

### Methods for determining rate:

- Rate=300/# of large boxes in R-R interval
- Count the # of large boxes in R-R interval and memorize 300/150/100/75/60/50/43
- Count the number of QRS in a six second span (30 large boxes) and multiply by ten – this is especially helpful for irregular rhythms
- Just look at the top of the EKG

### Chamber enlargement

#### RAE

- EKG: Tall peaked P wave >2.5mV in II, tall initial upstroke of P in V1, nl P wave width
- Causes: TR, RVH, ASD, Pulm HTN

#### LAE

- EKG: Notched P wave in II, negative deflection P wave in V1, P wider than .11 sec
- Causes: MS, MR, LVH

#### RVH

- EKG: RAD, R > S in V1, R > 7mm in V1, R > 5mm in aVR, S wave < 2mm in V1, Sokolow-Lyon voltage criteria: R in V1 + S in V5 or V6 > 1.1 mV
- Causes: Pulm disease, pulm emboli, congenital heart disease, PS, sleep apnea

#### LVH

- EKG: LAD, Deep S waves in V1 & V2, prolonged QRS, ST depression & asymmetric T wave inversion in left side precordial leads, voltage criteria:
  - Sokolow-Lyon Criteria: S in V1 or V2 + R in V5 or V6 > 3.5mV; R in V5 or V6 > 2.6mV; R in aVL > 1.2mV
  - Cornell Criteria: Females: R in aVL + S in V3 > 2mV; Males: R in aVL + S in V3 > 2.8mV
- Causes: HTN, AS, HOCM

#### Strain pattern:

- EKG: Downsloping convex ST segment with an asymmetrically inverted T wave opposite to the QRS axis in leads V5 and/or V6
- Extra: Marker of increased cardiovascular risk, especially in HTN patients

#### Low voltage

- EKG: QRS < 5mm in all limb leads or < 10 in all precordial leads
- Causes: pericardial effusion, obesity, diffuse CAD, restrictive CMY, myxedema, COPD (only precordial leads)

#### R wave progression

- EKG: Normally QRS become positive in V3 or V4
- Causes of early R wave progression: posterior MI, RVH

- Causes of poor R wave progression: anterior or septal MI, LVH, LBBB, COPD, cardiomyopathy, normal variant

### **Axis:**

#### **RAD**

- EKG: +90-180; - in I, + in aVF
- Causes: pulm HTN, PS, PE, RVH, congenital heart disease, lateral MI, LPFB, pacing

#### **LAD**

- EKG: -30-90; + in I, - in aVF, - in II
- Causes: LVH, aging, inferior MI, LAFB, pacing

### **AV Block:**

#### **First degree block**

- EKG: PR >.2 sec, constant PR interval
- Causes: Dig, BB, CCB, Class IC antiarrhythmics, increase vagal tone, hyperkalemia, inferior MI, myocarditis, age related degeneration

#### **Second degree block Mobitz I (Wenckebach)**

- Characteristics: Block occurs within the AV node, usually transient
- EKG: PR lengthens & R-R gets shorter until QRS dropped, P-P constant, irregular rhythm due to dropped beats, narrow QRS
- Causes: Dig, BB, CCB, Class IC antiarrhythmics, increase vagal tone, inferior MI, ischemic heart disease, myocarditis, normal variant

#### **Second degree block Mobitz II**

- Characteristics: Block often found below bundle of His within bundle branch system, can progress to CHB without warning
- EKG: PR constant, P-P is regular, irregular rhythm due to dropped beats, QRS a bit wider than normal
- Causes: Anterior MI, age related degeneration
- Treatment: Pacemaker, especially if symptomatic

#### **Third degree block (Complete heart block)**

- Characteristics: Complete absence of conduction with AV dissociation
- EKG: P-P & R-R intervals are constant, atrial & ventricular rates are different, no relationship between P & QRS, use QRS width & ventricular rate to determine escape pacer location
- Causes: Dig, BB, CCB, Class IC antiarrhythmics, MI, increase vagal tone, myocarditis, endocarditis, age related degeneration, cardiac surgery, congenital
- Treatment: Pacemaker

## **Bundle blocks:**

### **RBBB**

- EKG: QRS > .12 sec, rSR' in V1 & V2, wide deep S in left-side leads, downsloping ST & inverted T in V1 & V2
- Causes: RVH, RV strain, congenital disease (ASD), primary pulmonary disease, CAD, myocarditis, degenerative conduction disease, WPW, unknown causes

