**Case #1**

A 68 year old male presents to the ED with two hours of continuous, retro-sternal chest pressure that radiates into both upper arms. He is a long-term smoker and an insulin-dependent diabetic. On presentation he has a BP of 119/70, a heart rate of 72, elevated jugular venous waves, clear lung fields and no murmurs. The admitting ECG is pictured...

In the ED, the patient is immediately given ASA and sublingual NTG upon presentation, and cardiology is called. Within minutes, the patient's blood pressure drops to 75/55. The most likely explanation for this patient's acute drop in blood pressure is:

A. Severe, acute decrease in LV systolic function due to acute MI
B. Myocardial rupture with tamponade
C. Right ventricular infarction
D. Papillary muscle rupture with severe, acute mitral regurgitation

***PEARL: Inferior Infarction (+/- ST elevation in V1) + Elevated neck veins + Hypotension (especially post-NTG/morphine) = RV Infarction***

The diagnosis of RV infarction can be confirmed with right-sided EKG leads...
Right-sided Precordial EKG leads

Keys to management of RV Infarction:
1) Avoid NTG/morphine
2) IV fluid challenge to maintain RV preload (CVP 10 – 15 mmHg)
3) Emergent Revascularization
4) Inotropic support (dobutamine)

The patient undergoes emergent revascularization and has successful placement of a drug eluting stent to the proximal RCA. His blood pressure improves and he looks well clinically. He is transferred to the CCU for close monitoring. Two hours later, his nurse calls to report concerns about the patient’s rhythm. You arrive at the bedside and find the following telemetry strip:

Which of the following statements about this rhythm is TRUE:
A. The patient will need a permanent pacemaker
B. This represents a failure of revascularization, and the patient should return to the cath lab for evaluation
C. The level of block is at the AV node
D. This rhythm is found in nearly 80% of all inferior STEMI patients

***PEARL: Heart block in the setting of inferior MI occurs at the level of the AV node, is usually benign, and rarely progresses. It rarely requires permanent pacemaker placement (usually resolves).

Alternatively, heart block occurring in the setting of an ANTERIOR MI is associated with a very poor prognosis; if the patient survives, a permanent pacemaker is almost always required...

Heart Block in Inferior MI:
1) Vagal activation by posterior C fibers
2) AV node ischemia
3) Usually resolves

Heart Block in Anterior MI:
1) Extensive infarction leading to necrosis of the conduction system below the AV node
2) Associated with very poor prognosis
3) Those who survive will likely need pacemaker
Case #3

A 66 year old male is admitted to the hospital by the ED for new-onset congestive heart failure. The patient has a long-standing history of HTN, but no other major medical problems. He presented with worsening lower extremity edema, orthopnea, and exertional dyspnea after walking 20 feet, which is new for him. The patient receives IV diuretics with improvement in his symptoms over 2-3 days. As part of his evaluation, he has a nuclear stress test that is negative for ischemia. He has an Echocardiogram that reveals an EF of 60%, mild LVH, and Grade II Diastolic dysfunction (pseudonormal).

Which of the following should the patient receive upon discharge to improve his long-term survival:

A. ACE inhibitor
B. Beta-blocker
C. Non-dihydropyridine Calcium channel blocker (diltiazem)
D. Dihydropyridine Calcium channel blocker (amlodipine)
E. None of the above

***PEARL: To date, there is no therapy that improves survival in patients with HFPEF (Heart Failure with Preserved EF).

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Case #4

A 54 year old female presents to the ED with complaints of acute onset substernal chest pain and dyspnea. Her symptoms began soon after learning of the death of her father. Her workup revealed a troponin of 3.2, and her EKG is shown below:

The patient is taken to the cath lab, where she was found to have normal coronary arteries...a ventriculogram is performed and reveals the following:

A) Brugada Syndrome
B) Tako-Tsubo Syndrome
C) Williams Syndrome
D) Cadaver Syndrome

The name of this syndrome is:

D) Her prognosis is generally favorable, and most patients have full recovery of LV function
Case #4

***PEARL: Classic Tako-Tsubo patient:

1) Middle-aged female with an acute life stressor
2) Presenting with chest pain + elevated cardiac enzymes
3) EKG can vary at presentation (ST elev, ST dep, TWI, etc)
4) No obstructive disease at cath
5) ECHO/LV gram shows classic findings (apical, mid, basal HK)

Treatment is generally supportive care. Some may be sick enough to require inotropes, balloon pump, and mechanical ventilation. Most will recover, even the extremely sick. While beta-blockers and ACE inhibitors are routinely used, unclear if they help or not.

Case #5

A 71 year old male presents with complaints of progressive exertional dyspnea, lower extremity edema, and abdominal bloating. His medical history includes CKD stage III and multiple myeloma. He has no history of heart disease. On exam, you note 4+ pitting edema to the thighs, JVD to the angle of the mandible at 90 degrees, and abdominal ascites. He has a regular rhythm and no murmurs. The lung fields are clear.

Workup reveals the following EKG and ECHO...

