PCP Autonomous IV Program

Module I

E.C.G. RHYTHM INTERPRETATION

2014 OBHG Education Subcommittee

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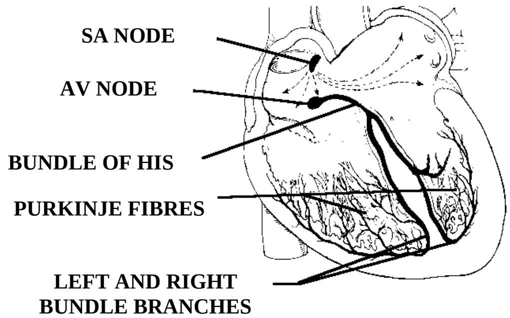
Section I – Electrophysiology

Cardiac Conduction System

Anatomy and Physiology

The cardiac conduction system is comprised of specialized tissues that unlike muscle tissue located elsewhere in the body can 1: generate electrical impulses (automaticity) and 2: conduct electrical impulses (conductivity). The cardiac muscle tissue is similar to other tissue in its ability to contract (contractility).

Specialized tissue located in the right atria known as the sinoatria node (SA) is the primary pacemaker of the heart producing electrical impulses at a rate of 60 to 100 per minute in the normal resting heart. The generation of the impulses results from the movement of electrolytes (Na^+, Ca^{++} and K^+) across the cell membrane leading to the depolarization of the cell. Other tissue similar in nature to the SA node, the atrioventricular node (AV) is capable of producing the same impulses but at a slower rate, 40 to 60 per minute. Tissues located in the ventricles (bundle branches and purkinje fibers) can also produce impulses at rates of 20 to 40 per minute.



In addition to the ability to automatically generate electrical impulses, the cardiac conduction system allows for the transmission of the impulses throughout the heart in a coordinated manner, producing the rhythmic contraction associated with normal cardiac output.

Impulses generated by the SA node travel along specialized tissue known as the conduction pathways. The impulse travels to the AV node where it is delayed, allowing for the contraction of the atria. Once the impulse passes through the AV node it transcends the Bundle of HIS, the Right and Left Bundle Branches and finally the Purkinje Fibres.

Conduction Times & Velocities

Conduction times and velocities are important in that they co-ordinate the electrical system of the heart with its mechanical function of a pump. The diagram below shows that the movement of the impulses through the atria (right and left) is significantly slower that the movement through the ventricles. The larger mass of the ventricle requires the rapid flow of the electrical impulses resulting in the complete depolarization and contraction of the entire ventricle at almost the same time. This action results in the forceful contracting of the heart producing cardiac output.

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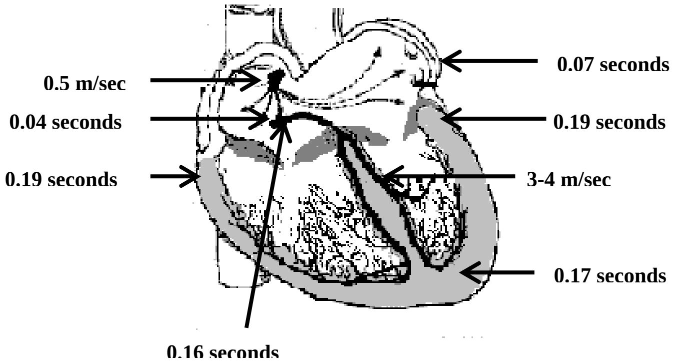
Section I – Electrophysiology

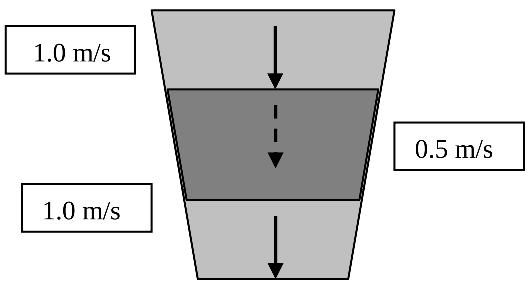
Cardiac Conduction System (cont'd)

Conduction Times & Velocities (cont'd)



Role of the AV Node

The AV Node plays a significant role in delaying the conduction of electrical impulses between the atria and the ventricles. The delay provides sufficient time to allow the ventricle to fully fill with blood and stretch sufficiently prior to the impulses entering the ventricle producing the contraction. The tissue of the AV Node becomes more dense slowing the impulse then breaks through to enter the ventricle.



Because of the unique qualities of the AV nodal tissue and it's ability to naturally slow the conduction of impulses, when things go wrong, such as in the diseased or injured heart, the AV Node is responsible for the cardiac arrhythmias involving the heart blocks of the 1^{st}, 2^{nd}, or 3^{rd} degree type.

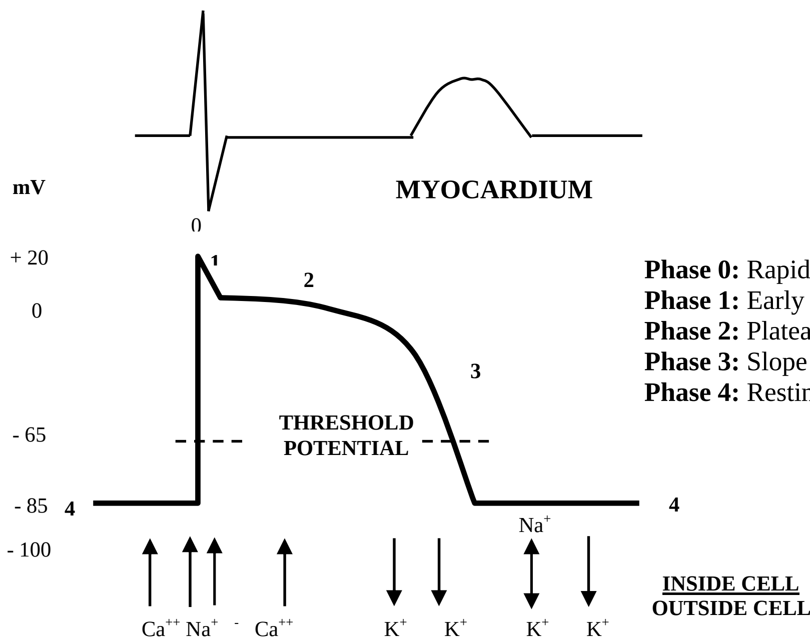
Notes:

Section I – Electrophysiology

Cardiac Conduction System (contd.)

