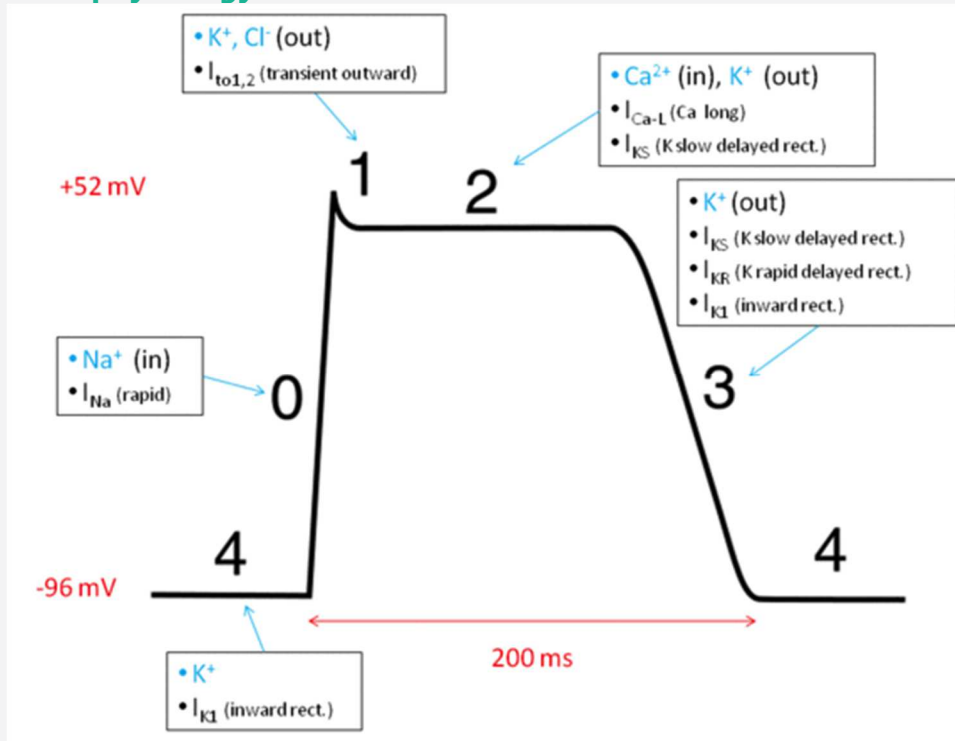


ECG Interpretation

Sept 2014

Pathophysiology



Pacemaker Rates SAN: 60-100 AVN: 40-60 Ventricle: 20-40

Rate

Horizontal: 1mm = 0.04s 5mm = 0.2s

Rate = 300 / big squares

1 line = 300 2 line = 150 3 line = 75 4 line = 60 5 line = 50 6 line = 42 7 line = 38

Or, count no. complexes in 6 secs and x10

Or, count no. complexes on strip and x6

Axis

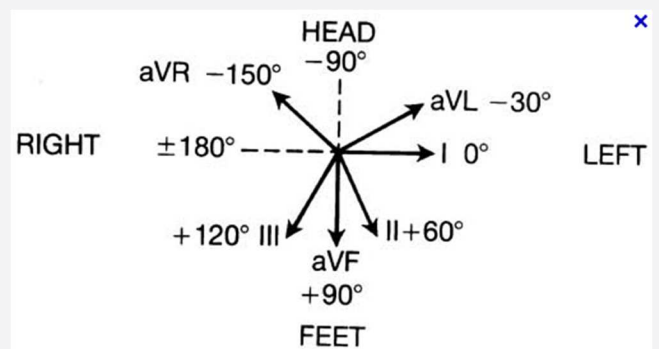
Normal = -30 to +90

Normal = +ve in I and either II/aVF

LAD = -90 to -30

RAD = +90 to +180

Extreme axis deviation = +180 to -90 (I, II and aVF negative)



