Topics in Emergency Medicine
October 3, 2007

Difficult ECG’s

Return to Basics
- Determine rate and rhythm
- Determine intervals and axes
- Define morphology of P-QRS-T-U
- Compare with normal values
- Integrate with clinical presentation into meaningful conclusion

ECG – Rate and Rhythm
- Normal rate for adults – 60 to 100 bpm
- Speed Reading – Normal, Slow, Fast
- Normal rhythm for adults – Sinus with less than 10% variability of beat to beat
- Don’t forget premature atrial or ectopic beats
- Review p wave morphology to determine if the rhythm is sinus

Normal Intervals & Axes
- PR – 0.12 to 0.21 sec
- QRS – ≤ 0.10; 0.11 to 0.12 incomplete IVCD; abnormal > 0.12 sec
- QTc ≤ 0.44 sec
- P axis – 0 - 60°
- QRS axis - -30 to 90°
- T axis – generally follows the QRS axis except in the precordial leads where T waves should be upright from V2-6

Speed Reading Intervals & Axes
- PR < one large box
- QRS < 3 small boxes
- QT < 50% of RR interval
- Axis = QRS positive in Leads I & II
- T waves
  - Same direction as major QRS vector in limb leads
  - In limb leads, amplitude ~ 25% of QRS
  - Upright in V2-6

55 yo man awaken by chest pain
Question #1
Which are the correct answers?

1. This ECG excludes an Acute MI
2. None of the above or below
3. This is a right-sided ECG
4. I am a ED-MD, it takes me longer than 1.8 sec to interpret (but I can’t take all the time I want)

Electrically silent MI’s

- Right Ventricular MI – Right sided ECG
- High Lateral MI – V7, V8, V9
- Small Apical MI

78 yo man with intracerebral hemorrhage

RA – RL Cable Reversal

- Far Field Signal in II
- No structural abnormality can explain this finding
- The Technician’s Test
43 yo obese man admitted with chest pain.

Evolution of ECG in AMI
- Hyperacute T wave
- ST elevation
- Q waves + Loss of R wave
- T wave inversion

Measure ST\textup{↑} in relation to preceding PR segment

Septal MI

Inferior Wall MI
If the patient’s history and clinical presentation is consistent with ACS – IT DOESN’T MATTER WHAT THE ECG LOOKS LIKE!

THE WORRISOME CLINICAL PRESENTATION OF CONCERN IS “THIS PATIENT LOOKS LIKE １/œ!!”
ST Elevation – Common Causes

- Acute myocardial injury pattern
- Acute pericarditis
- Early Repolarization / Normal Variant
  - $+0.1mV$ in any lead
  - Up to $0.3mV$ in young men in early precordial leads
- Myocardial aneurysm

ST Elevation – less common causes

- Pulmonary embolism and acute cor pulmonale (usually lead III)
- Cardiac tumor
- Acute aortic dissection
- After mitral valvuloplasty
- Pancreatitis and gallbladder disease and other catastrophic illness
- Myocarditis

- Septic shock
- Anaphylactic reaction
- J wave
- Hyperkalemia (“dialyzable” current of injury)
- With any marked QRS widening (e.g., antidepressant drug overdose) or Class IC antiarrhythmic drugs

45 yo woman with nausea and vomiting
Which of the following is **not** true about RBBB?

A. RBBB occurs in up to 29% of AMI  
B. The most common cause in children is post open heart surgery for Tetralogy of Fallot repair  
C. Post MI patients with persistent RBBB are at the same risk for cardiac mortality  
D. Transient RBBB is a complication of right heart catheterization

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**Differential Dx of Tall R wave in V1**

- Definition – R/S ratio > 1 in V1 or V2  
- RBBB  
- RVH  
- True Posterior MI  
- WPW  
- Brugada’s Abnormality

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**65 yo Asian man who presents with recurrent syncope. Echo was normal.**

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**Brugada Syndrome**

- Brugada Abnormality is defined as RBBB with ST elevation and occurrence of sudden cardiac death or syncope due to polymorphous VT (1990)  
- Hereditary bundle branch defect  
- Autosomal dominant trait with variable expression  
- Diagnosis by EPS with procainamide stimulation  
- TX: AICD

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**36 yo woman presenting to the ED with acute dyspnea.**

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**79 yo man admitted with CVA**
**Diffuse (“Global”) T Wave Inversion**
- Myocardial Ischemia (Evolving MI is focal)
- CNS event
- Apical HCM
- Pericarditis
- Myocarditis
- Takatsubo’s Cardiomyopathy
- Cardiac metastases
- Carotid endarterectomy
- Cocaine abuse
- Pheochromocytoma
- Acute illness in women

**Which of the following answers about LBBB is not true?**
A. LBBB can be caused by toxic, inflammatory changes, hyperkalemia, or digitalis toxicity
B. In most patients with LBBB, septal wall motion abnormalities can exist without coronary artery disease
C. In patients presenting to the ED with chest pain and a LBBB the management is observe with frequent ECGs, treat pain and await enzymes
D. The QRS duration is at least 0.12 seconds

**The most likely diagnosis is:**
A. Primary Pulmonary Hypertension
B. Atrial Septal Defect
C. Ventricular Septal Defect
D. Mitral Stenosis
E. Aortic Stenosis