Action Potential – Ventricular Muscle

The action potential of ventricular muscle involves the movement of anions and cations across the cell membrane of the cardiac muscle tissue. This movement results in the depolarization and subsequent repolarization of the cell with corresponding contraction of the heart muscle.



Phase 0: Rapid Depolarization
Phase 1: Early Repolarization
Phase 2: Plateau (Absolute Refractory Period)
Phase 3: Slope (Relative Refractory Period)
Phase 4: Resting Membrane Potential

Phase 0: Rapid Depolarization **–** cell is stimulated by conducted impulse causing sodium ions to enter the cell through slow channels. When polarity of cell becomes less negative (-65 mV) the fast sodium channels open and the rapid influx of positively charged sodium enters the cell bringing the polarity of the cell to + 20 mV.

Phase 1: Early Repolarization – Fast sodium channels close. Chloride channels open allowing negatively charged chloride to enter the cell, reducing the positive charge of the cell to neutral (0 mV). There is also a small efflux of potassium at this stage, hence the dip in the action potential.

Phase 2: Plateau (Absolute Refractory Period) – calcium channels remain open allowing the influx of positively charged calcium ions to enter the cell. This period is slower allowing for the full depolarization and contraction of the tissue.

Phase 3: Slope (Relative Refractory Period) – potassium ions (+ve) leave cell (efflux) reducing the intracellular polarity until it reaches – 85 mV again.

Phase 4: Resting Membrane Potential – constant efflux of potassium (K^+) keeps intracellular polarity at – 85 mV until next impulse stimulates Phase 0 sodium channel opening.

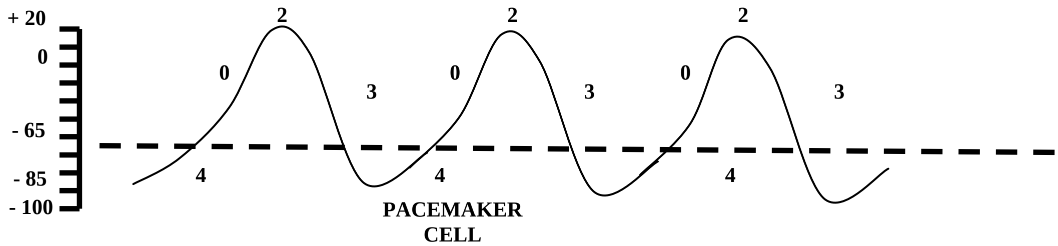
Notes:

Section I – Electrophysiology

1.0 Cardiac Conduction System (contd.)

1.4 Action Potential – Pacemaker Cell

Automaticity is the heart's ability to generate its own electrical impulses. The SA Node is the primary pacemaker of the heart depolarizing at a rate of 60 to 100 times per minute. Unlike ventricular muscle, the SA Node requires no external stimulation to cause the sodium channels to open. The cell wall membrane of the pacemaker cell is permeable to sodium allowing for the slow influx of this ion from outside to inside the cell. It is also believed that slow calcium channels are present allowing the movement of Ca^{++} inside the cell. The movement of the positively charged Na^{+} and Ca^{++} ions into the cell results in the slow depolarization of the pacemaker cell.



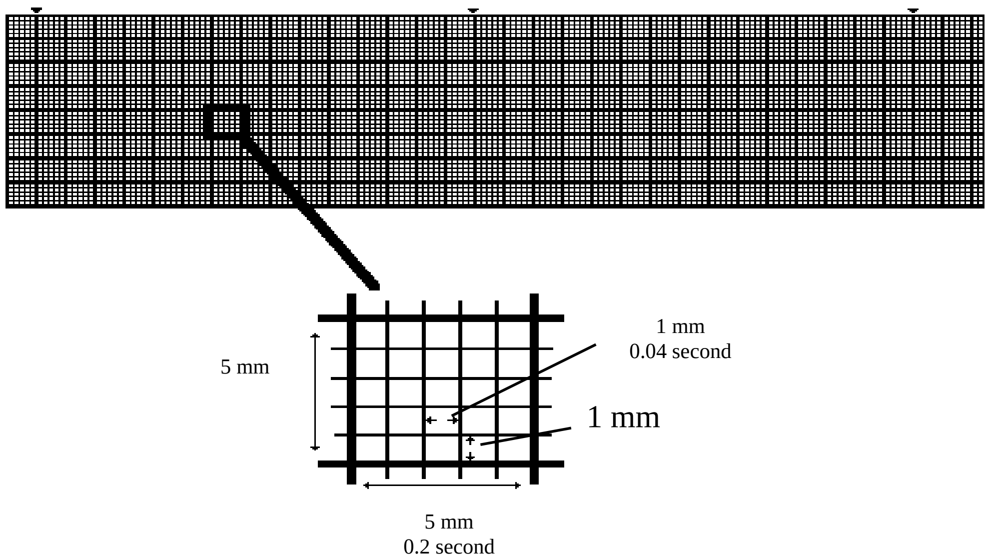
Phase 0: Slow Depolarization
Phase 1: Does not Apply
Phase 2: Plateau (Absolute Refractory Period)
Phase 3: Relative Refractory Period
Phase 4: Spontaneous Phase 4 Rise – constant influx of Na^{+}

Notes:

Section 2.0 – ECG Monitoring Equipment

ECG Paper

ECG monitors provide both dynamic and static monitoring capabilities. ECG paper has been standardized throughout the industry to maintain a running speed of 25 mm/second. It is the basis of this standard running speed that creates the large boxes with corresponding time frames. The need to know how the math works out is of little importance. The important information to remember is that each small square represents 0.04 second and each large square represents 0.2 second.