Use lead I and aVF to determine

If I+ and aVF-, use II

Young people have RAD, old people have LAD

Transitional Lead

Where QRS is equally positive and negative, should occur at V3-4

Transition zone at V1-2 = counterclockwise rotation

Transition zone at V5-6 = clockwise rotation

**LAD causes:**

LVH – most common

LAFB – LAD must be present for LAFB to exist

Inferior MI – due to Q wave in inferior leads

LBBB – LAD suggests severe conducting system disease

Mechanical shifts of heart eg. pneumothorax, emphysema

Others: Ectopic ventricular rhythms (eg. VT), Pacing, HyperK, Pregnancy, WPW (R sided accessory pathway), Tricuspid atresia, Ostium primum ASD

RAD causes:

RVH (PE; COPD (+/- pul HTN))

LPFB

Antero-lateral MI – due to Q wave in I

RBBB

Infants and children; normal young/thin adults

Mechanical shifts of heart eg. Pneumothorax

Dextrocardia – inversion of P and QRS in lead I, QRS complexes decrease from V1-6

Limb electrode misplacement – L/R arm reversal; P, QRS and T waves inverted in I

Others: Ectopic ventricular rhythms, WPW (L sided accessory pathway), ASD / VSD

Extreme/NW axis deviation:

Dextrocardia

COPD (emphysema)

Pacing

HyperK

VT

Incorrect lead placement

Anticlockwise rotation:

Electrical shift to R: RVH, WPW, post MI, L septal fascicular block

Septal shift to R: HOCM

Clockwise rotation:

IV conduction abnormalities 2Y to myocardial degeneration: RVHD

Septal shift to L: dilated CM

Heart shift to L: emphysema, tall thin person

Poor R wave progression:

Suggestive of infarction

Tall R waves in V1-2 suggest Q waves of posterior infarction

Dextrocardia

Reverse R wave progression:

R waves getting smaller from V1-4 suggests infarction/precordial lead reversal

Incorrect lead placement:

aVR should always have inverted P, QRS and T – if not, incorrect lead placement

L arm / R arm reversal = RAD

L arm / L leg reversal = taller P wave in I than II

R arm / L leg reversal = positive inflections in aVR, negative in all I, II and III

R leg / either arm reversal = flat line appearance to one of I – III

Precordial reversal = abnormality to R wave progression

Limb lead reversal: P/QRS inverted in lead I; normal R wave progression therefore not dextrocardia



Complexes

P wave:

atrial depolarisation

negative in aVR (maybe in aVF, aVL; biphasic in V1 (and maybe V2, III); Normal axis 0 to +75 (if abnormal, ectopic atrial focus)

Duration: 0.12s

Size: <2.5mm

RAH/RAD/P pulmonale: large P wave in aVF, II, III, +ive in V1 eg. pulmonary disease, congenital heart disease, incorrect lead placement, dextrocardia

P mitrale: in I and II; deeper negative aspect in V1; >0.11s; more prominent in I, aVL, V5-6; most specific ECG sign of LAH, usually due to MS

Biatlial abnormality: P wave wide and notched and large

Retrograde P' wave: impulses from near AVN activate atria; P waves inverted in II, III, aVF; may be buried in QRS complex

PR interval:

P + PR

Duration: 0.12 – 0.2s (Increases with age; decreases as rate increases)

Short: exercise tachycardia; WPW; low ectopic atrial rhythm, AV junctional rhythm; neonate

Long: AV block, hyperK, cardiomyopathy, digoxin, beta-blockers, hypothyroid, hypothermia, old

Elevation: myopericarditis (in aVR, V1), atrial infarction

Depression: myopericarditis (mostly in II, V5-6), atrial infarction, exercise- induced sinus tachy

Q wave:

depolarization heads L \diamond R in V septum

Normal = small in I, II, III (longer and deeper), aVL, aVR, V4, V5, V6

Duration: <1 box wide

Size: <4 small boxes / <25% height of R wave deep

Abnormal = any in V1-3; suggest MI, V enlargement, abnormal V conduction

R wave:

depolarization of L+R V myocardium, heads L and posterior

Normal = small R waves in V1-2; large R waves in V5-6; R wave progression

S wave:

depolarization goes posteriorly

Normal = S wave in anterior leads; usually largest in aVR and V2; get smaller from V1-6

