# ELECTROCARDIOGRAPHY

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## I. Name/Date

Always check to make sure this is the right ECG performed at the right time.
It is helpful to know what the patient's symptoms were at the time the ECG was done.
Never trust the computer's reading of the ECG (but it's pretty good for intervals, axis, etc.)

## II. Rate

Popular method: 300 / RR What if RR is variable?
More accurate method: QRS x 6 Assuming ECG runs at 25 mm/sec (standard)

## III. Rhythm

**Usually …**

| Rhythm | P waves | R-R | QRS width | Rate |
|---|---|---|---|---|
| Normal sinus | Normal | Regular | Narrow | 60-100 |
| Atrial | Abnormal | Irregular/regular | Narrow | 60-100 |
| Junctional | Negative/No/Late | Regular | Narrow | 40-60 |
| Ventricular | Dissociated | Regular | Wide | 30-40 |

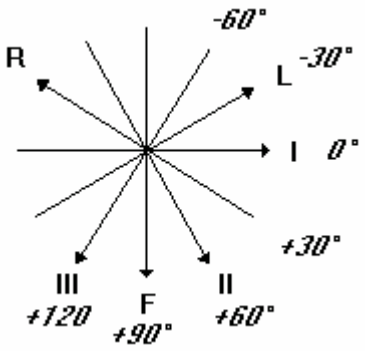
But there are many exceptions, including:

**Sinus arrhythmia**: PP interval varies by > 10% or > 0.16 s
**Sinus bradycardia**: HR < 60. Causes: vagal tone, IMI, drugs, hypothyroidism, hypothermia, hyperkalemia, sick sinus syndrome, increased intracranial pressure, PE, obstructive jaundice
**Sinus tachycardia**: HR > 100. Causes: pain, caffeine, sympathetic tone, anxiety, pheochromocytoma, hypotension, volume depletion, lots of others
**Wandering atrial pacemaker**: HR 60-100 and ≥ 3 P morphologies. Variable PP and PR.
**Multifocal atrial tachycardia**: HR > 100 and ≥ 3 P morphologies. Variable PP and PR.
Causes: pulmonary disease (COPD, PE, edema, pneumonia), aminophylline, hypoxia, MI, CHF, sepsis
**Paroxysmal atrial tachycardia**: HR 160-220, usually due to 1 focus
**Atrial flutter**: AR 240-350, inverted F waves in II/III/aVF without isoelectric baseline, small positive F waves in V1 usually with isoelectric baseline, AV block (2:1, 4:1) often present
**Atrial fibrillation**: P > 350/s, fibrillation most visible in II/III/aVF/V1/V2, irregularly irregular RR
**Causes of fib/flutter (PHART)**: pulmonary (PE, COPD), pheochromocytoma, hypertension, hypoxia, alcohol, acute MI, ASD, rheumatic/mitral valve disease, thyrotoxicosis
**Junctional tachycardia** may be due to junctional focus, AV nodal reentrant tachycardia, or AV reentrant tachycardia (including WPW and concealed bypass tracts)
Causes: digitalis (often with atrial flutter and complete heart block), IMI, myocarditis, congenital
**Accelerated idioventricular rhythm**: HR 40-100. **Not** always associated with poor prognosis (unlike VT).
Causes: myocardial ischemia, reperfusion, digitalis, normal
**Ventricular tachycardia**: HR > 100, AV dissociation + capture/fusion, concordance, LAD
Causes: myocardial disease, hypo/hyperkalemia, hypoxia, acidemia, drugs, MV prolapsed

## IV. Axis

P axis: normal 0 to 75 RAE ≥ 70
QRS axis: normal -30 to 105

| Axis | Lead I | Lead II |
|---|---|---|
| Normal | + | + |
| Left | + | - |
| Right | - | + |
| Indetermined | - | - |




Causes of LAD: LAFB, IMI, LBBB, LVH, primum ASD, COPD, hyperkalemia
Causes of RAD: RVH, COPD, PE, LPFB, lateral MI, secundum ASD, dextrocardia, lead reversal

## V. P Wave

Think of the P wave as a summation wave of RA and LA depolarization. Remember, the SA node depolarizes the RA first.
**RAE** > 2.5 mm tall in II, III, and aVF ("P-pulmonale"), or > 1.5 mm tall in V1 or V2
Causes: COPD, pulmonary HTN, PE, congenital, or normal variant (thin)
**LAE** Terminal negative P ≥ 1 mm deep and ≥ 0.04 s wide in V1, or
P ≥ 0.12 s wide and notched in II, III, or aVF ("P mitrale")
Causes: mitral/aortic valve disease, MI, LVH, heart failure
Interatrial block (P ≥ 110 msec) may increase the risk of embolic stroke. *Am J Card 2005;95:667-8*

**VI. PR Segment** Normal 0.12-0.20 s

**First degree AV block**: PR ≥ 0.20 s, PR constant, and P:QRS 1:1
Causes: high vagal tone (athletes), drugs, normal, congenital heart disease, myocarditis

**Second degree AV block, Type I (Wenckebach)** = progressive lengthening of PR interval and shortening of RR interval until a P wave fails to conduct. RR interval with non-conducted P is shorter than 2 PP intervals. Produces grouped beating and block usually occurs at the AV node level (i.e. narrow QRS).
Causes: normal (athletes), inferior MI, drugs, myocarditis

**Second degree AV block, Type II** = constant PR but intermittent non-conducted P wave. RR interval with non-conducted P is equal to 2 PP intervals. Block usually occurs below the AV node (wide QRS in 80%).
Causes: almost always due to myocardial damage or fibrosis, anterior MI

**Third degree AV block** = independent atrial and ventricular rhythms. Complete heart block is present when atrial rate > ventricular rate, whereas AV dissociation is present if ventricular rate > atrial rate.
Causes: MI, degenerative conduction defect (Lev's/Lenegre's), infiltration (sarcoid, amyloid), digitalis, endocarditis (usually preceded by prolonged PR), severe hyperkalemia, Lyme disease

**Short PR**
Causes: accelerated AVC, pre-excitation with delta wave due to accessory tract (WPW)

**PR depression** may be pathological (e.g. pericarditis) especially if > 0.8 mm depression

**VII. QRS Segment**

**Q waves** (any in V1-V3 or > 0.03 s in I, II, aVL, aVF, V4-V6)

| | |
|---|---|
| Anterolateral | V4-V6 |
| Anterior | V2-V4 |
| Anteroseptal | V1-V3/4 |
| Lateral | I, aVL |
| Inferior | II, III, aVF |