Formulas for paper speed and minute interval calculation.

25 mm/sec multiplied by 60 sec = 1500 mm/min

60 (1 minute) seconds divided by 1500 mm = 0.04 sec/mm

0.04 sec/mm x 5 = 0.2 sec/5mm (large square)

1500 mm/min divided by 5 = 300 large squared/min

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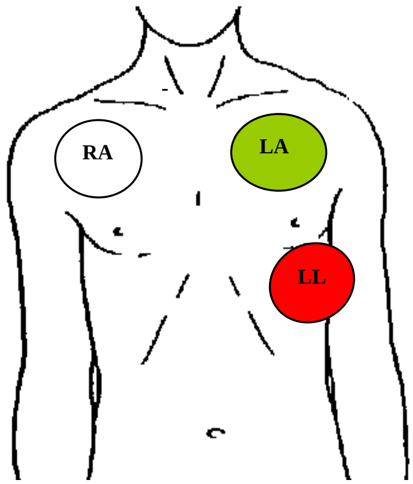
Section 2 – ECG Monitoring Equipment

ECG Cables/Electrodes

ECG cables are colour and letter coded: white (RA), red (LL), black/green (LA). The general rule for lead placement is:

White (**R**ight **A**rm) typically placed over right anterior chest, below clavicle
Red – (**L**eft **L**eg) placed at mid-axillary line below left nipple line
Black/Green – (**L**eft **A**rm) placed over left anterior chest, below clavicle
Ground lead (with 4 lead cable) – generally placed over the right lateral chest

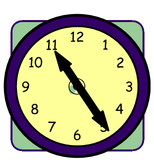


Factors affecting ECG quality:

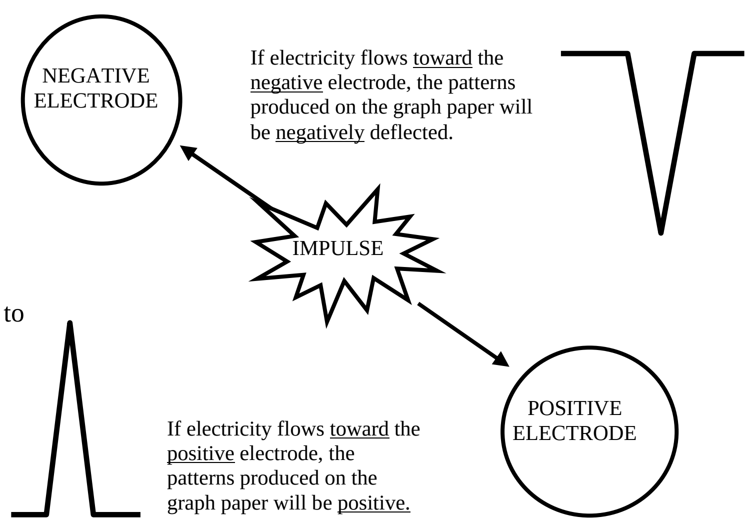
- Patient movement
- ECG electrode directly over large muscle (e.g. bicep)
- ECG electrode directly over bone
- dried out electrodes
- loose connections
- frayed cables
- improper lead selection

Rule of Electrical Flow

ECG complexes are the measurement of electrical flow registered on an oscilloscope (monitor) or plotted on a graph (ECG Paper). The ECG machine measures the electrical current flowing between the negative and positive electrodes. The diagram below describes the rule of electrical flow.



Normally conducted impulses travel the eleven to five o'clock vector



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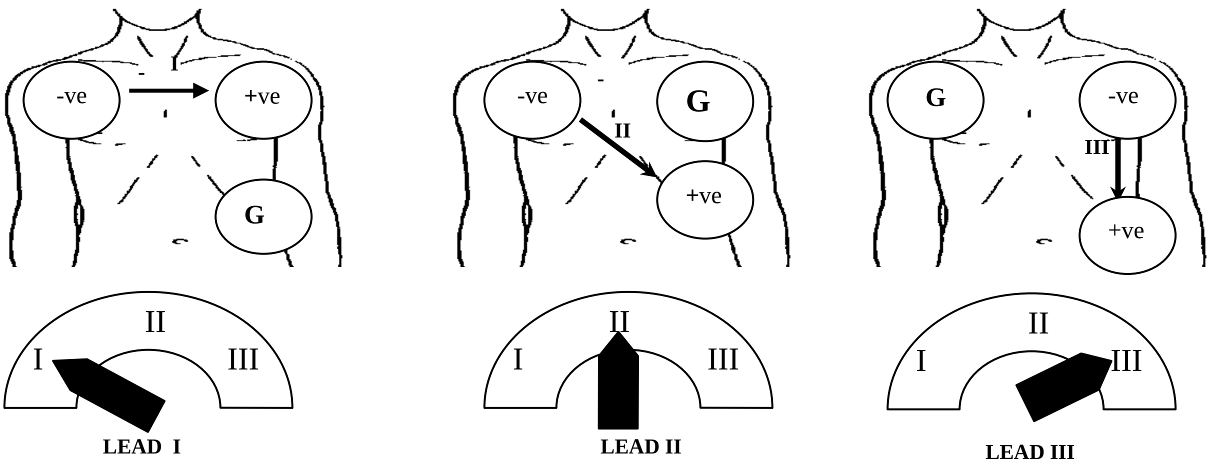
Section 2 – ECG Monitoring Equipment

Lead Placement - Frontal Plane

Most lead II ECG monitoring machines have the ability to look at three different frontal leads. When the lead selection is made by turning the switch to a specific lead, the polarity of the leads changes amongst the white, red and black leads as noted above.



