EKG's and ST Changes That Can Kill You

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Disclosure

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Objectives

- Recognize some different types of EKG and ST changes that can kill you
- Recognize what are the atypical EKG presentations in patients with acute myocardial infarction
- Recognize conditions that have EKG's changes that mimics acute myocardial infarction



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Typical ST morphology

• AMI

- Convex or straight ST elevation ("frowny face")
- Benign early repolarization
 - Concave ST elevation ("smiley face")
- Pericarditis
 - Concave ST elevation ("smiley face") and often associated with PR depression

• BBB

- Concave ST elevation ("smiley face") with discordant QRS complex, usually < 5 mm elevation
- LV aneurysm
 - Usually of V1-V2 and is unchanged if compared to prior EKG's. Usually has evidence of prior anterior infarction (poor R wave progression and Q waves)





Typical ST morphology

- AMI : convex or straight ST elevation ("frowny face")
- Benign early repolarization: concave ST elevation ("smiley face")
- Pericarditis: concave ST elevation ("smiley face") and often associated with PR depression
- BBB: concave ST elevation ("smiley face") with discordant QRS complex, usually < 5 mm elevation
- LV aneurysm --> Usually of V1-V2 and is unchanged if compared to prior EKG's. Usually has evidence of prior anterior infarction (poor R wave progression and Q waves)





EKG's and ST Changes That Can Kill You

- Early repolarization (well, not really!)
- LVH
- AMI
- RV AMI
- Inverted T-wave in avL
- T wave in v1
- Pericarditis that is not

- ST elevation in avR HCM
- Wellen's
- de Winter
- Torsades
- LBBB
- Pericarditis
- Brugadas

- LV aneurysm
- New RAD
- Prolonged QTc
- Pericardial Tamponade
- PE
- Slow Vtaq

- AIVR
- WPW
- Hyperkalemia
- Hypothermia
- CNS disorders
- Takotsubo
 Syndrome
- Spiked-Helmet Sign













Early Repolarization







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Early Repolarization









Early repolarization





LVH







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LVH Criteria



LVH by voltage criteria in chest leads



* > 35mm is significant







LVH Criteria

LEFT VENTRICULAR HYPERTROPHY

Large S wave in leads V1 and V2, large R wave in V5 and V6













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What is the first EKG sign for a STEMI?

- Hyperacute T waves
- ST segment elevation
- T wave inversion
- Significant Q waves





Cardiovascular Anatomy

- Coronary Circulation
 - Collateral Circulation







- Ischemia
- Injury
- Infarction
 - Subendocardial
 - Transmural







Evolution of Acute Myocardial Infarction





- Evolution of Acute Myocardial Infarction
 - Subendocardial Infarction



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Subendocardial MI

Associated with variable ST and T wave changes





- Evolution of Acute Myocardial Infarction
 - Transmural Infarction

Transmural Infarction

- Before coronary occlusion -

Heart muscle normal

Normal ECG

Subendocardial injury and myocardial ischemia. No cell death (infarction) yet

Onset and first several hours —

or nearly normal —

R wave normal

ST segment elevated

T wave

peaked

First day

Ischemia and injury extend to epicardial surface. Subendocardial muscle dying in area of most severe injury

R wave amplitude diminishing



ST elevation more marked





Evolution of Acute Myocardial Infarction



Transmural MI

- Anterior MI
 - LAD
 - STE $V_1 V_6$
 - Inferior reciprocal changes
 - Anteroseptal V₁-V₄
- Anterolateral
 - LAD or anterior trunk
 - STE V_3 - V_6 , I, aVL
 - Inferior reciprocal changes
- Lateral
 - Left circumflex or diagonal branch from LAD
 - STE $V_5 V_6$, I, aVL
 - Inferior reciprocal changes





Transmural MI

- Inferior
 - RCA (90%)
 - STE II, III, aVF
 - Reciprocal changes most common in lead aVL, may be I
 - Always consider possibility of concurrent posterior or RV involvement
- Posterior
 - Large R wave with ST depression in $V_1 V_3$
 - Mirror image of septal MI
 - Usually associated with inferior MI, less commonly with lateral MI
 - RCA or Left circumflex











What is an ischemic change?





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What is an injury change?





