Advanced EKG’s

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The patient is a 23-year-old white male with a history of polysubstance abuse who was found unresponsive, last seen the day before.
Interpretation

- Classic signs of systemic hypothermia with prominent sinus bradycardia (about 45 bpm) with marked J (Osborn) waves simulating a bundle branch block or ischemic pattern.
- There are also prominent T wave inversions and QT prolongation.
- His rectal temperature was 80.0 degrees F. Following rewarming, his ECG completely normalized.
Osborn waves
T wave inversions
Prolonged QT interval
Bradycardic Rhythm
The patient is a 70-year-old man with a history of smoking and COPD who presented with cough, sputum production, and shortness of breath. Physical exam was notable for low grade fever, oxygen saturation of 94% on 2 liters via nasal cannula, and diffuse expiratory wheezes on lung exam. He was admitted for COPD exacerbation secondary to acute bronchitis. Admission EKG was initially interpreted by the computer as Mobitz type I, 2nd degree AV block. Prior EKG’s showed normal sinus rhythm with right bundle branch block.

On further examination of the EKG, what is the correct interpretation?
• Normal sinus rhythm with frequent premature atrial complexes producing the pattern of bigeminy.

• Right bundle branch block.
The patient is a 37-year-old obese woman who presented with breathlessness, tachypnea, and dyspnea on exertion for 24 hours. Symptoms forced her to leave work early. At home, her mother noted cyanotic lips and fingers. She also complained of upper back pain and recent non-productive cough. She denied fever, chest pain, diaphoresis, syncope, and hemoptysis. The following ECG was obtained in the emergency room:
Acute Pulmonary Embolism

ECG findings noted during the acute phase of pulmonary embolism can include any number of the following:

- “S1Q3T3” - prominent S in lead I, Q and inverted T in lead III
- Right shift of QRS axis
- Shift of transition zone from V3-4 to V5-6
- ST elevation in VI and aVR
- Generalized low-amplitude QRS
- Sinus tachycardia, atrial fibrillation/flutter, or right-sided PAC/PVC
- T wave inversion in V1-4, often a late sign.
Acute Pulmonary Embolism

- Right bundle branch block (RBBB), complete or incomplete, often resolving after acute phase
Inverted T
Low voltage QRS throughout
Mild ST elevation
Sinus Tachycardic Rhythm
A 35 year old male presents following a syncopal episode while running.

Case 3
Features of this EKG

- SR w/1° AVB, RAD, ischemic changes anteriorly
- Features of ARVD:
  - Incomplete BBB (V3)
  - Epsilon wave in V1-2
  - T-wave inversion in V1-4
Features of ARVD

- Complete or incomplete BBB (may have either L or RBBB)
- Epsilon wave
  - Epsilon waves are small deflections just beyond the QRS complex; they are best visualized in leads V1 through V3.
  - Any potential in leads V1 through V3 that exceeds the QRS duration in lead V6 by more than 25 milliseconds should be considered an epsilon wave.
- T wave inversion in the right precordial leads.
Epsilon wave

Inverted T waves

RBBB
An 64 year old male with multiple medical problems including heart disease is brought into the ED from the nursing home as a medical precaution. The floor nurse in the nursing home reports that he had a change in mental status today. On exam, the patient is minimally responsive. He appears moderately dyspneic. An EKG is taken. What is wrong?
This EKG demonstrates sinus rhythm at a rate of 65.

Several abnormalities:
- Artifact in V1
- ST-T wave suggesting electrolyte disturbance
- Prolonged QT interval (0.52 seconds)

DX: Hypocalcemia. This pt’s serum calcium level was 5.9mg/dl (N=8.5 to 10.2 mg/dL)
Artifact

Prolonged QT

ST-T wave changes
Four Major causes of Prolonged QT Interval

- **Electrolyte abnormalities:**
  - Hypocalcemia
  - Hypokalemia

- **Drugs: (associated with torsades de pointes)**
  - Class la antiarrhythmic agents, amiodarone, phenothiazines, Tricyclic antidepressants, sotalol

- **Congenital or idiopathic prolonged QT syndromes**
  - **Romano-Ward syndrome:** autosomal dominant inherited cardiac disorder characterized by abnormalities affecting the electrical system of the heart.
  - **Sporadic long QT syndrome:** disorder of cardiac repolarization caused by alterations in the transmembrane potassium and sodium currents.
Four Major causes of Prolonged QT Interval

- Miscellaneous group, including patients with:
  - Third-degree and sometimes second-degree AV block
  - At the cessation of ventricular pacing
  - Left ventricular hypertrophy (usually minor degrees of lengthening)
  - Myocardial infarction (in the evolutionary stages where there are marked repolarization abnormalities)
  - Significant active myocardial ischemia
  - Cerebrovascular accident (subarachnoid hemorrhage)
  - Hypothermia
Heart Rate Determined QT

- 115 - 84 bpm: QT 0.30 to 0.37 seconds
- 83 - 72 bpm: QT 0.32 to 0.40 seconds
- 71 - 63 bpm: QT 0.34 to 0.42 seconds
- 62 - 56 bpm: QT 0.36 to 0.43 seconds
- 55 - 45 bpm: QT 0.39 to 0.46 seconds

Men have shorter QT intervals (0.39 sec) than women (0.41 sec).
A 61 year old male presents to the emergency department within 7 hours of onset of a substernal chest pain that waxes and wanes in intensity. He has never had such pain before and has an active lifestyle. He worked as a plumber and smokes 20 cigarettes a day. On the following EKG, what striking characteristics are prominent and what do you tell the patient?
• This EKG has a RBBB configuration with a RAD. Additionally, there is RVH, poor r-wave progression & P-pulmonale pattern

• Chronic Lung Disease is characterized by:
  – Poor R wave progression in precordial leads
  – RAD
  – RAE
  – Low voltage QRS
Classic finding in Severe Right Atrial Enlargement (RAE)

Tall Peaked and Pointed P waves in the Pulmonary leads (II, III, aVF). If the P wave looks "uncomfortable to sit on", think RAE!!!
Diagnosed by finding an *m*-shaped (notched) and widened P wave (≥ 0.12 second) in a "mitral" leads (I, II, aVL) and/or a deep negative component to the P in lead V1.

Caused by conditions that increase either pressure or volume loading on the atria leading to enlargement and/or hypertrophy.

- Longstanding hypertension
- Obstructive cardiomyopathy
- Aortic stenosis
- Aortic regurgitation
Low voltage QRS RVH
RBBB
poor r-wave progression
42 year old obese female with chest pain presents to your ED at 5:00am. Symptoms have been present for the past 45 minutes.

She has no cardiac history, doesn’t smoke or drink. No viral infection history. She works as a RN in a nursing home.
Dextrocardia

• Sinus rhythm, RAD

• Features of Dextrocardia
  – Inverted P wave in Lead I
  – Right Axis Deviation (usually)
  – Abnormal chest leads
    • No R wave progression
    • QRS complexes becoming smaller from V1 to V6

Unusually small R waves in the lateral leads
Inverted P wave

Small QRS complexes

Small R waves laterally

Poor R wave progression
Let’s take a Break!!
42 year old male presents to the ED with a complaint of chest congestion and cough. He has a history of a heart transplant for ischemic cardiomyopathy 1 year ago. He has been afebrile, but his chest hurts from coughing.

- No tobacco, alcohol, or drugs.
• Native heart
  – S. Tach, extreme RAD
  – Features of old anterolateral MI with deep Q-waves in leads I & V3-6

• Donor heart
  – S. rhythm, normal axis
  – Features of Dextrocardia
    • Negative P in lead I
    • Small QRS complexes decreasing in size from V1 to V6
Heterotopic Heart Transplant
Auxillary (Heterotopic or ‘Piggyback’) Heart Transplant

Donor QRS complexes

Native QRS complexes
Deep Q-waves

Negative P’s

Deep Q-waves

Small QRS complexes V1 to V6
A 23 year-old male presented to the ED with a history of palpitations and vague chest discomfort. The patient was in the local county jail when he apparently had a syncopal episode. He was then brought to the jail nurse's office where he was awake and fully responsive. He was subsequently transferred to the ED by rescue squad. The only other available history was that the patient also complained of left shoulder pain. He had a history of asthma, but no other medical problems. There was apparently no history of drug abuse, but the accuracy of that information is obviously questionable - considering the patient's location at the time of the event.