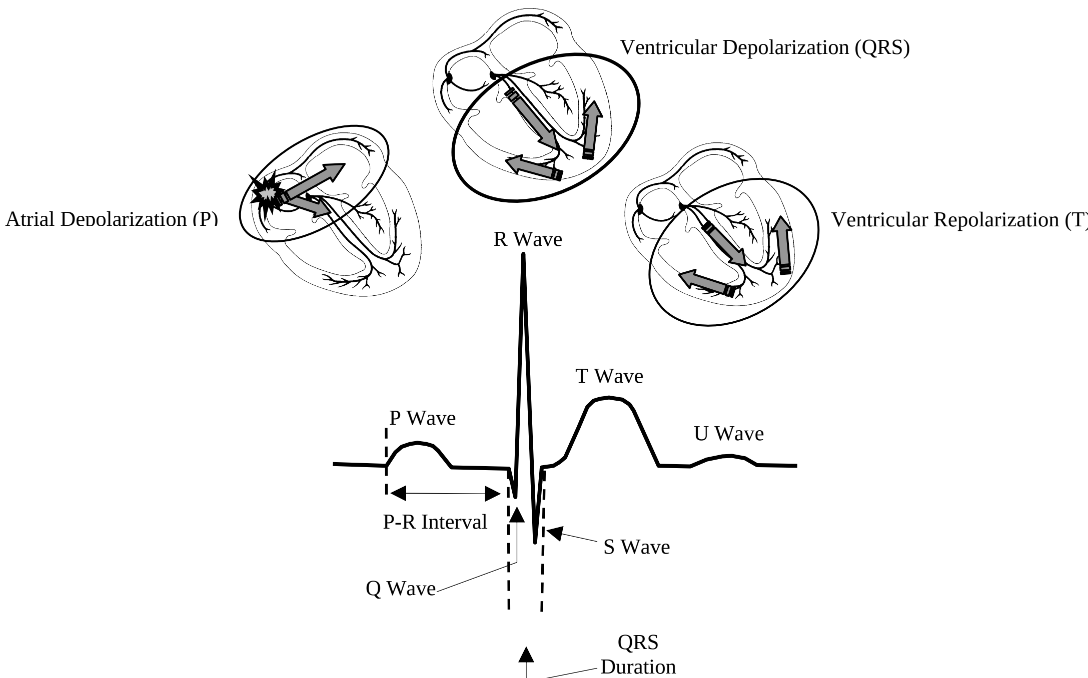
Lead I – monitoring atrial activity when difficult to see in lead II
Lead II – monitoring of normal conduction vector from right atria to left ventricle
Lead III – monitoring horizontal view of the heart with emphasis on left ventricle

Notes:

Section 3 – ECG Interpretation

P-QRS-T Configuration

The P-QRS-T configuration of the ECG is the electrical activity representative of the depolarization and repolarization of the atria and ventricles. The following diagram shows the correlation of the components of the ECG and the location within the heart that the electrical activity occurs.



P Wave - depolarization of the atria
- usually SA node
- upright

P-R Interval - impulse conduction from SA Node to and through AV node
- from beginning of P wave to beginning of QRS complex
- delay allows atria to fully contract and expel its content into the ventricles
- 0.12 to 0.20 seconds

QRS Complex - depolarization of the ventricle
- 0.08 to 0.10 seconds (or < 0.12)
- large amplitude due to large mass of ventricles producing more electrical activity
  - Q Wave - septal wall depolarization
    - first negative deflection on ECG complex after P wave
  - R Wave - ventricular wall depolarization
    - large mass of ventricles
    - first upright deflection
  - S Wave - lateral wall depolarization
    - downward deflection

T Wave - ventricular repolarization

U Wave - referred to as an “after-depolarization”. May result from electrolyte imbalance (e.g. Hypokalemia) or other causes.