R' wave:

any positive deflection that occurs following S wave

J Point:

Junction of terminal QRS and initial ST segment

Osborn wave: in hypothermia; elevation of the J point; prominent in II, III, aVF, V5 and 6

Elevation: hyperK

QRS:

Axis: -30 to +90

Duration: <0.12s

Size: 10 – 30mm in precordial leads



QRS Incr amplitude: V enlargement, athletes, normal variant, BER, hyperthyroid

QRS Decr amplitude: pericardial effusion, amyloid, myxoedema, nephrotic syndrome, anascara, pneumothorax, pleural effusion, restrictive cardiomyopathy, COPD

QRS Incr duration: V enlargement, LBBB, RBBB, ventricular ectopic, pacing, cardiomyopathy, **hyperK**, pre-excitation, pericardial effusion, amyloidosis, myxoedema, nephrotic syndrome, anascara, pneumothorax, pleural effusion, restrictive cardiomyopathy, COPD, **hypothermia**

QRS PROLONGERS = Na channel blockers = delay phase 0 = depolarisation

Type Ia (procainamide, quinidine), **Type Ic** (flecainide)

TCA's (abnormal R sided R in aVR), **Carbamazepine, Cocaine**

Quinine, chloroquine

Phenothiazines (eg. Chlorprom, prochlorperazine)

Antihistamines (diphenhydramine)

Type IV (Diltiazem, verapamil), **Propranolol**

LA's, amantadine

Ix: look for large R in aVR (>3mm), QRS >100 in II

Trt: HCO₃ if: QRS >100, persistent decr BP despite IVF, significant arrhythmia, seizures

T wave:

should be in same direction as QRS

Size: <10mm in precordial leads; May be flattened in aVF; inverted in aVR, III; variable in aVL/V1

Prominent: ACS (within 30mins), hyperK, BER, myopericarditis, BBB, LVH

Inverted: ACS, post-MI, BBB, pericarditis, PE, LVH, digoxin, CNS injury, paced rhythm, intra-abdominal disorders, metabolic syndromes, toxic, pre-excitation; Wellen's syndrome (deep T wave inversion of precordial leads)

ST segment:

Rule of appropriate discordance: in BBB, ST segment / T wave is directed opposite to terminal portion / major vector of QRS complex; so in RBBB, STE seen in lateral leads; in LBBB, STE seen in R to mid-precordial and inferior leads; this rule also applies to LVH (STE in R to mid-precordial leads) and paced rhythm

Elevated: ACS, Prinzmetal's angina, MI, acute pericarditis, BER, LV aneurysm, LBBB, RBBB, LVH, paced rhythm, cardiomyopathy, myocarditis, hypothermia, hyperK, post-cardioversion, contusion, CNS injury, Brugada syndrome, pre-excitation

Depressed: ACS, ischaemia, NSTEMI, BBB, LVH, paced rhythm, digoxin, rate-related, metabolic syndromes, post-cardioversion, contusion

QT interval: $QTc = QT (eg. 0.52) / \sqrt{RR}$

Duration: <0.44s (no more than half *preceding* RR interval if HR 60-100)

Short: digoxin, hyperCa, hereditary (rare)

Long: long QT, CNS system disease (eg. Incr ICP, SAH)

metabolic (hypoK, hypoCa, hypoMg, hypothyroid, hypothermia)

Lange-Neilson (autosomal recessive, assoc with sensorineural deafness)

Romano-Ward (autosomal dominant)

QTC PROLONGERS = K channel blockers = delays phase 3 = repolarisation

Type Ia (procainamide, quinidine) **type Ic** (flecainide)

TCA's, Carbamazepine, Cocaine

Quinine, chloroquine

Phenothiazines (eg. Chlorprom, prochlorperazine)

Antihistamines (diphenhydramine, terfenadine)