**LVH voltage criteria** – any of the following:

| | |
|---|---|
| R aVL + S V3(Cornell criteria, most accurate) | > 28 mm (males) or 20 mm (females) |
| R V5/V6 + S V1 | > 35 mm if age > 40 (>40 if age 30-40) |
| R V1-6 + S V1-6 | > 45 mm |
| R V5 | > 26 mm |
| R V6 | > 20 mm |
| R I + S II | ≥ 26 mm |
| R I | ≥ 14 mm |
| S aVR | ≥ 15 mm |
| R aVL (specific unless LAFB present) | ≥ 12 mm |
| R aVF | ≥ 21 mm |

Sensitivity decreased by low voltage (COPD, PTX, effusion, obesity), CAD, sarcoidosis, RVH, LBBB
Specificity decreased by high voltage (thin body, left mastectomy), LBBB, WPW, LAFB

**RVH voltage criteria** (less reliable) – RAD > $110^{o}$ and any of the following:

| | |
|---|---|
| R/S ratio in V1 or V3R | > 1 |
| R/S ratio in V5 or V6 | ≤ 1 |
| R V1 | ≥ 7 mm |
| R V1 + S V5-6 | > 10.5 mm |
| rSR' V1 present with R' being qR present in V1 | > 10 mm |

**QRS width**: normal is < 0.10 s.

**Bundle branch block**. *Look at the latter half of QRS in V1 and I/V5/V6 for morphology. Incomplete if QRS 0.10-0.119 s. Complete if QRS ≥ 0.12 s.*

| | **LBBB** | **RBBB** |
|---|---|---|
| QRS morphology in V1 | rS or QS (negative) | rSR' (positive) |
| *QRS morphology in I/V5/V6* | *Broad monophasic R (positive)* | *Wide, slurred S (negative)* |
| Interferes with diagnosis of LVH/MI? | Yes | No |
| Causes | MI, LVH, degenerative conduction defect, congenital heart disease | pulmonary disease, normal, HTN, myocarditis, cardiomyopathy |

**Fascicular block**: typically associated with normal to slightly long QRS width (< 0.12 s)

| | **LAFB** | **LPFB** |
|---|---|---|
| Axis | -45 to -90 | 100 to 180 |
| I, aVL | qR | S |
| II, III, aVF | R or rS | Q |
| Exclude other causes of abnormal axis | LAD: LVH, IMI, COPD, LBBB, primum ASD, severe hyperkalemia | RAD: RVH, COPD, PE, IMI, WPW, dextrocardia, lead reversal |
| Other features | Increases voltage in I, aVL. PRWP is common Can mask presence of IMI. | Most common cause is CAD. If LPFB develops in acute MI, it suggests multivessel disease with poor prognosis |

If 0.10-0.12 s without LVH, LBBB, or RBBB morphology, **interventricular conduction delay (IVCD)** is present. Incidentally discovered BBB is associated with higher mortality (10% over 20 years). Mayo Clin Proc 2005;80:1585
Features of AV Conduction Disturbances Complicating AMI (Wellens JJ, Conover MB 1992)

| | **Inferior MI** | **Anterior MI** |
|---|---|---|
| Site of block | AV node | Bundle branches |
| Artery involved | RCA | LAD |
| Escape rhythm | Narrow QRS<br>Rate 40-60 bpm<br>Dependable | Wide QRS<br>Rate < 40 bpm<br>Undependable |
| Duration of block | Transient | Transient |
| Increase in hospital mortality c/w same infarction location w/o block | 2 ½ times | 4 times |

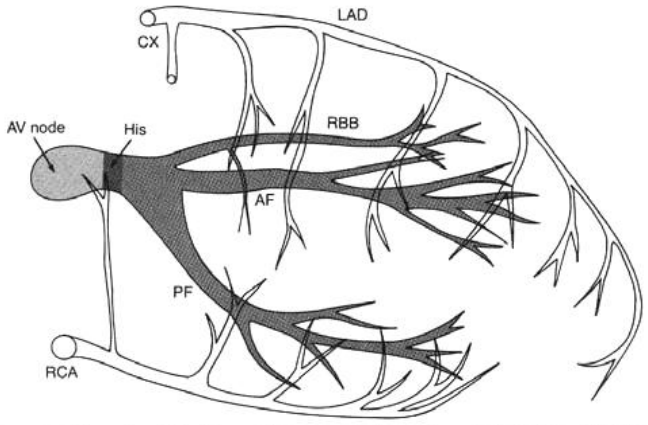



**Figure 1–9.** The trifascicular intraventricular conduction system. Note that the right bundle branch (RBB) and the superior division of the left bundle branch (AF) are both anterior structures and therefore are vulnerable in anteroseptal myocardial infarction. The posterior division of the left bundle branch (PF) is broad and supplied by both the left anterior descending (LAD) and the right coronary artery (RCA).

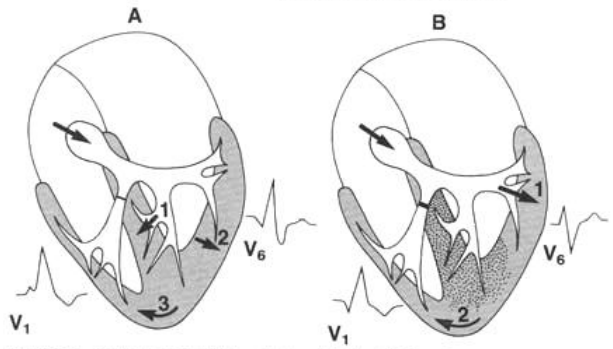



**Figure 1–12.** Mechanism of right bundle branch block. *(A)* Note that the right ventricle is activated last and without any opposing forces, resulting in the late R' in lead $V_1$ and the S wave in lead $V_6$. In anteroseptal infarction *(B)* a QR pattern develops in lead $V_1$. Loss of anterior wall tissue causes an R/S pattern in lead $V_6$.