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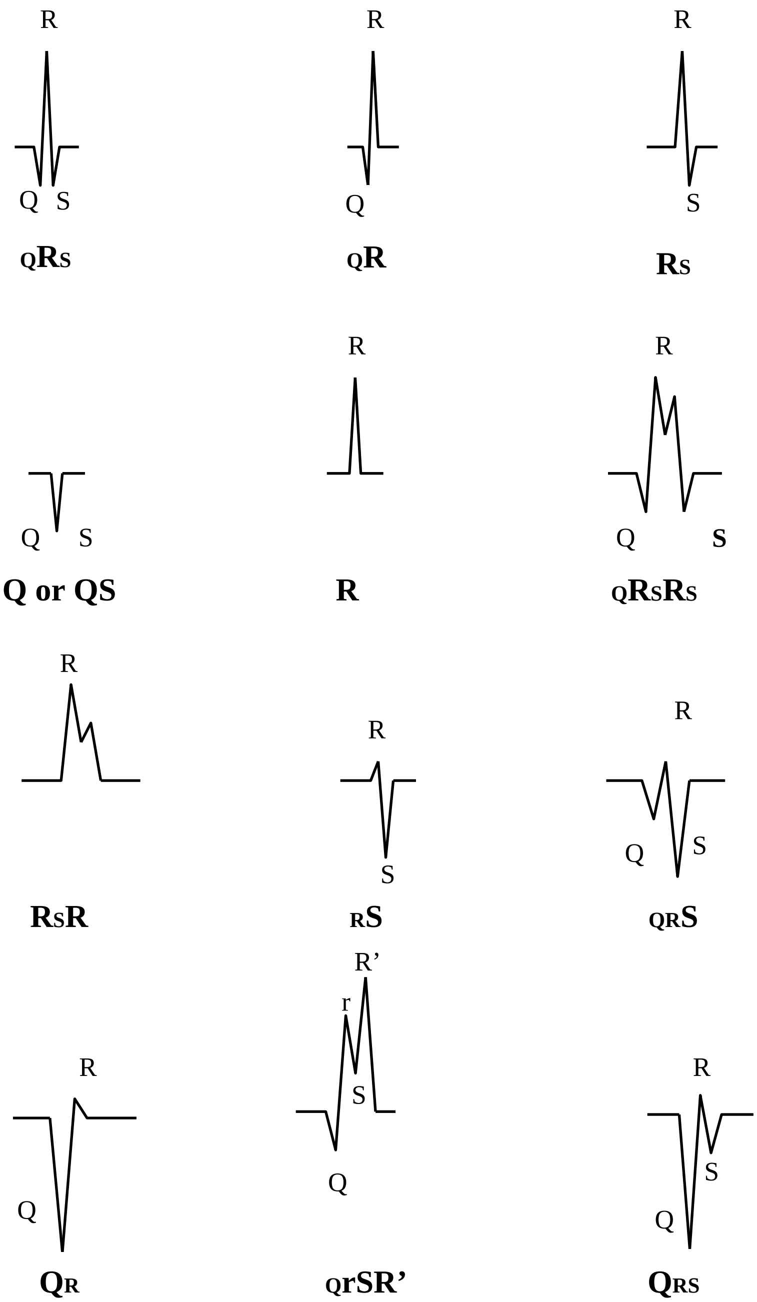
Section 3 – ECG Interpretation

QRS Morphology

The QRS complex of the ECG represents ventricular depolarization. The morphology or shape of the QRS complex can vary depending on the lead from which you're viewing the ECG, the individual patient or abnormal pathology. Although the QRS complexes below appear different, they may be "normal" for that individual or be suggestive of an underlying conduction disturbance (especially QRS's ≥ 0.12 second).



Notes:

Section 3 – ECG Interpretation

Five Steps For ECG Interpretation

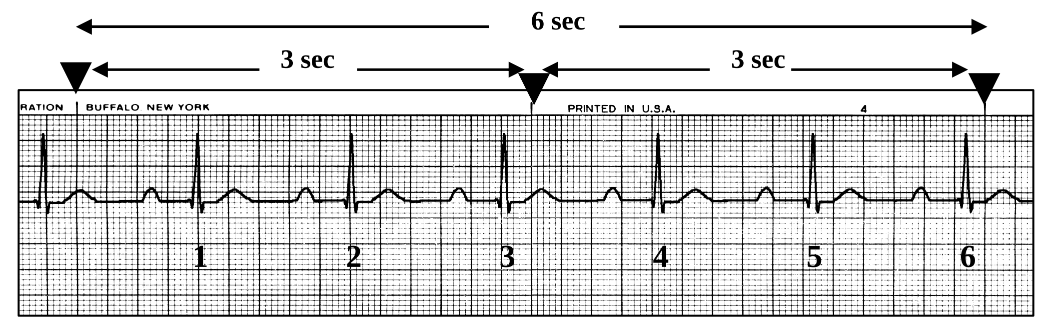
Listed below are the 5 steps to ECG interpretation. When interpreting the ECG it is important to follow the 5 steps to avoid what is known as "Pattern Recognition" whereby the interpreter assumes a rhythm is of one type by just looking at it whereas the rhythm would be identified correctly if the steps were followed.

Step 1 :	Rate	< 60	Bradycardic
		60 - 99	Normal
		$\geq$ 100	Tachycardic
Step 2 :	Rhythm	Regular	
		Irregular	
Step 3 :	P-R Interval	0.12 - 0.20 Second	
Step 4 :	P-QRS-T Relation	P Wave For Every QRS-t Complex	
Step 5 :	QRS Width	0.08 - 0.10 Second (or < 0.12 second is considered narrow)	
Step 6 :	Missing Or Added	Extra Beats Or Missing Beats	

Step One - Rate

Method 1

Count the number of R waves for a six second interval and multiply by ten.



6 x 10 = 60/min

Points to consider:

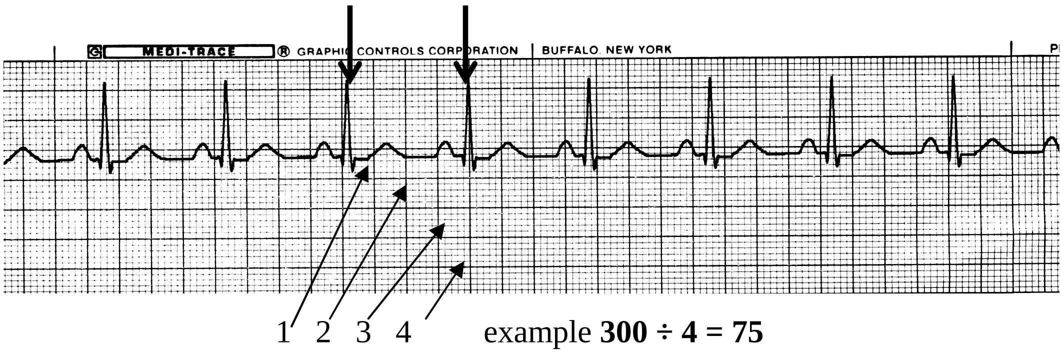
- reliable only when rhythm is regular
- good for normal heart rates – accuracy is diminished with fast or slow rates

Notes:

Section 3 – ECG Interpretation

Method 2:

Count the number of 5mm squares between each R wave and divide the number into 300. This will give you the approximate rate/minute.



