Irregular Heart Beats/Palpitations

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CASE 1

- A 12 year old presents with a fast heart rate. He is otherwise asymptomatic. A wide complex rhythm is noted at a rate of 260 bpm. His blood pressure is 92/50. The next best course of action would be:
A. Adenosine  
B. Sotalol  
C. Amiodarone  
D. Verapamil  
E. DC cardioversion

**SVT**

- In a hemodynamically stable patient, a supraventricular tachycardia with aberrant conduction is the most likely rhythm.  
- The best treatment for this patient would be administration of adenosine to terminate the tachycardia.
CASE 2

An 8 year old presents with a fast heart rate and difficulty breathing. Blood pressure is stable. An ECG is obtained and shown below. There is no change in the heart rate with adenosine. Your next course of action would be:

A. DC cardioversion
B. Sedation and atrial overdrive pacing
C. Digoxin
D. Beta blockers
E. Verapamil
Fascicular Ventricular Tachycardia

- Originating from the left ventricle.
- Adenosine is usually not effective in terminating the tachycardia.
- Calcium channel blockers are very effective and hence this tachycardia is also referred to as verapamil sensitive ventricular tachycardia.
- This is usually amenable to ablation.

CASE 3:
A 12 year old boy is undergoing a stress test for frequent ectopy at baseline. The echocardiogram at rest was normal. You notice the following rhythm at stage 5 of the standard Bruce protocol. The diagnosis is most consistent with:
Catecholaminergic ventricular tachycardia (CPVT).

- Polymorphic ventricular tachycardia with exercise
- Brugada syndrome and long QT type 3
  - polymorphic ventricular tachycardia generally at rest or during sleep.
- Myocarditis: decrease in cardiac function.
CPVT

- CPVT cannot be diagnosed on the basis of a resting ECG.1,2
- Exercise stress testing is an important part of a CPVT workup.
  - However, in as many as 20% of CPVT patients, formal exercise stress testing will not produce ventricular ectopy.1
- During exercise stress testing, bidirectional VT with a beat-to-beat 180-degree rotation of the QRS complex is often observed.1


Mutations in the Cardiac Ryanodine Receptor Gene (RYR2) Are the Major Cause of CPVT

- 60-65% of CPVT is caused by mutations of the cardiac ryanodine receptor gene, which encodes a sarcoplasmic calcium ion channel.1,2

It Is Important to Differentiate Between CPVT and LQTS

- CPVT is an LQTS mimicker.¹
- As many as 30% of CPVT patients have been misdiagnosed as having “Long QT with normal QTc.”²,³
- Differentiating CPVT from LQTS is important for:
  - Developing a comprehensive treatment plan
  - Family-specific testing


Beta-blockers Do Not Provide Reliable Protection Against Cardiac Arrhythmias Related to CPVT

- “However, in light of incomplete protection afforded by beta-blockers in CPVT, its distinction from long-QT is clinically relevant.”¹
- Nearly 50% of CPVT patients taking a beta-blocker continue experiencing cardiac arrhythmias and may require an ICD.¹

Brugada Syndrome

- The ECG of patients with BrS is characterized by
  - Coved- or saddleback-shaped ST-segment elevation in leads V1 through V3
  - complete or incomplete right bundle-branch block
  - T-wave inversion

- These ECG abnormalities may not be evident until unmasked by infusion of a sodium channel blocker (flecainide or procainamide).

References:

BrS Typically Presents in Men 30-40 Years of Age

- The most common presentation of BrS is a man 30-40 years of age with malignant arrhythmias and a history of syncope.
  - Men are 8-10 times more likely than women to have the BrS phenotype.
  - There is no difference between men and women in the prevalence of gene mutations associated with BrS.
- Men with an abnormal ECG and inducible ventricular arrhythmias have a poor prognosis.
- If untreated, men with BrS have a 45% chance of having a cardiac event over the course of their life.

References:
CASE 4

- A 14 year old patient with a history of WPW and SVT presents with this ECG. He is alert and hemodynamically stable. The MOST appropriate therapy is:

A. Adenosine  
B. Lidocaine  
C. Observation  
D. DC cardioversion  
E. IV calcium channel blocker
WPW

- Atrial fibrillation with rapid conduction over the accessory pathway results in a rapid ventricular response.
- At risk for ventricular fibrillation and compromised circulation.
- Adenosine can also induce atrial fibrillation.
- DC Cardioversion should be readily available.

CASE 5

- A 12 year old boy comes to you with palpitations with exercise. He also complains of occasional chest pain with exercise. You obtain an ECG which is shown below. The next most obvious recommendation would be to:
• a. Perform an exercise stress test.
• b. Clear him for sports without any restrictions.
• c. Perform an echocardiogram.
• d. Perform a CXR.
• e. Perform a 30 day event monitor.

Left ventricular hypertrophy with a strain pattern.

• At risk for ventricular tachycardia and sudden cardiac death with exercise
• Additional risk factors
  – family history of premature sudden death
  – extreme LV hypertrophy (> 30 mm)
  – NS ventricular tachycardia on Holter
  – unexplained (not neurally mediated) syncope
The Clinical Presentation of HCM

- HCM is a progressive disease associated with varying degrees of LVH with no apparent cause.\(^1\)
  - Thickening typically develops during late childhood and throughout adolescence.\(^3\)

Obstructive and Non-obstructive HCM

LVH is usually maximal in the interventricular septum, but other localized variants have been documented.\(^1\)
The Clinical Diagnosis of HCM Relies Primarily on Echocardiogram

**Echocardiogram can help determine:**

- Location and extent of hypertrophy
- Systolic and diastolic function
- The presence and degree of systolic anterior motion
- The severity of subaortic and/or midventricular obstruction
- Direction and degree of mitral regurgitation
- The presence or absence of additional mitral valve abnormalities
- Left atrial size

**QUESTIONS**
CASE 6
A 21 year old patient with classic Fontan presents with palpitations and fast heart rates for 3 days. ECG shows an unvarying heart rate of 140 bpm. He usually has a heart rate between 60 to 90 bpm. His blood pressure is stable. The following ECG is obtained. The next most appropriate intervention would be:

- a. Echocardiogram
- b. Digoxin
- c. Adenosine
- d. Sotalol
- e. Amiodarone