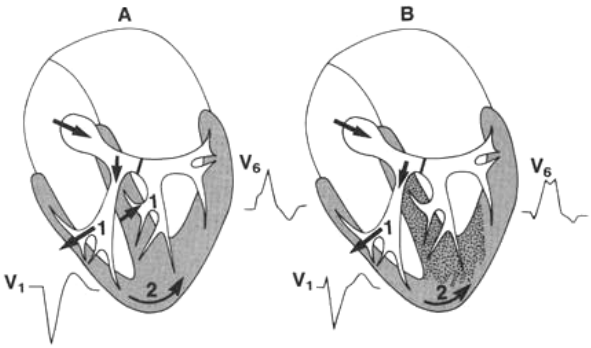



**Figure 1–15.** Mechanism of left bundle branch block. *(A)* Ventricular activation goes from right to left to produce a negative QRS complex in lead $V_1$ and a positive complex in lead $V_6$. In anteroseptal infarction *(B)* early unopposed right ventricular activation produces the initial R in lead $V_1$ and a Q in lead $V_6$.

**VIII.** **ST Segment** (NEJM 2003; 349:2128)

**Normal** < 0.5 mm below or 1 mm above baseline in limb leads

Early repolarization pattern: upwardly concave ST elevation (usually V2-5, sometimes II/III/aVF), notched/slurred at end of R, symmetrical upright (large) T, no reciprocal ST depression, ST < 25% T in V6

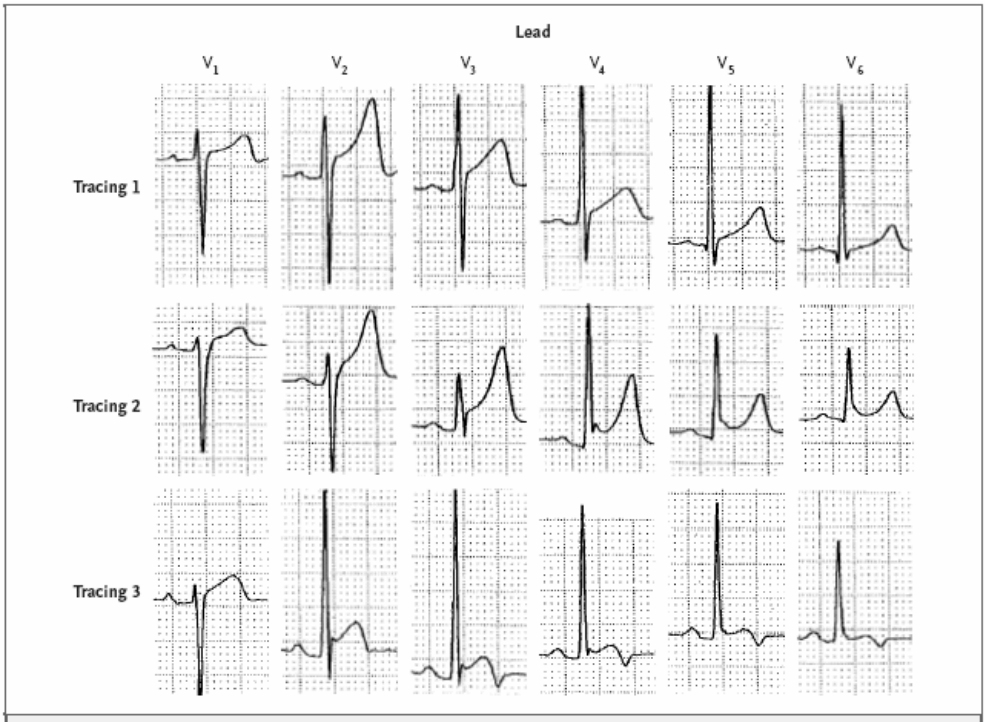



Figure 1. **Electrocardiograms Showing Normal ST-Segment Elevation and Normal Variants.**

Tracing 1 shows normal ST-segment elevation. Approximately 90 percent of healthy young men have ST-segment elevation of 1 to 3 mm in one or more precordial leads. The ST segment is concave. Tracing 2 shows the early-repolarization pattern, with a notch at the J point in $V_4$. The ST segment is concave, and the T waves are relatively tall. Tracing 3 shows a normal variant that is characterized by terminal T-wave inversion. The QT interval tends to be short, and the ST segment is coved.

**ST depression** ≥ 0.5 mm, flat or downsloping is more concerning than upsloping

Consider posterior MI if V1 or V2 shows ST depression ≥ 2 mm and R ≥ 0.04 s or R ≥ S. Check V4R.

**ST elevation** ≥ 2 mm in V1-3 or ≥ 1 mm in other leads, usually upwardly convex

Differential includes acute MI, pericarditis, ventricular aneurysm, LVH, myocarditis, hyperkalemia, BBB, CNS disease, Brugada syndrome, normal variant (≤ 3 mm in V1-3), or early repolarization.

In STEMI, degree of ST elevation and resolution correlate well with outcome. *Circ 2004;110:e506-10*

ST elevations localize, ST depressions do **not**.

| **Location** | **Leads** | **Vessels** |
|---|---|---|
| Anterior | V2-V4 | LAD |
| Anteroseptal | V1-V4 | LAD |
| Anterolateral | I, aVL, V1, V6 | LAD, Diagonal |
| Inferior | II, III, aVF | RCA, LCX |
| Lateral | I, aVL, V5-V6 | LCX, Diagonal |
| Posterior | V1-V3 (tall R) | RCA, LCX |
| RV | V4R | RCA |