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Localization of Acute Myocardial Infarction

Table 2-5	LOCATION OF MYOCARDIAL ISCHEMIA/INFARCTION
Location	Leads
Anterior	I, V_2 , V_3 , and V_4
Anteriolateral	I, aVL, V_5 , and V_6
Lateral	V_5 and V_6
High lateral	I and aVL (often with V ₅ , V ₆)
Inferior	II, III, and aVF
Inferolateral	II, III, aVF, and V ₆
True posterior	Reciprocal changes in V_1 and V_2



Septal = $V_1 - V_2$ Anteroseptal = $V_1 - V_4$



Anterior



Anterior infarct

Occlusion of proximal left anterior descending coronary artery



Significant Q waves and T wave inversions in leads I, V_2, V_3 and V_4 $\,$





Anterolateral



Anterolateral infarct

Occlusion of left circumflex coronary artery, marginal branch of left circumflex artery, or diagonal branch of left anterior descending artery



Significant Q waves and T wave inversions in leads I, aVL, V_5 and V_6





- Anterolateral
- Lateral
- High Lateral





Significant Q waves and T wave inversions in leads I, aVL, V_5 and V_6




Disease Findings

- Inferior
- Inferolateral



Occlusion of right coronary artery



Significant Q waves and T wave inversions in leads II, III and aVF. With lateral damage, changes also may be seen in leads V_5 and V_6





Disease Findings

True Posterior



True posterior infarct



circumflex artery

descending or distal right coronary arteries Since no ECG lead reflects posterior electrical forces, changes are reciprocal of those in anterior leads. Lead V1 shows unusually large R wave (reciprocal of posterior Q wave) and upright T wave (reciprocal of posterior T wave inversion)





Inferior MI







Inferolateral MI







Anterior MI







Anterorinferior MI







Anterolateral MI







Septal MI







Inferoseptal







Posterior MI



ST-depression \geq 0.5 mm in V₁-V₃ after a tall R wave





Posterior MI



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• You give ASA and NTG when the BP \downarrow ...

- What happened?
- Remember the first EKG?





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Right Ventricle MI

- BP will drop when giving NTG
- JVD +++; Lungs: CTA
- Extensive Inferior AMI
 - RCA occlusion
- Do right side EKG
- Treat hypotension with IVF's, Dobutamine





Right Side EKG







RV AMI Complications

- Cardiogenic shock
- High-degree AV Block
- Afib
- Ruptured of interventricular septum
- Tricuspid regurgitation
- Opening of patent foramen ovale
 - Causing right to left shunt
 - Hypoxia









Other EKG Changes Suggesting AMI



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- JOF CENTRA
- 46 y/o male with chest pain





Call STEMI



ECG #2





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• An inverted T-wave in aVL is often associated to inferior MI



- Exceptions
 - LVH
 - LBBB





• 63 y/o man with right arm and hand pain





Documented as artifact



• Why I called STEMI?

Upright T waves in V1 not normal, early STEMI







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ECG #2







- T wave in V1 is normally flat or inverted
 - Occasionally is upright
 - In the elderly, usually not good
- Associated to left circumflex or RCA disease
- Also,
 - LVH
 - LBBB
 - Brugada
 - Pulmonary emboli





• 28 y/o male with chest pain and SOB



- Patient was discharged with the diagnosis of early repolarization vs. pericarditis
 - Will follow up with cardiology





 Patient returned by ambulance c/o increased CP after collapsing





I called STEMI



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Pericarditis does not have reciprocal changes

Depressed PR







• 53 y/o female with CP and SOB





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The forgotten lead

- LMA occlusion
- If ST ↑ in aVR in addition
 - Consider LMA and/or LAD occlusions
- Treatment
 - PCI
 - CABG
 - Thrombolytic not work





"Widow Maker"

- Lesion in the main LCA or proximal LAD can have severe consequences, as suggested by the common nickname given to this vessel: "widow maker"
- The LAD supplies the anterior wall of the heart, including both ventricles, as well as the septum
- An occlusion in this vessel can result in serious ventricular dysfunction, thus placing the patient at serious risk for congestive heart failure (CHF) and death





52 y/o male with CP



Wellen syndrome




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Wellen's Syndrome

• Criteria

Criteria of Wellens' syndrome

Prior history of chest pain

Chest pain with normal ECG

Normal or minimally elevated cardiac enzymes

No pathologic precordial Q waves or loss of R waves

ST segment in V2 and V3 that is isoelectric or minimally elevated (1mm), concave or straight

Symmetric and deep T-wave inversion or biphasic T-waves in V2 to V5 or V6 in pain free periods

Tight proximal LAD stenosis

 Refers to these specific electrocardiographic (ECG) abnormalities in the precordial T-wave segment, which are associated with critical stenosis of the proximal left anterior descending (LAD) coronary artery





Wellens' syndrome

- Pattern of deeply inverted or biphasic T waves in V2-3
 - Highly specific for a critical stenosis of the left anterior descending artery (LAD).
- Patients may be pain free by the time the ECG is taken and have normally or minimally elevated cardiac enzymes;
 - However, they are at extremely high risk for extensive anterior wall MI within the next few days to weeks.