On examination in the ED, he was alert and cooperative and in no distress. Blood pressure was 118/64, heart rate was 95 bpm, respiratory rate was 20 bpm and his temperature was normal. Pulse oximeter reading was 96%. Examination of the heart and lungs were normal. JVP was not elevated. Abdominal examination was only remarkable for mild epigastric tenderness. There was no pedal edema or calf tenderness. The EKG is as shown:
YOU’RE WRONG!!!! It’s early repolarization
Characteristics of early Repolarization

- Notching or slurring of the terminal portion of the QRS wave
- Symmetric concordant T waves of large amplitude
- Relative temporal stability (when patients are followed over time there will often be some variation in the degree of ST segment elevation but generally the change persists for years).
- Most commonly present in the precordial leads V2-5, but often associated with pronounced ST segment elevation in the limb leads II, III, & AvF
Early Repolarization

Concave ST Segments
J-point Elevation
The Athletic Heart

- May show the following:
  - Early repolarization
    - Prominent “J-waves” best seen in leads V5-6
    - Concave upward, minimally elevated ST segments
  - Relatively tall, frequent symmetrical T waves (usually asymmetric)
  - Prominent mid-precordial U-waves
  - Sinus bradycardia
  - LVH by voltage criteria
J-waves
Symmetrical T-waves
U-waves
Concave, elevated ST segments
J-wave
Symmetrical T-waves
J-waves
A 68-year-old man presented to the hospital emergency department at 01:00 hours complaining of chest heaviness. This had started in the afternoon after an argument with his foreman. The chest tightness had been intermittent earlier in the day, but since he awoke, it has lasted about 35 minutes. He had had a stress EKG and stress echocardiogram about 18 months prior to this admission to hospital and had been told that the test results were normal. His blood pressure at the time of presentation was 180/120; he is a nonsmoker with no history of diabetes or high cholesterol. His maternal grandmother had heart disease when in her 60s.
What is your Diagnosis?
• Hyperkalemia
  – Tall peaked T waves
• RAD (Left posterior fascicular block?)
• ST & T wave abnormalities suggesting myocardial injury
• PR prolongation (Seen in V2 only)
Peaked T waves

ST & T wave abnormalities
- Broadening of the QRS
- Peaked T waves
- Prolonged PR interval
- Disappearance of P wave
- Tall narrow T waves are the first sign of acute hyperkalemia
- $[K+]$ exceeds 5.5 mEq/L

**REMINDER:**

- $\geq 5.5$ mEq/L \hspace{2cm} T wave abnormalities
- $\geq 6.5$ mEq/L \hspace{2cm} Intervals widen
- $\geq 7.0$ mEq/L \hspace{2cm} P wave changes start
- $\geq 8.8$ mEq/L \hspace{2cm} P waves disappear
EKG Changes – P Waves

• Amplitude decreases while duration increases.

• When [K+] exceeds 8.8 mEq/L, P-Waves become absent.

• P-Waves may be absent due to underlying junctional or ventricular escape rhythm.
EMEDU

HYPERKALEMIA (POTASSIUM = 8.5)

Near Sinusoidal Pattern

Wide QRS Complexes

PR Prolongation

Peaked T waves
Case 11

- A 31-year-old patient comes in with a 3-week history of palpitations. In talking to him you learn he has cardiac risk factors for smoking, positive family history, high cholesterol and hypertension. He has a sedentary lifestyle and has not participated in any strenuous physical activity for years. Although his cardiac review of systems is negative (excluding current symptoms), you are concerned that his activity level is very limited and he has significant risk factors. As part of the workup you obtain the following EKG to interpret:
• Wolf-Parkinson-White Syndrome (WPW)

• The accessory conduction pathways act as short circuits, allowing the atrial wave of depolarization to bypass the AV node and activate the ventricles prematurely.