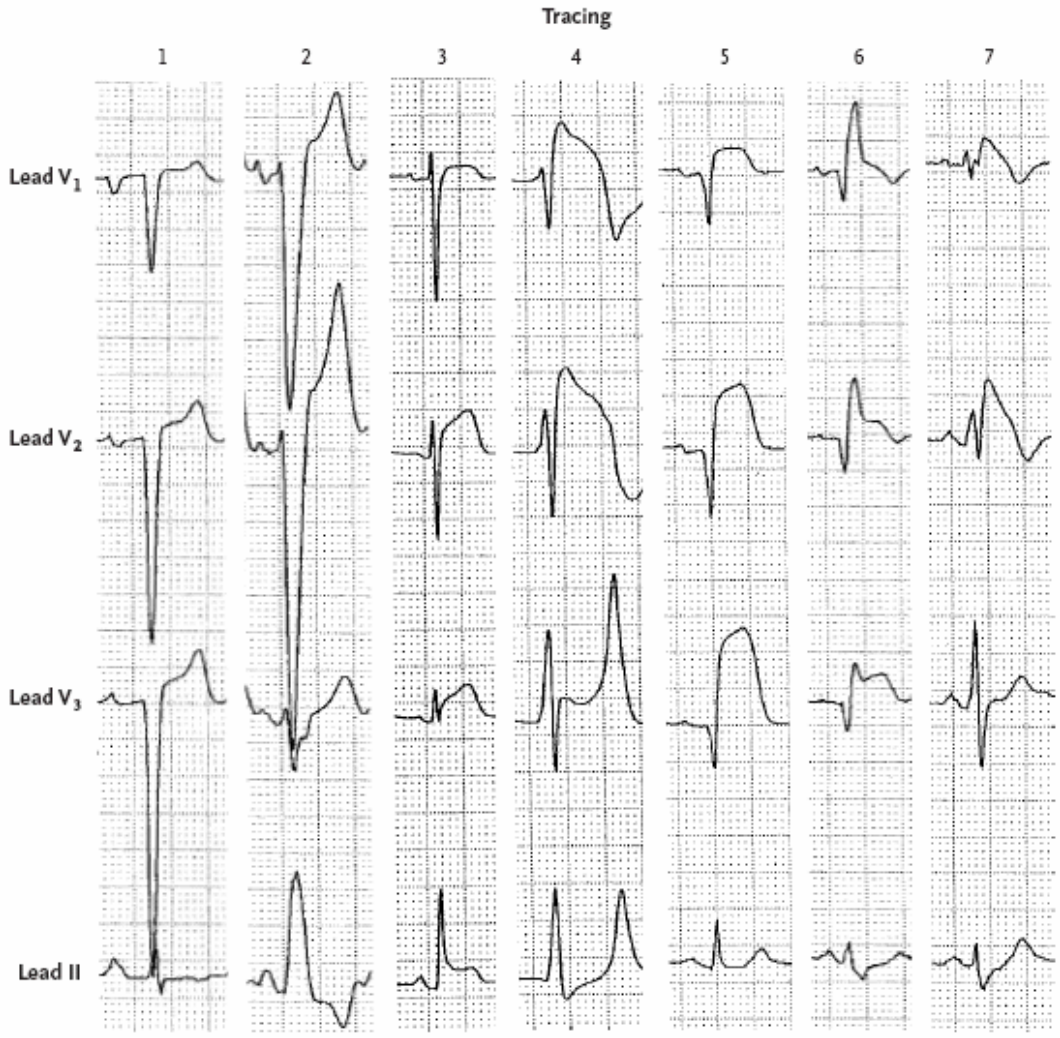



**Figure 2. Electrocardiograms Showing ST-Segment Elevation in Various Conditions.**

Tracing 1 is from a patient with left ventricular hypertrophy, and tracing 2 is from a patient with left bundle-branch block. Tracing 3, from a patient with acute pericarditis, is the only tracing with ST-segment elevation in both precordial leads and lead II and PR-segment depression. Tracing 4 shows a pseudoinfarction pattern in a patient with hyperkalemia. The T wave in $V_3$ is tall, narrow, pointed, and tented. Tracing 5 is from a patient with acute anteroseptal infarction. The distinctive features of tracing 6, from a patient with acute anteroseptal infarction and right bundle-branch block, include the remaining R' wave and the distinct transition between the downstroke of R' and the beginning of the ST segment. Tracing 7, from a patient with the Brugada syndrome, shows rSR' and ST-segment elevation limited to $V_1$ and $V_2$. The ST segment begins from the top of the R' and is downsloping.

**IX.** **T Wave**

Normal Upright I, II, V3-6
Inverted aVR, V1
Tall T waves Acute MI, hyperkalemia, LVH, anemia, CNS disorder, or normal
Ischemic T waves Biphasic T in V1-4 without symptoms suggests proximal LAD lesion
Deep symmetrically inverted T in V1-6 suggests LAD lesion (Wellens)

**X.** **QTC** (= QT divided by the square root of RR)

Normal QTC = 0.30 to 0.44 (in general, less than ½ between RR interval)
Long QT: hypocalcemia, hypomagnesemia, hypothermia, drugs, CVA, hypothyroidism, bradycardia
Hypocalcemia causes prolonged ST segment with normal T
Short QT: hypercalcemia, hyperkalemia, digitalis, acidosis, hyperthyroidism, hyperthermia

## XI. Wide Complex Tachycardia

First, look at the rhythm. Is it regular?

If regular.

| V1 | Favors VT | Favors SVT |
|---|---|---|
| **Positive** | V1 mono/biphasic | V1 triphasic (rSR') |
| | V6 deep S (R/S ratio < 1) | V6 triphasic (qRS), R/S > 1 |
| **Negative** | V1-2 R > 0.03 sec | V1-2 R narrow |
| | V1-2 downstroke notched | V1-2 downstroke quick, smooth |
| | V1-2 S nadir > 0.06 sec | V1-2 S nadir ≤ 0.06 sec |
| | V6 any Q wave | V6 no Q wave |

Other things that favor VT:

- QRS ≥ 0.14 sec (especially in V1-positive tachycardia)
- Abnormal axis (especially -90 to -180)
- AV dissociation
- Capture/fusion beats (conducted sinus impulse resulting in a narrower QRS beat)
- Concordant precordial pattern (especially negative concordance in V1-6)

Brugada criteria for VT (any one the following is suggestive of VT):

- Absence of RS in prechordial leads
- If RS present, R to nadir of S > 100 ms
- AV dissociation or fusion beat
- Morphologic features:
  - RBBB pattern: Monophasic R or qS/qR in V1; R/S < 1 or qR/qS in V6
  - LBBB pattern: Notched S > 60 ms or R > 30 ms in V1; qR/qS in V6

## XII. Miscellaneous Stuff

Reversal of leads

- Left arm and right arm
  - Mimics dextrocardia in limb leads (inverted P-QRS-T in I and aVL)
  - Leads II and III transposed
  - Leads aVR and aVL transposed
- Left arm and left leg
  - Leads I and II transposed
  - Leads aVF and aVL transposed
  - Lead III inverted
- Right arm and left leg
  - Leads I, II, and III inverted
  - Leads aVR and aVF transposed