Wellens' syndrome

- Due to the critical LAD stenosis,
 - These patients usually require invasive therapy
 - Poorly with medical management
 - May suffer MI or cardiac arrest if inappropriately stress tested





- The following sequence of events is thought to occur in patients with Wellens' syndrome:
 - 1. A sudden occlusion of the LAD,
 - a. Causing a transient anterior STEMI.
 - b. The patient has chest pain & diaphoresis.
 - c. This stage may not be successfully captured on an ECG recording.





- The following sequence of events is thought to occur in patients with Wellens' syndrome:
 - 2. Re-perfusion of the LAD (e.g. due to spontaneous clot lysis or prehospital aspirin).
 - a. The chest pain resolves.
 - b. ST elevation improves and T waves become biphasic or inverted.
 - c. The T wave morphology is identical to patients who reperfuse after a successful PCI.





- The following sequence of events is thought to occur in patients with Wellens' syndrome:
 - 3. If the artery remains open, the T waves evolve over time from biphasic to deeply inverted.
 - 4. The coronary perfusion is unstable, however, and the LAD can re-occlude at any time.
 - a. If this happens, the first sign on the ECG is an apparent normalization of the T waves — so-called "pseudo-normalization".
 - b. The T waves switch from biphasic/inverted to upright and prominent.
 - c. This is a sign of hyperacute STEMI and is usually accompanied by recurrence of chest pain, although the ECG changes can precede the symptoms.





- The following sequence of events is thought to occur in patients with Wellens' syndrome:
 - 5. If the artery remains occluded, the patient now develops an evolving anterior STEMI.
 - 6. Alternatively, a "stuttering" pattern may develop, with intermittent reperfusion and re-occlusion.
 - a. This would manifest as alternating ECGs demonstrating Wellens' and pseudonormalization/STEMI patterns.





- This sequence of events is not limited to the anterior leads similar changes may be seen in the inferior or lateral leads, e.g. with RCA or circumflex occlusion.
- Also, the inciting event does not necessarily have to be thrombus formation:
 - Wellens' syndrome may also occur in normal coronary arteries following an episode of vasospasm, as in this case of cocaine-induced vasospasm.
- However, it is safer to assume the worst (i.e. critical LAD stenosis) and work the patient up for an angiogram.









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New ECG Sign of Proximal LAD Occlusion

- de Winter STEMI
 - 1-3 mm upsloping ST-segment depression at the J point in the leads V₁ to V₆ that continued into tall, positive symmetrical T waves in the precordial leads
 - Absence of ST elevation in the precordial leads
 - ST segment elevation (0.5mm-1mm) in aVR
 - "Normal" STEMI morphology may precede or follow the deWinter pattern
 - Found to have proximal LAD occlusion



"de Winter" ST/T-wave complexes



The de Winter ECG pattern

- An anterior STEMI equivalent that presents without obvious ST segment elevation.
- Key diagnostic features include ST depression and peaked T waves in the precordial leads.
- Seen in ~2% of acute LAD occlusions and is under-recognised by clinicians.
- Unfamiliarity with this high-risk ECG pattern may lead to under-treatment (e.g. failure of cath lab activation), with attendant negative effects on morbidity and mortality.





- Patient started complaining of Chest pain when suddenly.....
- She collapsed.....







- What is going?
 - She is having a seizure?
 - Someone is tickling her?
 - She is dancing?
 - Torsade's de Pointe
- What is the treatment?
 - Anticonvulsant?
 - Stop tickling her?
 - Defibrillate
 - Add MgSO4





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STEMI Torsade's de Pointe







- Chest pain
- Irradiates to the left side
- SOB
- DOE
- PMHx: none
- Meds: none
- Etc.









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Old EKG





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Sgarbossa Criteria

- ST-segment elevation >1 mm in lead with concordant QRS complex = 5 points
- ST-segment depression >1 mm in leads v1, v2, or v3 = 3 points
- ST-segment elevation >5 mm in lead with discordant QRS complex = 2 points
- (3 points or more is positive ~70%)







- Chest pain
- Irradiates to the left side
- SOB
- DOE
- PMHx: none
- Meds: none
- Etc.