### **LBBB**

- EKG: QRS > .12 sec, wide deep S in V1 & V2, rsR' in left side leads, ST & T oriented opposite to QRS
- Causes: STEMI, LVH, cardiomyopathy, HTN, CAD, WPW, idiopathic degenerative disease

### **LAFB**

- Characteristics: Single long & thin blood supply, more vulnerable
- EKG: LAD, small q & tall R in left leads; small r & deep S in inferior leads, poor R wave progression

### **LPFB**

- Characteristics: Double short & thick blood supply, supplies more area & more serious
- EKG: RAD, small r & deep S in left leads; small q & tall R in inferior leads

## **Rhythm:**

### **Normal sinus rhythm**

- EKG: upright P waves in I, II, V1 with constant morphology, P-P & R-R intervals are equal & regular, atrial & ventricular rates identical 60-100

### **Supraventricular dysrhythmias:**

#### **Premature Atrial Complexes**

- EKG: Irregular R-R interval, P wave shape different from the sinus P
- Extra: If APC occurs early & AV node is not repolarized, P wave will be blocked and not conduct causing a pause

#### **Atrial tachycardia**

- Characteristics: Rhythm originating in atria, but outside SA node, rate 120-250
- EKG: P-P & R-R interval equal, P waves different from sinus P

Atrial Flutter – F waves at atrial rate 250-350, regular or irregular depending on conduction, QRS narrow

Atrial fibrillation – f waves at atrial rate >350, irregular R-R, QRS narrow

#### **Wandering pacemaker**

- Characteristics: Pacer from 2 or more sites in atria

- EKG: P wave shape varies, P-P & R-R intervals vary, QRS unchanged & narrow, rate 60-100
- Extra: MAT similar, except with 3 or more atrial foci, rate 100-250, associated with COPD

#### Paroxysmal SVT – reentry circuits

- AVNRT
  - Characteristics: More common, down micro-reentry circuit in AV node, conducted rapidly down a second pathway in AV node
  - EKG: Retrograde P usually buried or barely seen, usually ventricular rate 100-200
- AVRT
  - Characteristics: Uses a macro-reentry circuit called bundle of Kent bypassing the AV node
  - EKG: Retrograde P commonly seen after QRS, usually ventricular rate > 100
  - Orthodromic AVRT – more common
    - Characteristics: Down AV node to ventricles & back up atria; narrow QRS
  - Antidromic AVRT
    - Characteristics: Down bypass tract to ventricles & back up through AV node; wide QRS. Looks like VT but irregularly irregular with some very short R-R intervals. No AV nodal agents (BB, CCB, Dig)!!

#### Wolf-Parkinson-White

- EKG: PR interval < .12 sec, wide QRS, delta wave
- Type A
  - Characteristics: Bypass tract links left atrium & ventricle
  - EKG: Positive QRS in V1
- Type B
  - Characteristics: Bypass tract links right atrium & ventricle
  - EKG: Negative QRS in V1

#### Junctional escape rhythm

- EKG: Rate 40-60, regular, narrow QRS, P before, before or buried in QRS
- Extra: Accelerated junctional rhythm is rate 60-100, junctional tachycardia is >100

#### Ventricular escape rhythm

- EKG: Rate 15-40, regular, wide QRS
- Extra: Accelerated idioventricular rhythm is rate 40-100, ventricular tachycardia is >100

#### Ventricular dysrhythmias:

##### Premature ventricular complex

- Characteristics: premature ectopic impulse originating below bundle of His
- EKG: Irregular R-R, QRS wide & different shape, ST & T are in opposite direction of the ectopic QRS, compensatory pause as sinus node is not reset

Ventricular tachycardia – wide QRS, three or more beats in a row, complexes roughly similar in appearance, regular

Ventricular flutter – rate 250-300

Ventricular fibrillation – no cardiac output, heart rate or blood pressure; early u see coarse V fib, later is finer V fib

Ventricular asystole – no waveforms

Torsades de Pointes

- Characteristics: A variant of VTach, associated with prolonged QT
- EKG: VTach where QRS twist around baseline
- Causes: Class IA drugs, Class III drugs, electrolyte abnormalities (hypokalemia, hypomagnesemia, hypocalcemia), psychotropic drugs, liquid protein diet, congenital QT prolongation, MI
- Treatment: discontinue offending agent, fix electrolytes, cardioversion, magnesium, lidocaine, isoproterenol, dilantin or overdrive pacing; beta blockers for congenital Long QT Syndrome, but not acquired forms