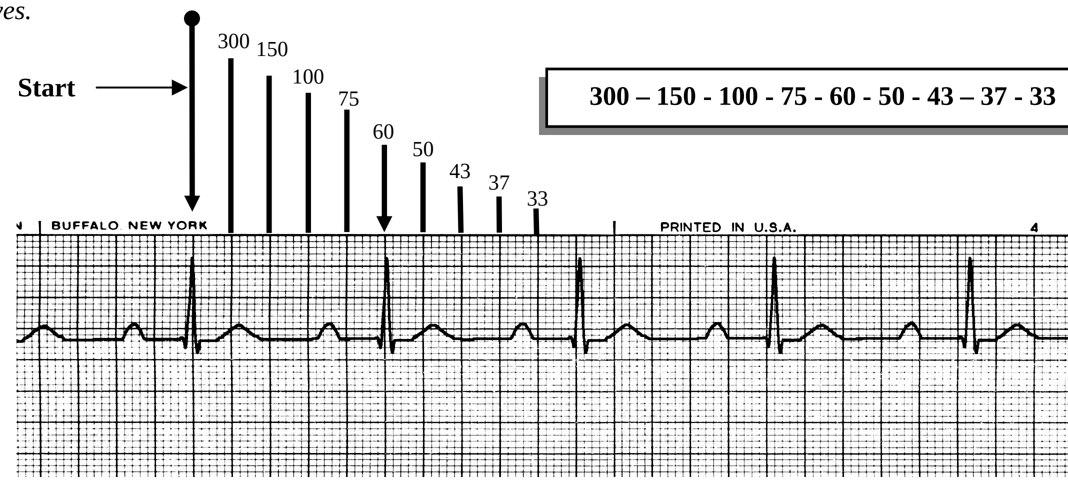
example **300 ÷ 4 = 75**

Points to consider:

- must be able to do division in your head

Method 3:

Using the following scale, where each number represents a 5mm square, count each 5 mm square between two R waves.



300 – 150 - 100 - 75 - 60 - 50 - 43 – 37 - 33

Points to consider:

- memorize the above numbers
- find a QRS complex that falls on a dark line
- count down from the next dark line as shown above
- reliable for regular rhythms
- recommended for brady and tachyarrhythmias

Notes:

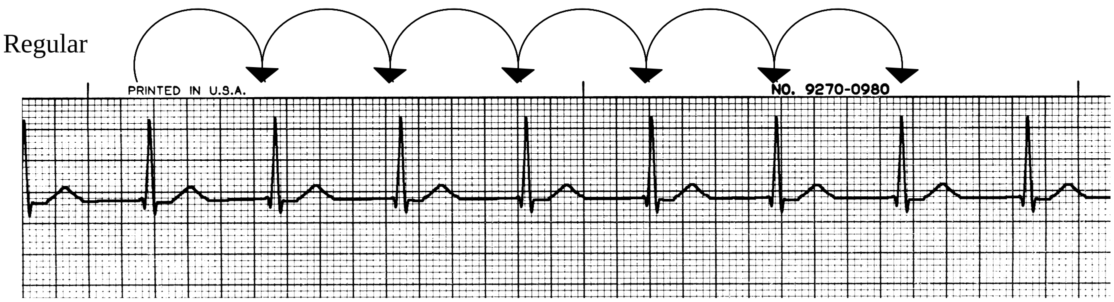
Section 3 – ECG Interpretation

Five Steps For ECG Interpretation (cont'd)

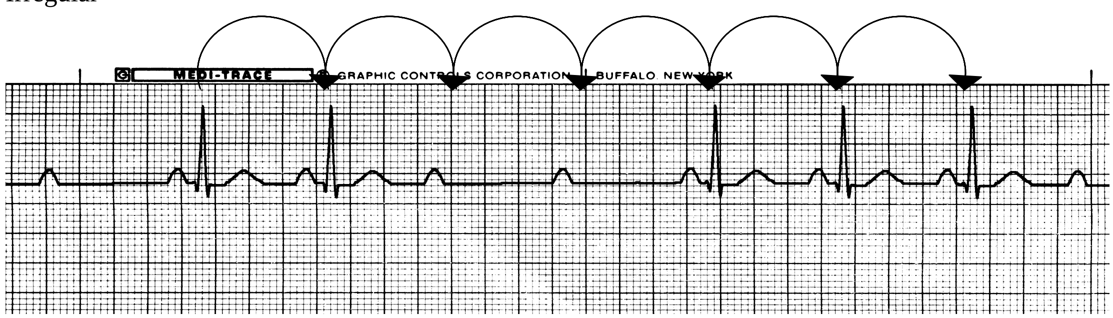
Step Two - Rhythm

Measure the R to R intervals to determine if the rhythm is regular in nature. It is acceptable to allow for a difference of +/– one small square when determining regularity of the rhythm

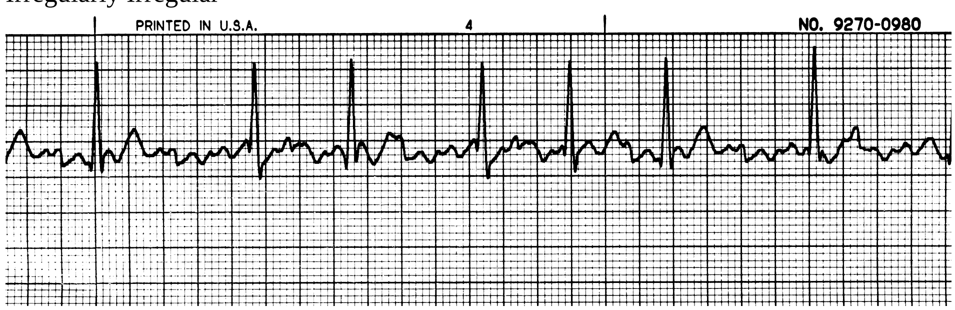
Regular



Irregular



Irregularly Irregular



Points to consider:

- regular rhythms are produced by a single focus
- regularly irregular rhythms may occur with heart blocks or regular PVCs
- with very fast rhythms it is sometimes difficult to determine if a regular pattern exists

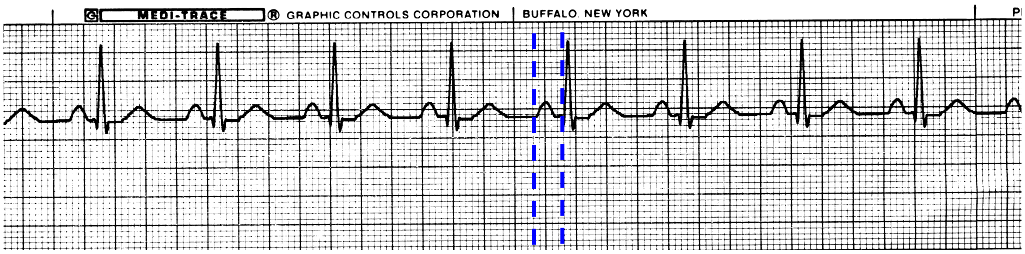
Notes:

Section 3 – ECG Interpretation

Step Three - P-R Interval

The P-R Interval is the time required for the impulse to travel from the SA node through the AV node. Impulses generated in the atria should take between 0.12 to 0.20 seconds to travel through the AV node. Any delay of > 0.20 seconds is considered a significant conduction delay.

≤ 0.20 SEC



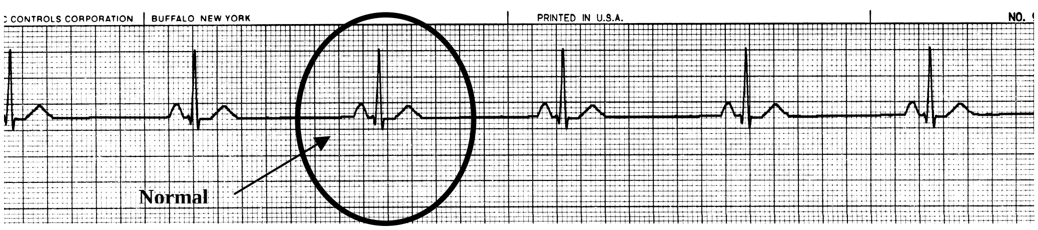
0.12 - 0.20 second
1 Large Square or 5 Small Squares

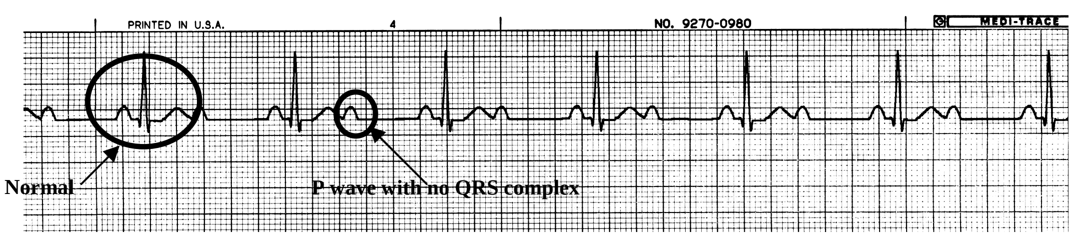
Points to consider:

- measure PR Interval from beginning of P wave to beginning of QRS deflection regardless of morphology
- must identify P wave over T wave

Step Four - P-QRS-T Relationship

Look for a marraige of P waves to QRS complexes. Normal complexes will always have the components as in the diagram below. P waves with no corresponding QRS complexes or QRS complexes with no associated P wave will signal heart block or ectopic beats.





Points to consider:

- fast rhythms may cause P waves to encroach on the preceding T wave
- more than one P wave per QRS may signal heart block or non-conducted PACs

Notes:

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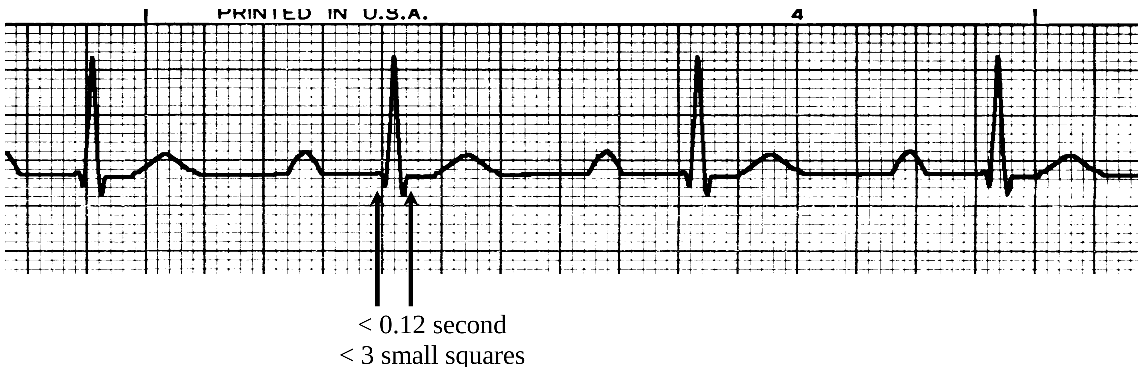
Section 3 – ECG Interpretation

Five Steps For ECG Interpretation (cont'd)

Step Five - QRS Duration

Measure from the first deflection (+ve or –ve) to where the QRS complex comes back to the isoelectric line (J point). The normal range for the QRS width is < 0.12 second or < 3 small squares.