Tremor artifacts

- Physiologic tremor (e.g. shivering) = 500 per minute
- Parkinson's tremor = 300 per minute

## References


O'Keefe JH, Hammill SC, Freed MS, Pogwizd SM. The complete guide to ECGs, 2nd ed. (Physicians' Press: Royal Oak, MI 2002)

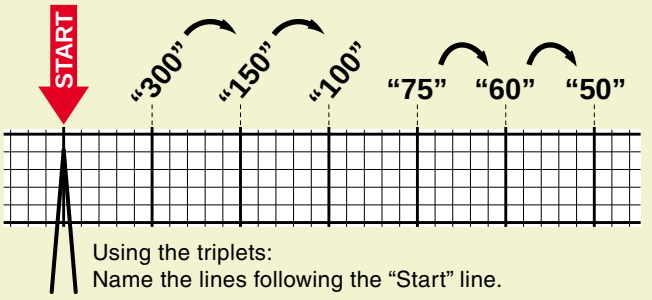
Surawicz B, Knilans TK. Chou's electrocardiography in clinical practice, 5th ed. (Saunders: Philadelphia, PA 2001)

Wang K et al. ST-segment elevation in conditions other than acute myocardial infarction. NEJM 2003; 349:2128

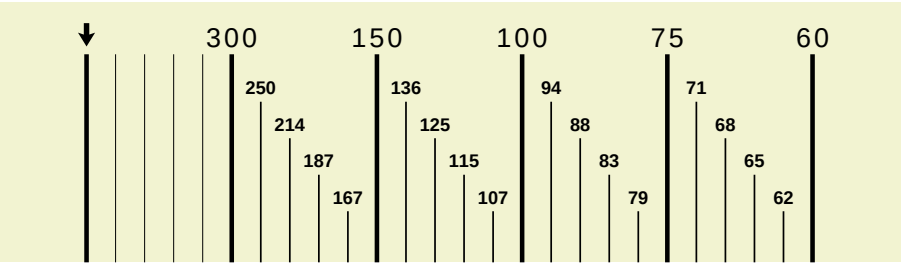
Wellens JJ, Conover MB. The ECG in emergency decision making (W.B. Saunders 1992)

# Rate

## Determine Rate by Observation




## Fine division/rate association: reference




May be calculated: $\frac{1500}{\text{mm. between similar waves}}$ = RATE

## Bradycardia (slow rates)

- Cycles/6 second strip X 10 = Rate
- When there are 10 large squares between similar waves, the rate is 30/minute.

## *Sinus Rhythm:* origin is the SA Node ("Sinus Node"), normal sinus rate is 60 to 100/minute.

- Rate more than 100/min. = *Sinus Tachycardia*
- Rate less than 60/min. = *Sinus Bradycardia*

## Determine any co-existing, independent (atrial/ventricular) rates:

- Dissociated Rhythms:
  A Sinus Rhythm (or atrial rhythms) may co-exist with an independent rhythm from an automaticity focus of a lower level. Determine rate of each.

## Irregular Rhythms:

- With Irregular Rhythms (such as Atrial Fibrillation) always note the general (average) ventricular rate (QRS's per 6-sec. strip X 10) or take the patient's pulse.

# Rhythm

★ **Identify basic rhythm…**

…then scan entire tracing for pauses, premature beats, irregularity, and abnormal waves.

★ **Always:**

- Check for: P before each QRS.
  QRS after each P.
- Check: PR intervals (for AV Blocks).
  QRS interval (for BBB).
- Has QRS vector shifted outside normal range? (to rule out Hemiblock).

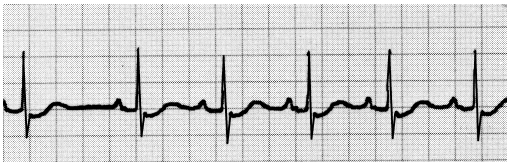
## Irregular Rhythms

### *Sinus Arrhythmia*

Irregular rhythm that varies with respiration.

All P waves are identical.

Considered normal.



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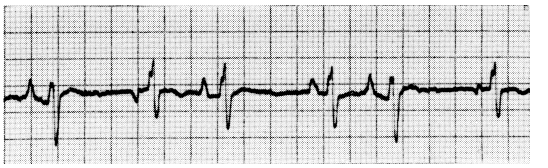
### *Wandering Pacemaker*

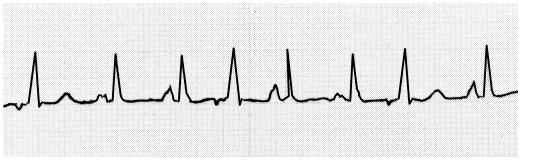
Irregular rhythm. P waves change shape as pacemaker location varies.

Rate under 100/minute…



…but if the rate exceeds 100/minute, then it is called

### *Multifocal Atrial Tachycardia*



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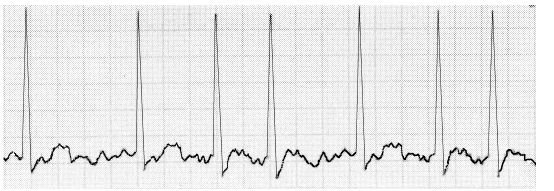
### *Atrial Fibrillation*

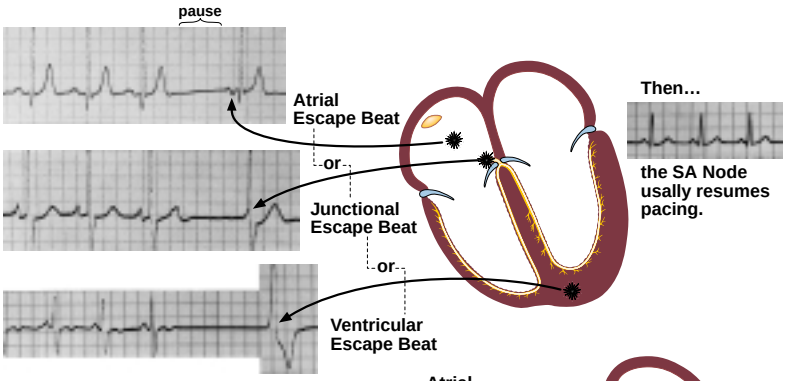
Irregular ventricular rhythm. Erratic atrial spikes (no P waves) from multiple atrial automaticity foci. Atrial discharges may be difficult to see.