In this EKG: LAD, Q-waves, T-wave inversion in I, AVL, V3-6 representing ischemic changes.
ST elevation
Shortened PR interval
Q-waves
Flipped T’s
WPW Criteria

- Short PR interval, less than 3 small squares (< 0.12 sec) with a normal p wave
- Slurred upstroke to the QRS indicating pre-excitation (delta wave)
- Wide QRS complex (> 0.11 sec)
- Secondary ST and T wave changes
- Associated with paroxysmal tachycardia’s
WPW Types

• **Type A:**
  – QRS complexes are primarily upright in precordial leads.
  – Associated with accessory pathways in the left side of the heart

• **Type B:**
  – QRS complexes are negative in V1 or V2, and upright in the left sided precordial leads (V5-V6).
  – Associated with accessory pathways in the right side of the heart

• **Type C:**
  – QRS complexes are positive in V1-V4, and negative in V5 & V6.
  – This type is rare
Type C. Starts off as type A, but has negative delta waves in left lateral precordial leads.
Type B with pseudoinfarct pattern in the inferior leads. Also noted RBBB & LAD (bifascicular block?). The 6th complex is an PAC. Underlying rhythm is wandering atrial pacemaker.
Interpretation

- **Lown Ganong Levine Syndrome**
- Criteria for LGL includes:
  - PR interval less than or equal to 0.12 seconds
  - Normal QRS complex duration
  - Rare occurrence of supraventricular tachycardia but not atrial fibrillation or atrial flutter.
  - No delta wave

Case 12
Short PR interval, LVH by two criteria: R in aVF exceeds 20 mm & S+R is > 35 mm
A 69-year-old patient presents to the ED on an extremely busy Monday afternoon. The patient complains of several hours of severe chest pressure and as he lives nearby, he thought he would come to the ED and try to see a physician. The patient is not on any medications and has not seen a physician in years. Although the vital signs were remarkable only for some bradycardia, the triage nurse is concerned that the patient appears ill. A stat EKG was done and you are asked to see this patient immediately. The following EKG is handed to you as you are on your way to see this patient.
Ok, you suspect that he is having an MI,
But can you localize it?
Right Ventricular MI

- There are ST elevations & Q waves in II, III, & AVF. There are reciprocal changes present in leads I & AVL. Note that the ST elevation is taller in III than II, and there is ST elevation in V1 to V3. The p waves are only truly noted in V3 to V6 because they are impeding on the preceding T wave in other leads. The biphasic t-waves in the lateral precordials are caused by LVH with strain.

- Right sided leads were obtained. Notice the elevation in V4R as well.
P waves
ST elevation
Reciprocal changes
ST segment elevation greater in lead III than II
Inferior wall MI
ECG Evidence of RVI

- Inferior MI (always suspect RVI)
- ST elevation right Ventricular leads
- Proximal RCA Occlusion
- 12-lead ECG does not view right ventricle
- V3R - V6R
Right Ventricular MI Criteria

- Inferior wall MI
- ST segment elevation greater in lead III than II
- ST segment elevation in $V_1$ (possibly extending to $V_6$)
- ST depression in $V_2$ (unless elevation extends, as in #3 above)
- ST depression in $V_2$ cannot be > half the ST elevation in aVF
- More than 1 mm of ST elevation in the right-sided leads ($V_4R$ to $V_6R$)
Right Precordial Leads

- On right side of chest
- Same anatomical landmarks as V3 - V6

Figure 15-31: Placement of right-sided leads.
The right chest leads (V4R, V5R and V6R) show ST elevation indicating RVI. The proximal RCA must be occluded.
Physical Evidence of RVH

- Dyspnea with clear lungs
- Jugular vein distension
- Hypotension
  - Relative or absolute
Treatment for RVI

• Begin treatment like any other MI
• Use caution with vasodilators
  – unpredictable hemodynamic responses
  – Small incremental doses of MSo4
  – NTG by drip for afterload reduction
    • 0.3-0.5 mcg/kg/min IV initial infusion; use 0.5 mcg/kg/min increments and titrate to desired effect; average dose is 1-6 mcg/kg/min
Infarct in the anteroseptal and anterior wall (Q waves in V2-V4 there is also a probable inferior infarct (Q waves in II, III, and aVF)).
Ischemia across the entire anterior and lateral wall (T wave inversions in V2-V6, I and aVL). Also note, the injury pattern in V2-3 of ST elevation, the prominent Q waves in V2 and V3 show that some of the myocardium has also reached the infarct stage.
Left anterior fascicular block (left axis deviation and Q1, S3).
Left posterior fascicular block (right axis deviation and S1,Q3).
Right Bundle Branch Block and Left Anterior Fasicular Block

Q waves in I, AVL

QRS duration > 0.1 sec

rSR\(_1\) in V1-V3
Thank you!