• VS

• HR 82; BP 148/90; RR 18; T 99; O2sat 98%

• **PE**

- HEENT: WNL
- Chest: CTA; RRR
- Abd: WNL
- Ext: no edema
- Neuro: WNL





EKG



- What is it?
- STEMI?







<u>.</u>

- Diffused ST elevations with
- No reciprocal changes
- PR depression





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• 28 y/o woman with syncope







• Patient is discharged

mmmmmmmm

 Patient returned to ED by EMS pulseless with this rhythm



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Brugada







ECG changes in the Brugada syndrome



500ms



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- More common cause of sudden death (4-5%)
 - Up to 20% in individuals without structural heart disease
 - Most common in young males <40 y/o



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- 19 y/o college student in training
- Dehydrated, sweating
- Not drinking a lot of fluids
- Near-syncope, palpitations






- Patient was discharged after fluids and electrolytes were replaced
- Patient returned to ED by EMS pulseless with the following rhythm





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HCM

- Genetics factors involved
- 0.02-2% of general population
- Hypertrophied but nondilated left ventricle
 - Thickening is usually asymmetric, involving the septum to a greater extent than the free ventricular wall
 - C-Xray with no CM
- Mortality 3.5% per year
- Symptoms often associated with exertion









Enlarged septum





HCM

Q Wave in Idiopathic Hypertrophic Cardiomyopathy

















I recognized this patient





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- Persistent left shoulder pain for 5 hours (no CP)
 - I saw this patient and admitted last week c/w mid spine pain
 - I thought that she had a dissection; ordered EKG (not done for 2 hours, nurses thought it was not needed);
 - showed STEMI in anterior leads
- LV Aneurysm







LV aneurysm

- Chronic ST elevation or T wave inversion suggests ventricular aneurysm
- Most common with anterior wall infarcts



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ID 279 – This 57 year old woman who had rheumatic fever at age 17 has been suffering from severe dyspnea and fatigue during the past year









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New RAD

- Most common
 - Lead misplaced
 - PE
 - Na channel blocking agent
- RVH
- LPFB
- Dextrocardiac
- Lateral MI
- Ventricular ectopy
- Hyperkalemia
- COPD
- Acute pulmonary disease with right heart strain (PE)
- Overdoses of sodium channel blocking drugs (TCA)





• 40 y/o female presenting with seizures







- Patient was observed for 4 hours and discharged home after no seizures
 - CT was WNL
 - Labs WNL except HCO3 = 17; attributed to "seizure"
 - Neurology was called and will follow up patient as OPD
- Patient returned to ED by EMS pulseless with the following rhythm





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- Patient tried to kill herself with TCA earlier
- Patient had syncope episode not a seizure
 - You need to ask the correct questions



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EKG's and ST Changes That Can Kill You

- Early repolarization (well, not really!)
- LVH
- AMI
- RV AMI
- Inverted T-wave in avL
- T wave in v1
- Pericarditis that is not

- ST elevation in avR HCM
- Wellen's
- de Winter
- Torsades
- LBBB
- Pericarditis
- Brugadas

- LV aneurysm WPW
- New RAD
- Prolonged QTc
- Pericardial Tamponade
- PE
- Slow Vtaq

- AIVR
- • •
- Hyperkalemia
- Hypothermia
- CNS disorders
- Takotsubo
 Syndrome
- Spiked-Helmet Sign











Prolonged QT-Interval

- Causes
 - Electrolytes abnormalities
 - Hypokalemia
 - Hypomagnesemia
 - Hypocalcemia
 - Na channel blocking medications
 - Antiarrhythmic IA
 - Anticholinergics
 - Cocaine
 - Many antipsychotics (TCA)
 - Some antibiotics (combinations of some macrolides, quinolones with antiarrhthymics)
 - AMI
 - CNS lesions
 - Hypothermia
 - Congenital
 - Deafness
 - Jervell and Lange-Nielsen syndrome
 - No deafness
 - Romano-Ward syndrome





- "corrected" QT-interval: QTc = $QT/\sqrt{(RR)}$
- Male?
 - > 450 msec
- Female and children?
 - > 460 msec
- Not good if > 500 msec
- Treatment
 - Correct
 - Discontinue
 - MgSO4







- Chest pain
- Irradiates to the left side
- SOB
- DOE
- PMHx: none
- Meds: none
- SocHx: smoker





• VS

• HR 112; BP 95/40; RR 18; T 99; O2sat 98%

• **PE**

- HEENT: WNL
- Chest: rales in bases; Taq; distant heart sounds; +JVD
- Abd: WNL
- Ext: no edema
- Neuro: WNL













CXray



















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Pericardial Tamponade







- Beck's triad
 - Hypotension
 - JVD
 - Muffled heart sounds



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- Echocardiogram
 - Large pericardial effusion
 - Diastolic collapse of the right ventricle and the right atrium
 - Swinging motion of the heart



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Cardiac Tamponade

- Management
 - Maintain airway.
 - Administer oxygen.
 - Establish IV access.
 - Consider medication administration:
 - Morphine sulfate
 - Nitrous oxide
 - Furosemide
 - Dopamine/dobutamine





Cardiac Tamponade

- Rapid Transport
 - Run for it!!!
- Pericardiocentesis
 - Pericardiocentesis is the definitive treatment.
 - Insertion of a cardiac needle and aspiration of fluid from the pericardium.
 - Procedure should be performed only if allowed by local protocol.
 - Procedure should be performed only by personnel adequately trained in the procedure.





• 45 y/o female with chest pain, SOB







12.4mCl Xe 133 INHALED	EQUILIBRUM	0-30 SEC	30-60 SEC	RT ANT LT	LT POST RT	RT RAO LT
19926au 60-90 SEC	90-120 SEC	2 120-150 SEC	150-180 SEC	LT LPO RT	RTLAT	LT LAT
				LT RPO RT	RT LAO LT	DOSE:5.3mCl TC99M MAA IV LT ARM





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ΡE

- S1Q3T3
- Inverted T waves in anterior leads



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FEMC Florida Emergency Medicine Clerkship • 67 y/o male with AMS



- Went to slow VTAQ (HR< 120)
- Was given amiodarone when,







• ESRD; missed HD





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EKG#2











ECG EFFECTS OF HYPERKALEMIA

Hyperkalemia exists when the serum potassium level is greater than 5.1 mEq/L. Causes for hyperkalemia include renal failure, adrenal insufficiency, acidosis, trauma or ischemia, potassium replacement therapy, and potassium-sparing drugs (e.g., diuretics such as spironolactone). Changes in the T wave (tall, peaked or tented) provide the earliest indication that a patient has a high serum potassium level. The following ECG changes may be seen in moderate (above rhythm) to extreme hyperkalemia:

Wide, tall and tented T waves
Wide, flat or absent P waves
Prolonged P-R interval
S-T segment depression
Widened QRS complexes





Slow VTACH

- If < 120/min
 - Think about something else
 - Hyperkalemia
 - AIVR
 - Na channel blocking medications
 - Antiarrhythmic IA
 - Anticholinergics
 - Cocaine
 - Many antipsychotics (TCA)
 - Some antibiotics (combinations of some macrolides, quinolones with antiarrhthymics)
- Treat underlying condition





Also, you may have AIVR after reperfusion post STEMI





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- LV aneurysm WPW
- New RAD
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- Prolonged QTc •
- Pericardial Tamponade
- PE
- Slow Vtaq

- AIVR
- Hyperkalemia
- Hypothermia
- CNS disorders
- Takotsubo
 Syndrome
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24 y/o male with chest pain and palpitations

• Diltiazem was given, then





• Patient collapsed

• Old ECG







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- RV AMI
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- Pericarditis
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- LV aneurysm
- New RAD
- Prolonged QTc
- Pericardial Tamponade
- **PE**
- Slow Vtaq

- AIVR
- WPW
- Hyperkalemia
- Hypothermia
- CNS disorders
- Takotsubo Syndrome
- Spiked-Helmet Sign





- If you have what it looks like a SVT but
 - HR >220/min
 - WPW
 - Different QRS morphology and irregular
 - Aflutter with VR aberrancy (WPW)
 - MAT
- Treatment
 - Cardioversion
 - Procainamide
 - Amiodarone?





- Patient homeless found unresponsive behind a gas station
- His respirations are agonal
- His pulse is bradycardic
- And is coldddddd...







Sorry for the EKG but it was from a patient of mine during residency maaaannnyyy years ago











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AIVR

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Osborne Waves or "J-Waves"



Here we see the Osborn waves of severe hypothermia (blue arrows).

The rhythm is atrial fibrillation.

Bradycardia is present.

The QT/QTc is prolonged.

The patient's core temperature was measured at 76°F (24°C).





- You are transporting a patient who has AMS
- While transporting the patient, he developed left side weakness, aphasia, and becomes unresponsive
- EKG monitor shows...



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CT showed....





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AIVR

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ROSC after cardiac arrest







EKG in the ED after arrival













Increased ICP

- It is a clear proof that heart and brain are interconnected by neural network
- All the noted changes occur during myocardial repolarization (i.e., ST segment)
 - Causing deep T inversion
- The current thinking is...
 - Mediated by adrenergic surge initiated by CNS insult transmitted to myocardium by the sympathetic system
 - Hypothalamic stimulation as well as autonomic dysregulations have been implicated as causative for the ECG changes





Increased ICP

Causes

- SAH
- ICH
- Traumatic brain injury
- Massive stroke
- You may also see...
 - Prolonged QTc
 - Bradycardia
 - Diffused ST elevations





- of central ergency Medicin
- 48 y/o white female that drove to the ED with the c/o dizziness, chest pressure
- She has a monitor attached to her that looks like this







- She removed it because it got activated and advised people to "get cleared"
- She removed it because she was afraid that it will "shock her" making her to fall down
- She did not take her metoprolol because her BP was low!!!





- PMHx: cardiac condition; had heart catherization two weeks ago
- PSHx: none
- All/Meds: in the computer!!!
- FamHx: none

















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- Holy crap!!!
- STEMI?
- Panic?
- Meds?





- Differential diagnosis
 - AMI
 - ACS
 - Myocarditis
 - Electrolyte imbalance
 - Cardiomyopathy
 - Idiopathic







Think AMI but also



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Takotsubo syndrome!




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Takotsubo Syndrome



Broken Heart Syndrome or Stress Cardiomyopathy







Normal ventricle



Takotsubo



Heart catherization



Echo-2D



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Stage 2: sub acute stage. This stage can last days. QT segment prolongation and large and deep negative T waves

Stage 1: acute stage, This stage lasts only a few hours. Stage of ST elevation and fairly short QT interval. The R wave might be preserved.



University of Central Florida/HCA GME Consortium Emergency Medicine Residency Program Of Greater Orlando Stage 3: recovery stage. Flipped T wave persists for days to weeks, but QT interval is again normal



- So,
- Back to our patient
- Most likely, she did have a run of Vfib or Vtaq
- Patient admitted, metoprolol given, started on Amiodarone while in the ICU





- Patient 65 y/o that was brought in by EMS after complaining of upper abdominal pain for the past 3 hours. The spouse stated that the patient was complaining of some discomfort when he started to vomit and to sweat.
- The spouse went to the bathroom after hearing a noise where the patient was found unresponsive.
- VS: HR 125/min RR 18/min BP 95/60 afebrile O2Sat 92%
- PE: diaphoretic, diffused tenderness





EKG







- Should call STEMI?
- No chest pain, but symptoms are atypical....
- Cardiac enzymes are WNL



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- Patient went to CT and had perforated bowel...
- Patient was taken to the OR then, to the ICU...
- Patient did not survive the hospital stay.



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EKG











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Spiked-Helmet Sign

- Appearance of helmet sign resembling a German military spiked helmet on electrocardiogram more commonly in inferior leads was described earlier in very sick people suffering from non-cardiac ailments.
- Mechanism underlying the appearance of helmet sign is unknown but it was postulated to be due to sudden increase in intra-abdominal pressure or synchronized contraction of the diaphragm with heart.
- Patients with acute abdominal or acute thoracic events occasionally develop a curious electrocardiographic ST-segment elevation, where the upward shift of the baseline starts before the onset of the QRS complex.





- The presence of this "spiked helmet" sign was found to be associated with critical noncardiac illness and high risk of in-hospital death.
- Prompt recognition and management of underlying acute noncardiac conditions resulted in resolution of the spiked helmet sign.
- The changes have been shown to accompany acute abdominal and thoracic events such as pancreatitis, pancreatic cancer, pneumomediastinum, and pneumothorax.





- The exact cause of the spiked helmet ECG pattern is uncertain.
- The most likely mechanism is pulsatile epidermal stretch resulting from an acute rise in intracavitary pressure.
- When seen in the inferior leads, this curious ECG pattern should raise the possibility of an acute abdominal event
- One should suspect an intrathoracic pathology if it shows up in the precordial leads





- Study a lot....
- See a lot of EKG's....
- So you can see a lot of normal EKG's....
- EKG's changes can mimic anything....
- The important thing is....
- Be able to recognize the one that is not normal!!!