Clues for VTach

- Characteristics: More likely if history of cardiomyopathy or ischemic heart disease
- EKG: QRS > .14 sec, regular, LAD at -90 to -180, AV dissociation, precordial leads all positive or negative

Clues for SVT with aberrancy

- EKG: QRS < .14 sec, irregular, initial part of QRS resembles normal QRS

### **Ischemia, infarct:**

Ischemia

- Characteristics: Temporary reversible blood flow reduction
- EKG: Inverted symmetric T waves or pseudonormalization of a pre-existing T wave inversion

Injury

- Characteristics: From acute prolonged reduction in blood supply, reversibly if flow established before death occurs
- EKG: ST depression represents subendocardial injury; ST elevation represents subepicardial injury

Infarction

- Characteristics: Irreversible death
- EKG: Significant Q waves which are > 25% of R wave height and > .04 sec

See page 39 for Angiographic correlation & EKG changes of various MIs

## **EKG in various medical conditions:**

### Hypothermia

- Characteristics: Depressed electrical & mechanical activity
- EKG: Sinus brady, PR prolongation, QRS widening, QT lengthening, ventricular dysrhythmias, Osborn waves (J wave) in the terminal portion of QRS
- Extra: Height of J wave proportional to the degree of hypothermia

### CVA

- Characteristics: Affects repolarization, resolves over time
- EKG: Tall peaked or deeply inverted T waves, wide T waves, prolonged QT, prominent U waves, Q waves

### PE

- Characteristics: Acute right heart strain
- EKG: Acute RAD, RAE, new RBBB, S1Q3T3 (S wave in lead I, Q wave in lead III, inverted T in III)

COPD – RAD, RAE, RVE, low voltage, poor R wave progression

Hypothyroid – Low voltage, sinus brady, inverted T without ST deviation in most leads

Hyperthyroid – Sinus tachy, supraventricular dysrhythmias

### Pericarditis

- EKG: Widespread concave up ST elevation in every lead except aVR, no pathologic Q, PR depression
- Extra: With resolution of pericarditis, elevated ST segments resolve, but T waves may invert or become biphasic and stay that way for weeks

Ventricular aneurysm – Persistent ST elevation, may stay for years

ASD – RBBB, RAD, RAE, RVH

MS – LAE, RVH, supraventricular dysrhythmias

AS – LAE, LVH

### Orthotopic transplant

- EKG: occasionally 2 P waves are present from a retained native SA node, non-specific ST changes, normal resting baseline tachycardia
- Rejection: low voltage, prolonged PR, RAD, RBBB, dysrhythmias

## **Drug effects:**

### Digoxin

- Dig effect: ST depression in inferior & lateral leads and look scooped out & coved, elevated segment in V1, flattened or inverted T, shortened QTc, prolonged PR, depressed J, decreased ventricular rate

- Dig toxicity: similar to dig effect, significant PR prolongation, supraventricular & ventricular dysrhythmias, high-degree AV block

IA drugs - Prolonged QTc, widened QRS, ST depression, T wave changes, prominent U

IB – Shortened QTc

IC – Prolonged PR, widened QRS, prolonged QTc, serious & refractory ventricular dysrhythmias

III – Prolonged QTc

### **Electrolyte abnormalities:**

Hypokalemia

- Characteristics: Prolongs repolarization
- EKG: T flattening or inversion, U waves best in V2-V4, T-U fusion, ST depression, prominent P, prolonged PR

Hyperkalemia

- Characteristics: Abnormal slowing of conduction
- EKG: Initially, there are tall peaked T waves best in V2-V4 & wide flat or absent P waves. In severe cases, there's widened QRS & QRS-T fusion that progress to sinusoidal waveform. Also, can see ST depression, prolonged PR to AV block, ventricular dysrhythmias

Hypomagnesia – similar to hypokalemia

Hypermagnesia – similar to hyperkalemia

Hypocalcemia – prolonged ST, prolonged QTc, dysrhythmias

Hypercalcemia – shortened ST, shortened QTc, dysrhythmias

$QTc = QT/\sqrt{RR}$