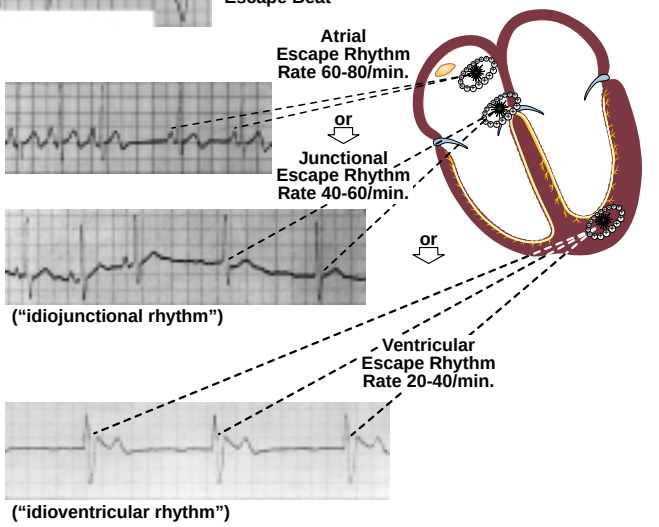
# Rhythm

## Escape – the heart's response to a pause in pacing

- An unhealty Sinus (SA) Node may fail to emit a pacing stimulus ("Sinus Block"); this pause may evoke an escape beat from an automaticity focus.

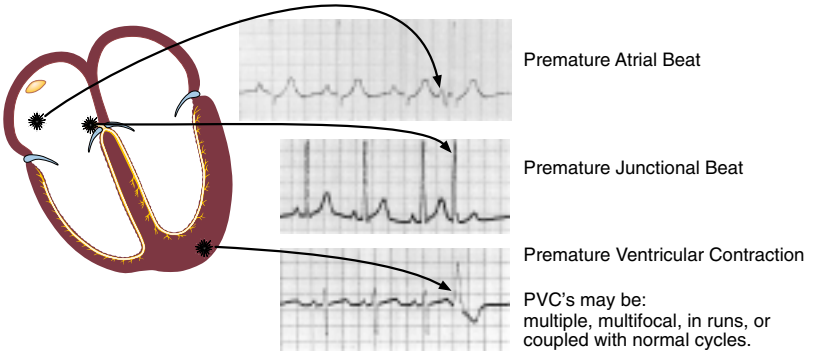



- But a sick Sinus (SA) Node may cease pacing ("Sinus Arrest"), causing an automaticity focus to "escape" to assume pacemaker status.



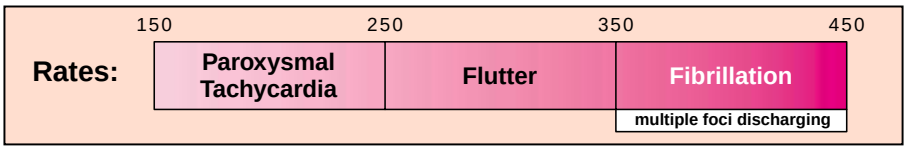

## Premature Beats – from an irritable automaticity focus

- An irritable automaticity focus may suddenly discharge, producing a:



# Rhythm continued)

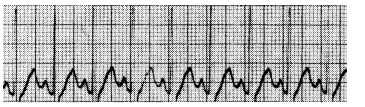
## Tachyarrhythmias "focus" = automaticity focus



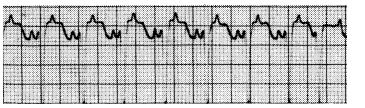

### Paroxysmal (sudden) Tachycardia …rate: 150-250/min.

"Supraventricular Tachycardia"

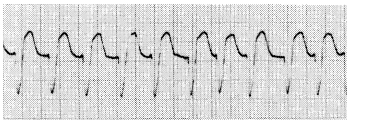
***Paroxysmal Atrial Tachycardia***
An irritable atrial focus discharging at 150-250/min. produces a normal wave sequence, if P' waves are visible.



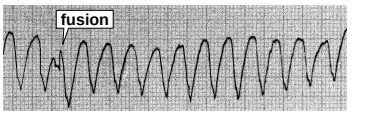
***• P.A.T. with block***
Same as P.A.T. but only every second (or more) P' wave produces a QRS.



***Paroxysmal Junctional Tachycardia***
AV Junctional focus produces a rapid sequence of QRS-T cycles at 150-250/min. QRS may be slightly widened.

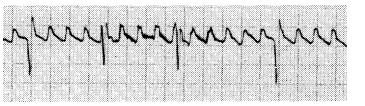


***Paroxysmal Ventricular Tachycardia***
Ventricular focus produces a rapid (150-250/min.) sequence of (PVC-like) wide ventricular complexes.

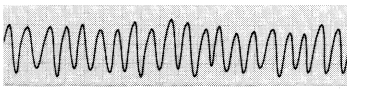



### Flutter …rate: 250-350/min.

***Atrial Flutter***
A continuous ("saw tooth") rapid sequence of atrial complexes from a single rapid-firing atrial focus. Many flutter waves needed to produce a ventricular response.

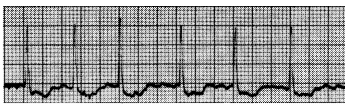


***Ventricular Flutter*** **also see "Torsades de Pointes"**
A rapid series of smooth sine waves from a single rapid-firing ventricular focus; usually in a short burst leading to Ventricular Fibrillation.

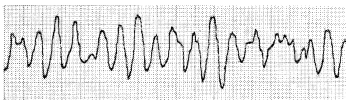


### Fibrillation …erratic (multifocal) rapid discharges at 350 to 450/min.

***Atrial Fibrillation***
Multiple atrial foci rapidly discharging produce a jagged baseline of tiny spikes. Ventricular (QRS) response is irregular.



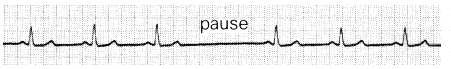
***Ventricular Fibrillation***
Multiple ventricular foci rapidly discharging produce a totally erratic ventricular rhythm without identifiable waves. Needs **immediate** treatment.