**Biatrial Abnormality**
- A large diphasic P in V₁ with the positive component > 1.5 mm and the terminal negative component reaching 1mm in amplitude, > 0.04 sec in duration or both
- Tall peaked P wave (>1.5mm) in the right precordial lead and a wide notched P wave in the limb leads or lateral leads (V₅-V₆)
- Increase in both the amplitude (>2.5mm) and duration (>0.12 sec) of the P wave in the limb leads
Non Conducted PAC’s
Rate-Related RBBB
56 yo man presents with flu
This ECG represents:
1. Acute Myocardial Infarction with alternating bundles
2. Sinus rhythm with PVC’s
3. Ashman’s Phenomenon
4. He’s got more going on than a flu
5. A cardiac emergency requiring pacemaker implantation
70 yo woman admitted for weakness
Prolonged QTc = 498 msec
24 yo woman with FHX of SCD presents with syncope
Afib with AV Dissociation
78 yo man with hx of HF presents with nausea
What is the most appropriate next test?

1. Serial Troponins
2. Serial CK with iso’s
3. Digoxin level
4. Serum potassium level
5. I don’t know

AFib with AV Dissociation

AFib or AFib/Aflutter or Aflutter?

What is the rhythm? What is the treatment?

What is the rhythm? What is the treatment?
Supraventricular Tachycardias: Which statement is true?

1. If hemodynamically stable, acute management includes Adenosine by slow IV push
2. The majority of SVT is AVNRT
3. AVRT does not involve accessory bypass tracts
4. If the patient after adenosine converts from a narrow complex tachycardia to a wide complex tachycardia, the patient has degenerated to ventricular tachycardia

Slow-fast form of AVNRT: Representation of dual pathway physiology involving the AV node and perinodal atrial tissue in the common form of AVNRT. Left panel: A normal sinus beat (A1) is conducted through the fast pathway (F) to the final common pathway (F1g) in the AV node and into the Bundle of His. The conduction through the slow pathway (S) runs into the refractory period of the impulse through the fast pathway and is extinguished ( Insets panel). At critically timed atrial premature beat (A2) founds the fast pathway refractory but is able to conduct through the slow pathway which has a shorter refractory period. If excitability in the fast pathway has recovered by the time the impulse reaches the Fg, there may be the retrograde activation of the fast pathway. Right panel: The retrograde impulse travels up the atrium (A'), and, if the slow pathway has recovered its excitability, the impulse re-enters the slow pathway and produces ventricular depolarization (V'). If the mechanism persists, a repetitive circuit is established that creates a sustained, ventricular tachycardia. The sequence of antegrade (S) and retrograde (F) conduction is called the slow-fast form of AVNRT.

Generation of ECG in common form of AVNRT: Ladder diagram of the common (slow-fast) form of AVNRT showing transmission of the impulse in atrial, AV nodal, and ventricular tissues and the resultant electrocardiogram. The first two cycles show the normal sinus beat (A1) conducting through the fast pathway (F) to the ventricle (V1) with the impulse blocked in the slow pathway. The premature atrial beat (A2) is of critical maturity; it finds the fast pathway refractory, but is able to conduct through the slow pathway resulting in an inverted P wave (P), a long PR interval, and a ventricular depolarization (V2). It is also able to reenter and conduct retrogradely up the now recovered fast pathway, resulting in an inverted P wave that is buried in the QRS. This impulse gives rise to an atrial extra beat (A*) and also reenters the now recovered slow pathway, which eventuates in ventricular depolarization (V*). The cycle repeats and the circus movement tachycardia is sustained.
34 yo woman with idiopathic cardiomyopathy became dizzy

Wide QRS-complex tachycardia
(QRS > 120 ms)

Regular or irregular?

Regular

Previous MI or structural heart disease? If yes, VT is likely

1:1 AV relationship?

Yes or unknown

QRS morphology in precordial leads

VT

Typical RBBB or LBBB = SVT

Precordial leads

VT

Right axis

 Concordant

No R/S pattern

Onset of R to nadir > 100 ms

RBBB pattern

VT

qR, Rs or Rr’ in V1

Frontal plane axis range from +90 degrees to –90 degrees

LBBB pattern

VT

R in V1 > 30 ms

R to nadir of S in V1 > 60 ms

qR or qS in V6

VT

Atrial Flutter

Atrial Flutter/AT with variable conduction and

a) BBB or

b) Antegrade conduction via AP

Ventricular Tachycardia

Atrial Tachycardia

Atrial Flutter
A fib with WPW

65 yo man presents with dyspnea and palpitations

82 yo man presents with chronic fatigue. Why is he on your service?

The correct diagnosis is:
1. Acute anterior myocardial infarction
2. Congestive Heart Failure
3. Sick Sinus Syndrome
4. Complete Heart Block with an artifact
5. Wandering Atrial Pacemaker

82 yo man presents with chronic fatigue. Why is he on your service?

75 yo AA male presents with prolonged CP
Prognostic Value of Lead aVR in Patients with a First Non-ST segment Elevation Acute Myocardial Infarction

Odds ratio for death in groups 2 and 3 – 4.2 and 6.6


First Degree AV Block

- Generally benign
- Bad prognostic sign in:
  - Bifascicular Block
    - RBBB + LAFB
    - RBBB + LPFB
    - LBBB
  - Infectious endocarditis

Q: Had this patient suffered a myocardial infarction?

1. No!
2. Yes. Inferior
3. Yes. Posterior
4. Yes. Septal
5. Yes. Localization not possible due to RBBB

75 yo man presents with palpitations
80 yo man with hx of IMI presents with syncope

Thank you