disease

- A new LBBB is a predictor of all cause mortality

- Any LBBB must be worked up comprehensively
  - ECHO
  - CMRI

- Think DCM and HCM, possibly even Sarcoid
Figure 15  ECG with complete LBBB demonstrating a QRS $\geq$120 ms, predominantly negative QRS complex in lead V1, upright R wave in leads I and V6, and ST segments and T waves in the opposite direction of the QRS. LBBB is always an abnormal finding in athletes and warrants a comprehensive evaluation to exclude myocardial disease. LBBB, left bundle branch block.
Non specific Intraventricular conduction delay

Uncertain, but if > 140ms, evaluate

Especially if concerning family history

Start with echocardiogram
Epsilon Waves

- Distinct low amplitude deflection at end of QRS complex and onset of T wave in leads V1-V3
- Highly specific marker for ARVC and therefore a major criteria for diagnosis
- Marker of advanced disease and unlikely to be found alone
- Must be worked up with Echo, CMRI, holter, exercise ECG, and signal average ECG
Figure 16  ECG in a young athlete with arrhythmogenic right ventricular cardiomyopathy showing several abnormal features including anterior T wave inversion (V1–V4) preceded by a non-elevated J-point and ST segment, an epsilon wave in V1 (magnified and marked with arrow), delayed S wave upstroke in V2, and low voltage (<5 mm) QRS complexes in limb leads I and aVL.
Abnormal Findings

Primary Electrical Disease
Ventricular Pre-excitation

- When accessory pathway bypasses the AV node and results in abnormal conduction to the ventricle.

- Shortens the PR interval and widens the QRS

- Wolf-Parkinson-White (WPW)
  - PR < 120ms; delta wave – slurring of initial QRS; and QRS duration > 120ms
  - 1-4/1000 athletes

- Can predispose to SCA/D from predisposing to VF
WPW

- Short PR without delta wave or prolonged QRS does not need further eval
- Those diagnosed with WPW should undergo echo, stress ECG, and likely will get EP study or at least 24 hour ECG study
- Accounts for about 1% of sudden cardiac deaths in athletes
- High risk accessory pathways found on EP studies should likely be ablated
Figure 17  ECG demonstrating the classic findings of Wolf-Parkinson-White pattern with a short PR interval (<120 ms), delta wave (slurred QRS upstroke) and prolonged QRS (>120 ms).
Long QT interval

- Genetically mediated ventricular arrhythmia
- Hallmark is QT prolongation on resting ECG
- Syncope/seizures/SCA/D from Torsades de pointes
- Caused by prolonged depolarization due to loss of function mutations in genes coding voltage gated potassium channels that govern phase 3 repolarization
- Estimated to affect 1/2000 but likely grossly underestimated
Long QT

- 25-40% of sudden death under 40 unexplained at autopsy
  - Often considered to be an ion channelopathy
  - Likely responsible for 25-30% of these unexplained sudden deaths
Calculating the QTC

- Use Bazett’s formula $\text{QTC} = \frac{\text{QT}}{\text{square root of the RR}}$
- Make sure HR is 60-100 bpm to not over or under estimate
- If Sinus arrhythmia present, use an average QT and RR, but do not take the maximum QT and shortest RR
- Leads II or V5 should be used to really see the end of the T wave
Calculating the QTC

- Do not use a low amplitude U wave to calculate.
  - “Teach the Tangent” or “Avoid the tail”

- The morphology of the T wave can also predict LQTS
  - A notched T wave in the late precordial leads where the amplitude of the second portion is greater than the first portion may represent LQT-2 even if no over QT prolongation
Figure 2. ECG from a 27 year old male with long QT syndrome (QTc = 520 ms). Heart rate is 52 bpm. Bazett’s formula: QTc = QT/√RR. Note the RR interval is measured in seconds.
LQTS

- Cut-offs
- The current consensus guidelines conclude
  - >470ms in males
  - >480ms in females
Figure 4. This figure illustrates the “Teach-the-Tangent” or “Avoid-the-Tail” method for manual measurement of the QT interval. A straight line is drawn on the downslope of the T wave to the point of intersection with the isoelectric line. The U wave is not included in the measurement.
Short QT syndrome

- Very Rare
- Cut off is <320ms
- Usually only investigated in presence of syncope, atrial fibrillation, ventricular arrhythmias, or relevant family history
Figure 3. ECG from a patient with short QT syndrome. QT interval is 240 ms. Note also the tall, peaked T waves across the precordial leads characteristic of short QT syndrome.
LQTS Evaluation

- QTC < 500ms unlikely to represent true LQTS
- Will need help of cardiac rhythm Specialist
- Will use symptoms, genetic testing, family history, repeat ECG, exercise ECG testing, thorough review of patient medication list, and evaluation of electrolytes
Brugada Type 1 pattern

- 3 types but Type 1 is now considered diagnostic
- Loss of function mutation in sodium channel gene SCN5A in 20%
- 4% of sudden death in general population and 5-20% of cases of SCD with structurally normal heart at autopsy
- Characteristic high take off ST segment in right precordial leads – a coved rSr’; ST segment elevation of > 2mm, inversion of the t wave V1-3
Brugada

- Type 2 & 3 have a saddleback appearance
  - J point elevation $\geq 2\text{mm}$
  - ST elevation $> 1\text{mm}$ type 2
  - ST elevation $< 1\text{mm}$ type 3