# Rhythm: (“heart”) blocks

## Sinus (SA) Block

An unhealthy Sinus (SA) Node misses one or more cycles (sinus pause)…




the Sinus Node usually resumes pacing, but the pause may evoke an “escape” response from an automaticity focus.

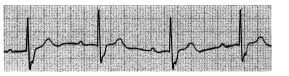
## AV Block

Blocks that delay or prevent atrial impulses from reaching the ventricles.

★ **Always Check:**
- PR intervals less than one large square?
- Is every P wave followed by a QRS?

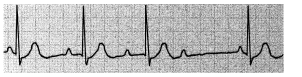
### *1° AV Block* …prolonged PR interval

PR interval is prolonged to greater than .2 sec (one large square).

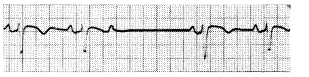


### *2° AV Block* … some P waves without QRS response

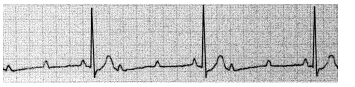
**Wenckebach** …PR gradually lengthens with each cycle until the last P wave in the series does not produce a QRS.



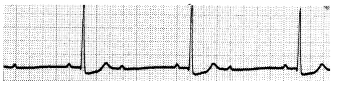
**Mobitz** …some P waves don’t produce a QRS response. If “intermittent,” an occasional QRS is droped.



More advanced Mobitz block may produce a 3:1 (AV) pattern or even higher AV ratio

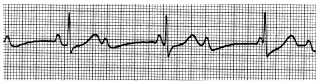


**2:1 AV Block** …may be Mobitz or Wenckebach. PR length and QRS width or vagal maneuvers help differentiate.

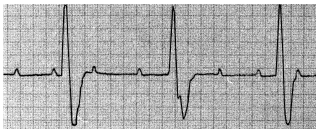


### *3° (“complete”) AV Block* …no P wave produces a QRS response

3° Block: P waves—SA Node origin. QRS’s—if narrow, and if the ventricular rate is 40 to 60 per min., then origin is a Junctional focus.



3° Block: P waves—SA Node origin. QRS’s—if PVC-like, and if the ventricular rate is 20 to 40 per min., then origin is a Ventricular focus.



## Bundle Branch Block …find R,R' in right or left chest leads

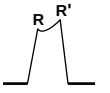
★ **Always Check:**
- is QRS within 3 tiny squares?

### Right BBB


**QRS in $V_1$ or $V_2$**

With Bundle Branch Block the criteria for ventricular hypertrophy are unreliable.

### Left BBB




**QRS in $V_5$ or $V_6$**

*Caution:* With Left BBB infarction is difficult to determine on EKG.

## Hemiblock …block of Anterior or Posterior fascicle of the Left Bundle Branch.

★ **Always Check:**
- has Axis shifted outside Normal range?

### Anterior Hemiblock

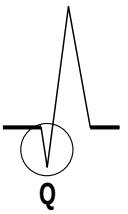
Axis shifts Leftward → L.A.D.
look for $Q_1S_3$

### Posterior Hemiblock

Axis shifts Rightward → R.A.D.
look for $S_1Q_3$

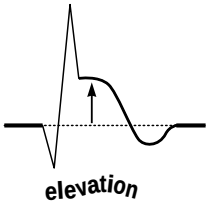
# Infarction

**Q wave = Necrosis (significant Q's only)**



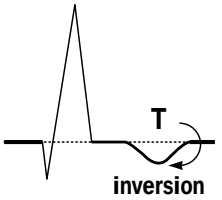

- Significant Q wave is one millimeter (one small square) wide, which is .04 sec. in duration…
  … or is a Q wave 1/3 the amplitude (or more) of the QRS complex.
- Note those leads (omit AVR) where significant Q's are present … see next page to determine infarct location, and to identify the coronary vessel involved.
- Old infarcts: significant Q waves (like infarct damage) remain for a lifetime. To determine if an infarct is acute, see below.

**ST (segment) elevation = (acute) Injury (also Depression)**




- Signifies an acute process, ST segment returns to baseline with time.
- ST elevation associated with significant Q waves indicates an acute (or recent) infarct.
- A tiny "non-Q wave infarction" appears as significant ST segment elevation without associated Q's. Locate by identifying leads in which ST elevation occurs (next page).
- ST depression (persistent) may represent "subendocardial infarction," which involves a small, shallow area just beneath the endocardium lining the left ventricle. This is also a variety of "non-Q wave infarction." Locate in the same manner as for infarction location (next page).

**T wave inversion = Ischemia**

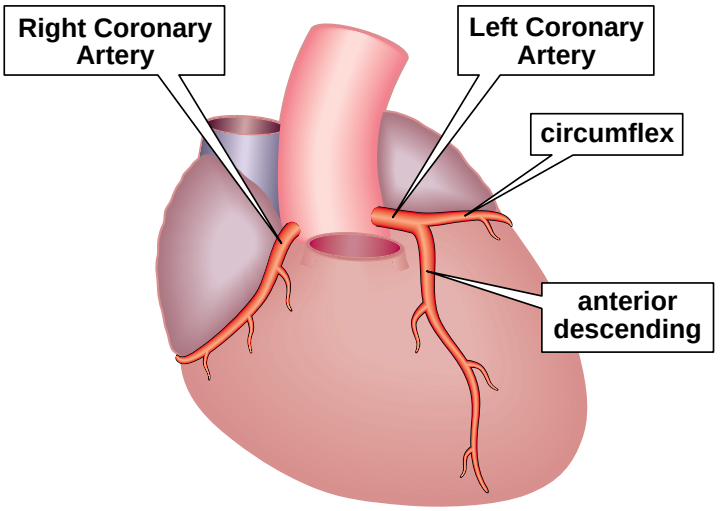



- Inverted T wave (of ischemia) is symmetrical (left half and right half are mirror images). Normally T wave is upright when QRS is upright, and vice versa.
- Usually in the same leads that demonstrate signs of acute infarction (Q waves and ST elevation).
- Isolated (non-infarction) ischemia may also be located; note those leads where T wave inversion occurs, then identify which coronary vessel is narrowed (next page).

NOTE: Always obtain patient's previous EKG's for comparison!