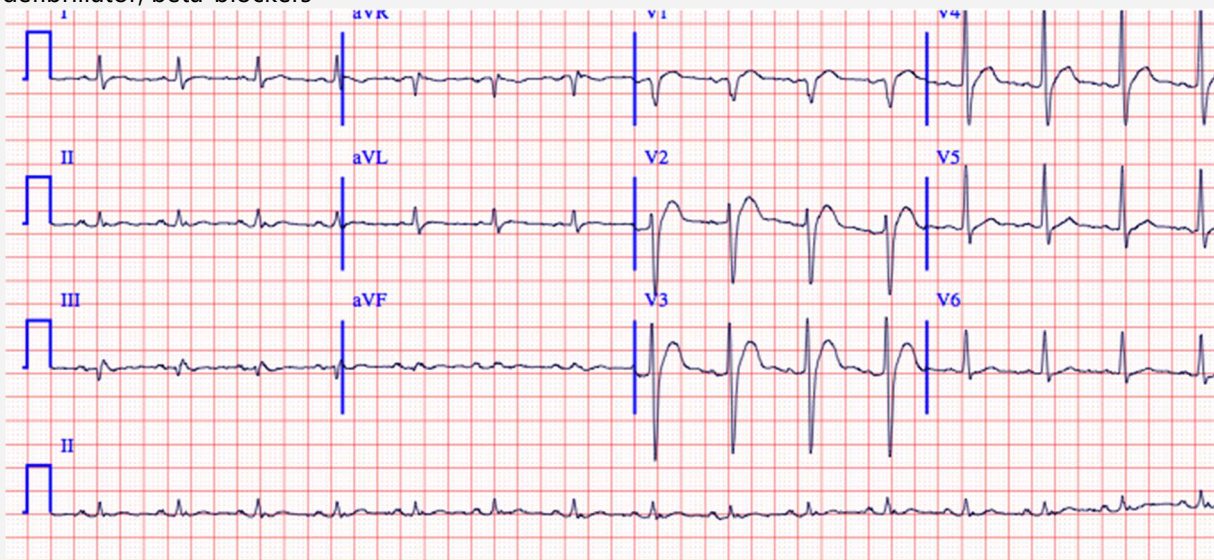
Type III (amiodarone, sotalol), **Type IV** (not verapamil / diltiazem)

Sumatriptan, antipsychotics (Haloperidol, quetiapine, droperidol), **SSRI, methadone, lithium**
Erythromycin, clarithromycin, tetracyclines

OP's, omeprazole, ondansetron

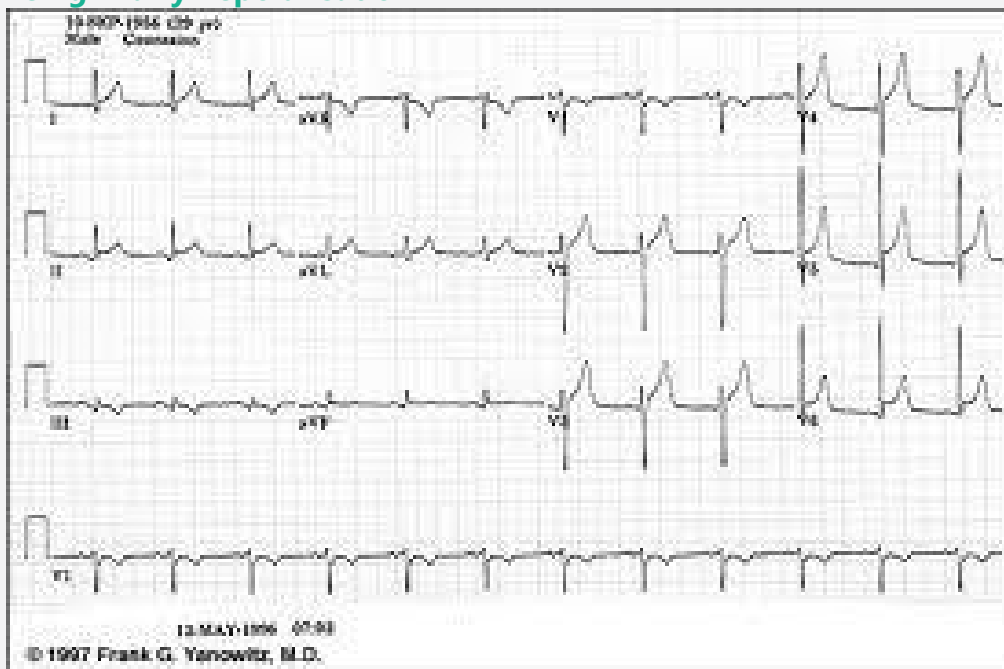
Treatment: HCO₃, MgSO₄, K to 4.5-5, overdrive pacing, Ca