- Type 2&3 do no require further testing at this time

- Ventricular Fibrillation is generally rhythm that leads to sudden death and occurs more often at rest than with exercise
Figure 8. Brugada pattern ECGs. Type 1 Brugada pattern ECG is defined as a high-takeoff and downsloping ST segment elevation $\geq 2$ mm followed by a negative T wave in at least two contiguous leads (V1-V3). Type 2 and 3 Brugada pattern ECGs have a “saddleback” appearance with J-point elevation $\geq 2$mm, ST segment elevation $>1$ mm in type 2 and $\leq 1$ mm in type 3, and either a positive or biphasic T wave.
Brugada Evaluation

- Repeat ECG/check leads
- Repeat with leads V1 and V2 in 2\textsuperscript{nd} and 3\textsuperscript{rd} IC space
  - High lead placement
- Check for hyperkalemia, sodium blocking medications, and fever
- Refer to EP specialist
Figure 9. Brugada type 1 ECG (left) should be distinguished from early repolarisation with “convex” ST segment elevation in a trained athlete (right). Vertical lines mark the J point (STJ) and the point 80 ms after the J point (ST80), where the amplitudes of the ST segment elevation are calculated. The “downsloping” ST segment elevation in Brugada pattern is characterised by a STJ/ST80 ratio >1. Early repolarisation patterns in an athlete show an initial “upsloping” ST segment elevation with STJ/ST80 ratio <1.
Profound Sinus Bradycardia

- Less than 30bpm or pause > 3 seconds warrants further evaluation

- Assess heart rate response to exercise
  - If goes up and asymptomatic – stop
  - If symptomatic or no response further eval for sinus node disease
Profound 1\textsuperscript{st} degree heart block

- A PR interval > 400ms requires further eval
- Should shorten with mild amount of aerobic activity
- If symptomatic or family h/o SCA/D, then further investigation – echo, exercise ECG, event monitor
High Grade AV block

- Mobitz type 2 and Grade 3 (complete) heart block are abnormal and need further evaluation
- Exercise ECG, Echo, cardiac MRI and referral to Cardiology
Multiple PVCs

- 2 or more PVCs on a 10 second ECG is a potential marker for heart disease and requires further evaluation
- Evaluate to see if >2,000 PVCs per 24 hours. 30% chance of heart disease
- Exercise ECG, echo, cardiac MRI, and EP study might be needed
CPVT

- Catecholaminergic Polymorphic Ventricular Tachycardia
  - Inherited; ventricular ectopy triggered by exercise or emotional stress; syncope, v. tach/fib.cardiac arrest
  - Often found around age 7-9, but as late as 4th decade
  - RYR2 encoded cardiac Ryanodine receptor/calcium release channel
  - Found in structurally normal heart
  - Consider eval in those with syncope with extreme emotion or maximal exertion
CPVT

- Estimated 1/10,000
- 4-10% autopsy negative SCD
- Resting EKG IS NORMAL! ECHO IS NORMAL!
- If suspected with need exercise treadmill with progressive graded exertion
Figure 7. 15 year old boy undergoing exercise stress test for evaluation of CPVT. Polymorphic ventricular ectopy is evident late in stage 3 of the exercise stress test. Heart rate 148 bpm.
Atrial Tachyarrhythmias

- SVT = AVNRT, AVRT, ST, atrial tach., atrial fibrillation and flutter
- Rarely seen on resting ECG
- Rarely life threatening and lead to symptoms
  - Certainly if found work up
- Sinus Tachycardia > 120 bpm – retest after period of rest and look for etiology
- Rarely seen on resting ECG – athletes are usually symptomatic
Paroxysmal supraventricular tachycardia (SVT) refers to narrow complex tachycardias including atrioventricular nodal reentrant tachycardia (AVNRT), atrial tachycardia, and other rare atrioventricular reciprocating tachycardias. Atrial fibrillation and flutter are types of SVT but do not fall into the paroxysmal classification. Here, there is a narrow complex tachycardia at 240 bpm.
Ventricular Arrhythmias

- Couplets, triplets, nonsustained VT
- Always require evaluation
- Cardiomyopathies, ion channelopathies, myocarditis, ischemic heart disease, or sarcoidosis
- Evaluation will include family history, echo, ambulatory ECG, cardiac MRI, and exercise stress
### Normal ECG Findings
- Increased QRS voltage for LVH or RVH
- Incomplete RBBB
- Early repolarization/ST segment elevation
- ST elevation followed by T wave inversion V1-V4 in black athletes
- T wave inversion V1-V3 ≤ age 16 years old
- Sinus bradycardia or arrhythmia
- Ectopic atrial or junctional rhythm
- 1° AV block
- Mobitz Type I 2° AV block

### Abnormal ECG Findings
- T wave inversion
- ST segment depression
- Pathologic Q waves
- Complete LBBB
- QRS ≥ 140 ms duration
- Epsilon wave
- Ventricular pre-excitation
- Prolonged QT interval
- Brugada Type 1 pattern
- Profound sinus bradycardia < 30 bpm
- PR interval ≥ 400 ms
- Mobitz Type II 2° AV block
- 3° AV block
- ≥ 2 PVCs
- Atrial tachyarrhythmias
- Ventricular arrhythmias

### Borderline ECG Findings
- Left axis deviation
- Left atrial enlargement
- Right axis deviation
- Right atrial enlargement
- Complete RBBB

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**Figure 1**  International consensus standards for ECG interpretation in athletes. AV, atrioventricular; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; PVC, premature ventricular contraction; RBBB, right bundle branch block; RVH, right ventricular hypertrophy; SCD, sudden cardiac death.