# Infarction Location — and — Coronary Vessel Involvement

## Coronary Artery Anatomy




## Infarction Location/Coronary Vessel Involvement

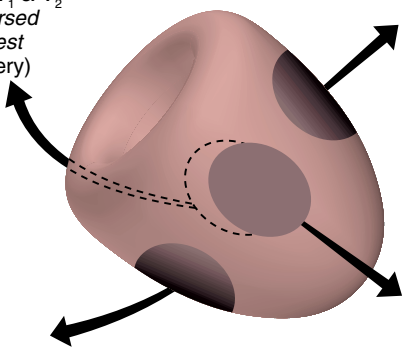
***Posterior***

- large R with ST depression in $V_1$ & $V_2$
- *mirror test* or *reversed transillumination test*

(Right Coronary Artery)

***Lateral***

Q's in lateral leads I and AVL
(Circumflex Coronary Artery)



***Inferior***

(diaphragmatic)
Q's in inferior leads II, III, and AVF
(R. or L. Coronary Artery)

***Anterior***

Q's in $V_1$, $V_2$, $V_3$, and $V_4$
(Anterior Descending Coronary Artery)

# Miscellaneous

## Pulmonary Embolism

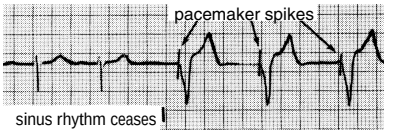
- $S_1Q_3L_3$ – wide S in I, large Q and inverted T in III.
- acute Right BBB (transient, often incomplete)
- R.A.D. and clockwise rotation
- inverted T waves $V_1 \rightarrow V_4$ and ST depression in II.

## Artificial Pacemakers

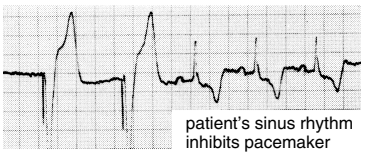
Modern artificial pacemakers have sensing capabilities and also provide a regular pacing stimulus. This electrical stimulus records on EKG as a tiny vertical spike that appears just before the "captured" cardiac response.

***Demand Pacemakers:*** (page 301)

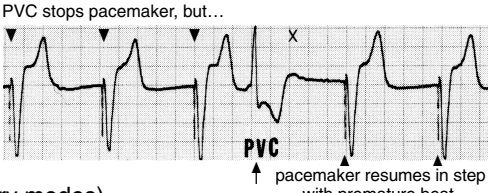
- are "triggered" (activated) when the patient's own rhythm ceases or slows markedly.



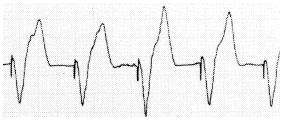

- are "inhibited" (cease pacing) if the patient's own rhythm resumes at a reasonable rate.



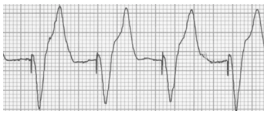

- will "reset" pacing (at same rate) to synchronize with a premature beat.



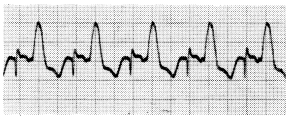

## Pacemaker Impulse (delivery modes)



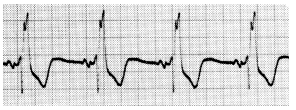
Ventricular Pacemaker
(electrode in Right Ventricle)



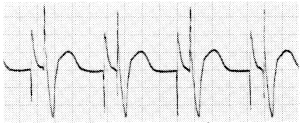
(Asynchronous) Epicardial Pacemaker
Ventricular impulse not linked to atrial activity.



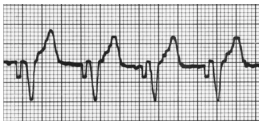
Atrial pacemaker



Atrial Synchronous Pacemaker
P wave sensed, then after a brief delay,
ventricular impulse is delivered.



Dual Chamber (AV sequential) Pacemaker



External Non-invasive Pacemaker

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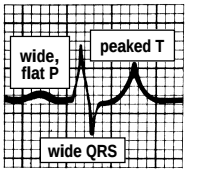
# Miscellaneous continued

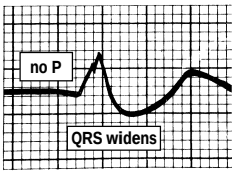
## Electrolytes

### *Potassium*

Increased $K^+$
(hyperkalemia)

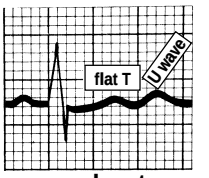


moderate

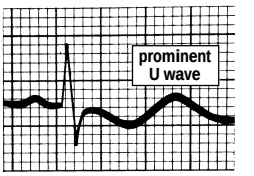


extreme

Decreased $K^+$
(hypokalemia)



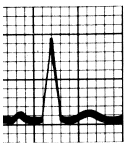
moderate



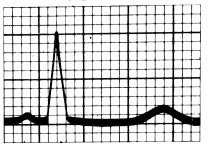
extreme

### *Calcium*

Hyper $Ca^{++}$


short QT

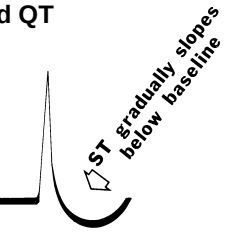
Hypo $Ca^{++}$


prolonged QT

## Digitalis

EKG appearance with digitalis ("digitalis effect")

- remember Salvador Dali.
- T waves depressed or inverted.
- QT interval shortened.




Digitalis Excess ⟶ Digitalis Toxicity

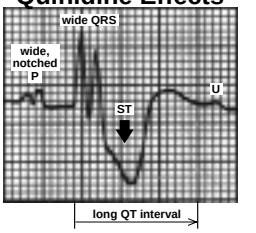
(blocks)
- SA Block
- P.A.T. with Block
- AV Blocks
- AV Dissociation

(irritable foci firing rapidly)
- Atrial Fibrillation
- Junctional or Ventricular Tachycardia
- multiple P.V.C.'s
- Ventricular Fibrillation

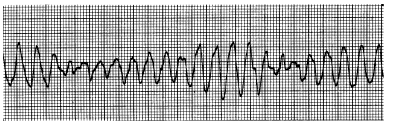
## Quinidine

- EKG appearance with quinidine (page 320)

Quinidine Effects




- Excess quinidine or other medications that block potassium channels (or even low serum potassium) may initiate… (page 158)


Torsades de Pointes