Based on the available information, which of the following is the most likely cause of the patient's presentation:

A. Constrictive Pericarditis
B. Cardiac Tamponade
C. Amyloid cardiomyopathy
D. Hypertrophic Obstructive Cardiomyopathy (HOCM)

***PEARL: Marked LVH on ECHO + Low voltage on EKG = Infiltrative Cardiomyopathy (e.g. amyloid)

OTHER PEARLS ABOUT AMYLOIDOSIS:

*Amyloid* is a generic term that can apply to any protein that folds into beta-pleated sheets and deposits in organs...leading to organ dysfunction

Amyloid proteins: AL (light chain), AA (serum amyloid A), ATTR (Transthyretin – senile or familial variants), Beta2-microglobulin

AL and ATTR most commonly affect the heart (AA usually does not have significant CV effects)

AL (light chain) can occur in the setting of multiple myeloma, but more commonly is a result of a small clonal population of plasma cells that hypersecrete the protein

AL has a generally poor prognosis (6 months) if untreated

Traditional therapies for CHF are generally ineffective; diuretics may help with edema/symptoms

Therapy should be directed at preventing progression of disease (esp. AL)

Transplantation (Heart + BMT) may be an option for some
A 16 year old male presents to your office for a routine physical exam prior to starting competitive sports. He has no significant past medical history and has an active lifestyle that involves playing basketball and soccer. His father died suddenly at the age of 38. On exam, you note a grade 3/6 crescendo-decrescendo murmur along the left sternal border. When the patient assumes a squatting position, the murmur intensity is reduced. The remainder of the physical exam is normal, including his blood pressure.

Given this abnormal exam, you order an EKG.

Considering the patient’s history, exam findings, and EKG, which of the following is the most likely diagnosis:

A. Mitral Valve Prolapse (MVP)
B. Hypertrophic Obstructive Cardiomyopathy (HOCM)
C. Hypertensive Heart Disease
D. Premature Coronary Disease

***PEARL: Family history of unexplained sudden death + crescendo/decrescendo murmur + LVH = HOCM

***PEARL: The murmur of HOCM is reduced by squatting and increased by standing or valsava. Similarly, the murmur of MVP is shortened by squatting and prolonged by standing or valsava (think in terms of preload... Squatting increases preload and therefore increases the LV volume...Standing and valsava have the opposite effect)

An ECHO is performed which reveals asymmetric septal hypertrophy (ASH) with a septal thickness of 21 mm, systolic anterior motion (SAM) of the mitral valve anterior leaflet, and an outflow tract gradient of 40 mmHg.

Considering the diagnosis of HOCM, which of the following is appropriate counseling for the patient regarding participation in competitive sports:

A) He is free to compete in intensive athletics, including competitive basketball and soccer
B) He should refrain from all activity, as he will be at high risk of sudden death
C) It is safe to participate in low-intensity sports (e.g. golf, bowling)
D) If his gradient normalizes with medical therapy, he can then participate in intensive athletics, including competitive basketball and soccer

An ECHO is performed which reveals asymmetric septal hypertrophy (ASH) with a septal thickness of 21 mm, systolic anterior motion (SAM) of the mitral valve anterior leaflet, and an outflow tract gradient of 40 mmHg.
A 34 year old male has just returned to Atlanta after completing a 4-month excursion along the Appalachian Trail. He is out to dinner with his wife, when suddenly he feels extremely “woozy” and nearly passes out. EMS is called and brings him to Emory Hospital for evaluation. By history, he has been well with the exception of generalized fatigue and malaise for the last 3 weeks. In the ER an EKG is obtained which shows the following:

**INITIAL EKG IN ER**

EKG 24 hours later:

Which of the following skin lesions is commonly associated with the patient’s underlying disease process:

- **ERYTHEMA MIGRANS**
- **A**
- **B**
- **C**
- **D**

***PEARL: RASH + AV CONDUCTION DISEASE = LYME DISEASE***

**OTHER PEARLS, FACTS, and NUMBERS**

- Wide complex tachycardia + ANY history of heart disease = guess V-TACH
- Tachyarrhythmia = clinically unstable = SHOCK!
- Tachyarrhythmia and pharmacological therapy = amiodarone
- Brady = symptoms = pacemaker (unless an obvious, reversible etiology)
- Goal door-to-balloon time for STEMI = 90 minutes
- Goal door-to-needle (lytics) time for STEMI = 30 minutes
- LVEF < 35%
- Positional, pleuritic chest pain + diffuse ST elevation = pericarditis
- Acute, “tearing” chest pain into the back in a HTN patient = aortic dissection
- Treating Torsades de Pointes = shock, magnesium, induce tachycardia (isuprel or pacing)
- QT prolonging drugs = antipsychotics, anti-emetics, antibiotics (macrolides, quinolones), anti-arrhythmics
OTHER PEARLS, FACTS, and NUMBERS

SVT and HR 150 = atrial flutter
SVT and HR 180 = AVNRT (give adenosine)

Mechanical complications of STEMI (VSD, Pap rupture, Free wall rupture) = 2-5 days post-MI

Endocarditis prophylaxis = prosthetic valve, prior endocarditis, cyanotic congenital heart disease, valvular disease of a transplanted heart

Cardiac tamponade = elevated neck veins, distant heart sounds, hypotension

Medications ok for use in Pregnancy with CHF = loop diuretics, hydralazine, digoxin...BB probably safe (but can cause growth retardation)

Peripartum cardiomyopathy = 1/3 get better, 1/3 stay the same, 1/3 get worse

Chest pain and pregnancy = consider coronary dissection!

THE END!