Long QT: Broad T waves with notching – increased risk of TdP, syncope, sudden death; consider for implantable defibrillator, beta-blockers



HyperCa: short ST and short QT

Benign Early Repolarisation



1-2% population; more common in males, athletes, 20-40yrs, blacks

23-48% cocaine users with chest pain; more prominent at slow HR's

DDx: pericarditis (but no PR depression and pericarditis doesn't have large T waves), MI, LBBB, LVH, LV aneurysm, V paced rhythm, high take off



ST ELEVATION in BER

Greatest in precordial leads (V2-V5)

Usually < 2mm

Minimal in limb leads

Usually < 0.5mm

ST MORPHOLOGY in BER

Upward concavity of initial ST segment

Notching or slurring of terminal QRS

T WAVES in BER

Symmetric, concordant, large

J point: junction of QRS and ST segment; often notched; best seen in V4-5

Often notching of downstroke of QRS

R waves: tall in L precordial leads; R shift of transition zone

IS IT ISCHAMEIA?

T waves should not be biphasic/invert/change over time

Q waves should not evolve/develop

NO RECIPROCAL CHANGES!

Athlete's heart

Sinus brady, sinus arrhythmia, maybe pauses up to 2s; 1st or 2nd deg (mobitz I) HB; incr R + S wave size; minimal ST elevation at J point (concave); peaked T waves (although may be biphasic or even inverted); deep Q waves in inferior leads; displaced apex beat; ESM; incomplete RBBB, BER

CXR: prominent pul vasculature due to incr CO

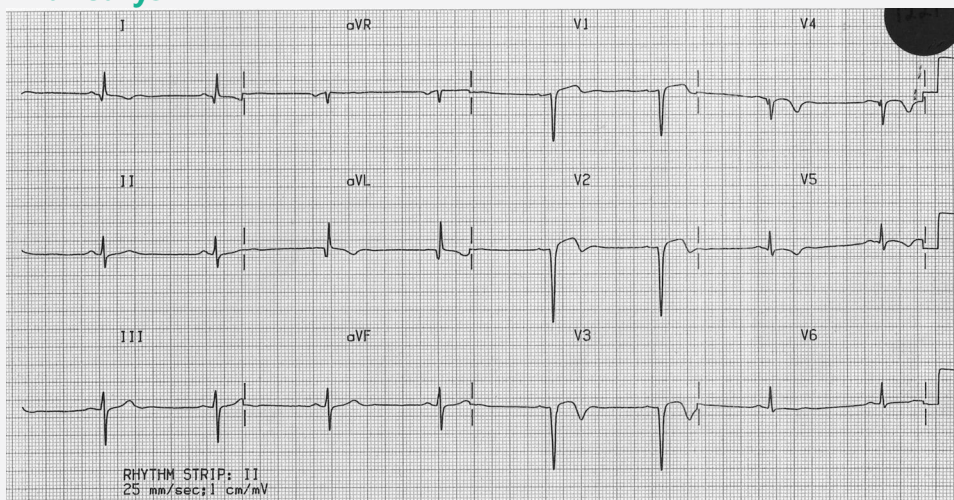
Echo: uniform hypertrophy and normal MV

DD: HOCM, LVH, MI, BER, LV aneurysm, pericarditis

LVH: S in V1 + R in V5 > 35 mm

RVH: R in V1 + S in V5 > 10 mm

LV aneurysm



Persistent STE (concave / convex) > 2/52 following AMI, most commonly in precordial leads; usually assoc with Q waves; small amplitude T waves in comparison to QRS

No reciprocal ST depression (unlike in STEMI)

RV strain: deep TWI V1-3