Points to consider:

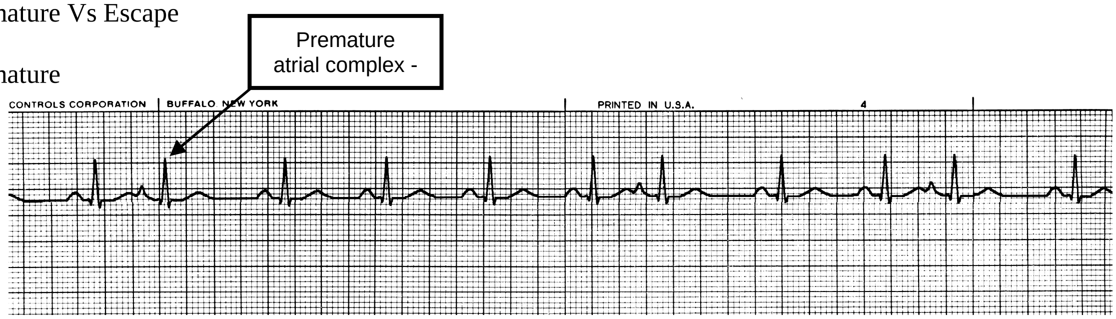
- QRS widths of $\geq$ 0.12 are not always ventricular in origin, but can be supraventricular beats with aberrant conduction as a result of a bundle branch block, drug effect, etc.

Step 6 – Anything Missing/Added

This is the unofficial step that can help aid in the interpretation of any ECG. Look for missing beats where you expect them to be, or added complexes where they weren't expected. Look for premature beats or escape beats.

Premature Vs Escape

Premature



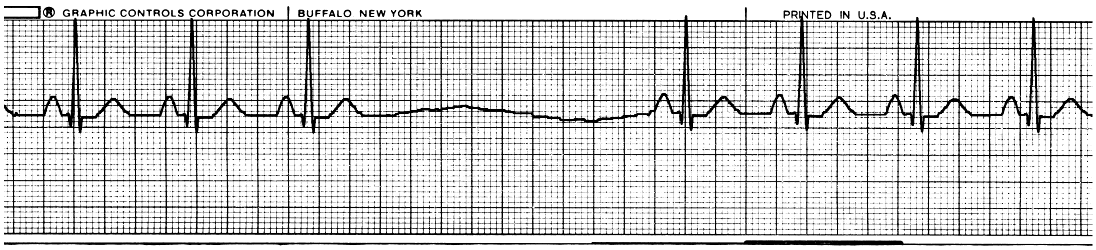
Extra QRS complexes which occur before the next expected beat are referred to as premature complexes. There are multiple premature atrial (P waves present) beats in the above strip. Note how they occur prior to the next expected beat.

Notes:

Section 3 – ECG Interpretation

Step 6 – Anything Missing/Added (cont'd)

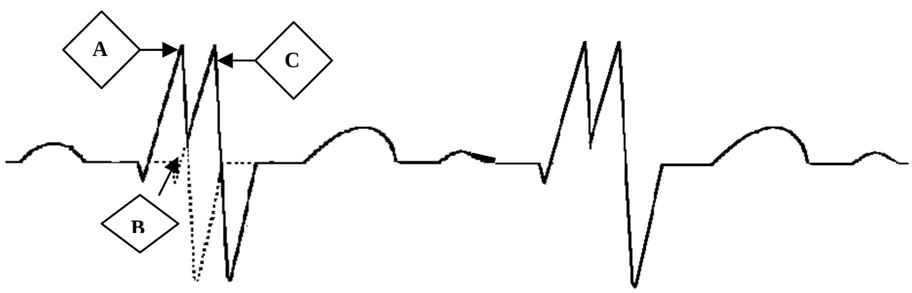
Escape



The above ECG is an example of a **Sinus Arrest**. Note that an *escape beat* (compensatory mechanism) occurs when there is a missing complex. The complex that appears after the delay is referred to as an escape beat.

3.3 Aberrant Conduction

Aberrant conduction occurs when the QRS complex appears wider than normal ($\geq$ 0.12 second). The impulse may follow the normal pathways in the atria but as it enters the ventricles it is blocked in either the left or right bundle branches. This results in one ventricle depolarizing just prior to the other ventricle depolarizing. The delay can occur in either the right or the left bundle branch. It is impossible to determine the exact location of the delay using leads I, II or III and requires the use of modified chest leads or a 12 lead ECG to determine the location.



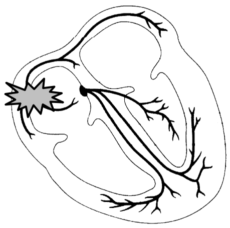
Example: **A** – right ventricle depolarizes
B – delayed conduction in left bundle branch
C – left ventricle depolarizes

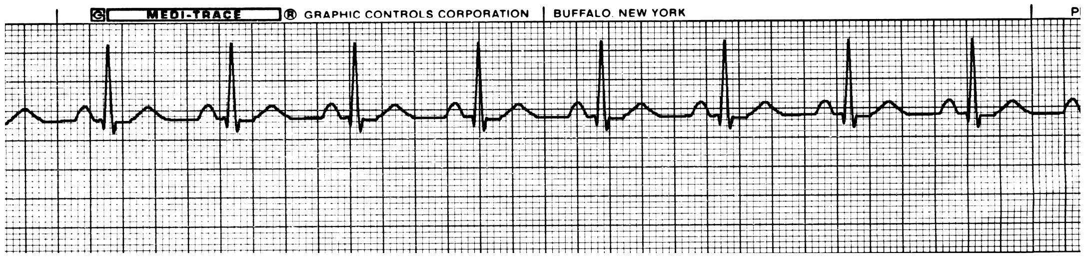
Notes:

Section 4 – Cardiac Rhythms/Arrhythmia's

Supraventricular Rhythms

Normal Sinus Rhythm (NSR)





Sinus Bradycardia

Notes: