Study Guide for the NTA Telemetry Certification

Disclaimer: the intent of this site is for educational purposes only, NOT diagnostic. ECGs are complex with many subtle variations and can be difficult or misleading to interpret. Full and proper analysis requires much specialized training and experience. Most non-medical instructors (and even some who are medical instructors) do not have the expertise for diagnostic interpretations of abnormal ECGs and might not even be able to detect some abnormal situations. Most, if not all, ECGs that are run on students in a classroom setting or by persons on themselves will be "normal." However, if anyone encounters something on an ECG that appears like it might be suspicious or not normal, the person should see a physician and have another ECG run under proper clinic or hospital diagnostic conditions and read by someone who is fully trained in ECG interpretation.

A microbit of history about the ECG / EKG

Our understanding of biological electricity began and gradually increased through the efforts of many persons during the 17th-19th centuries. The term "electrocardiogram" and our modern-day combination of leads started around 1890 with Einthoven (and possibly his immediate predecessors), first with three main limb leads, the addition of the six chest leads during the 1930′s, and finally the three augmented limb leads in 1942 to produce the total of 12 leads used up to the present. Some of the earliest equipment and techniques were downright amazing for being able to detect and record the slight amounts of electricity (in millivolts or mV) coming from the heart (and other muscles).

Initially, and yet today with the "resting 12-lead" ECGs, the subject needed/needs to be at rest to minimize electrical signals from other muscles in the body. However, over time and with improved computing algorithms, it became possible to measure ECGs during exercise and "stress" tests. Portable or "Holter" units, named after Dr. N.J. Holter, a Montana physician who initially developed it in 1949, permit recordings over long periods of time during routine daily

life including various activities and exercise. The original Holter unit was a 75 pound backpack! Modern Holter units, whether they record with cassette tapes or digitally, are small in size.

Introduction to the standard 12-lead ECG / EKG

At first encounter for someone who has not seen one before, a standard 12-lead ECG is a bewildering and confusing array of squiggles on a piece of paper! What does it mean and how does it relate to the heart?

Well, let's start with the basic object of interest, the heart, and make sure we know where it is in the body and how it operates. It is in the chest cavity just under the sternum, as shown on the following models.

Contractions of skeletal ("voluntary") muscles are initiated directly from the nervous system whereas contractions of heart muscle are initiated internally, in the muscle fibers themselves, and are only speeded up or slowed down by the nervous system (and a variety of chemical factors). The process involves movements of ions in and out of the muscle cells and cellular depolarization and repolarization. For more on all of that, refer to a basic biology textbook.

For our interest, electrical activity from the muscular contractions spreads throughout the body and can be detected on the surface on the skin. Contacts on the skin are called electrodes or "leads" which connect to the recording/measuring devices, or ECG machines.

The main, typical waves of an ECG are identified as P, Q, R, S, and T (the symbols A, B, C, … and X, Y, Z etc. had already been used for other physiologically-related items at the time when the system was first developed). Note: all of the waves do not appear on all recordings and there are also some other waves (with other names) that sometimes show up. The following recording, for example, does not show a "Q" wave, a downward wave just before the R wave, although the position of a Q wave (when present) is shown.

The pattern can be broken down separately for the atria and ventricles as follow:

Note: atrial repolarization is obscured by ventricular depolarization.

The specific appearance of a tracing depends on the location of the electrode (position on the body and, for those leads within a few centimeters on the heart, on the chest, the distance from the heart) and what the heart's electrical activity is doing (resting or active, normal vs various abnormalities, etc.).

Healthy hearts vary (somewhat like fingerprints) in their performance and output but produce a normal range of values. When something in the heart isn't working correctly as a result of

disease, accidents, or genetic and developmental malformations, the signals are different or abnormal (and require a trained physician or specialist to diagnose).

Serum Creatine Kinase

The traditional "gold standards" for diagnosing MI is the rise and fall of the serum MB fraction of CK. The CK-MB serum levels elevate 4 to 8 hours after MI, Peak at 15 to 24 hours, and remain elevated for 2 to 3 days. Serial samples are usually sufficient to support or rule out the diagnosis of MI.

Traponin

The traponins are a structurally related groups of proteins found in both cardiac and skeletal muscle and are termed cardiac troponin I (cTnI) and cardiac troponin T (cTnT). Because of the specific cardiac nature of TnI and TnT, both are used as makers of acute MI, both in centralized laboratory tests and in bedside point-of-care testing. Several different methods of laboratory assay are currently in clinical use, which means that "normal" serum levels will vary between different clinical settings, although serum cardiac TnI and TnT levels are very low in the absence of myocardial muscle damage.

Myoglobin

Myoglobin is a nonspecific indicator of myocardial damage because it is identical in both cardiac and skeletal muscle. Myoglobin is useful because it is the enzyme that rises earliest in the serum, about 2 hours after myocardial injury. It is never used alone but can be used in conjunction with other, more cardiac-specific enzyme markers.

Trending Hematologic Studies

Hematologic laboratory studies routinely ordered for the management of patients with altered cardiovascular status include red blood cell (RBC), hemoglobin (Hgb), hematocrit (Hct), and white blood cell (WBC) concentration.

Red Blood Cells

The normal amount of RBCs present varies with age, gender, altitude, and exercise. Anemia is a clinical condition in which insufficient RBCs are available to carry oxygen to the tissues. Polycythemia occurs when excess RBCs are produced.

Hemoglobin and Hematocrit

Normal Hgb levels are 14 to 18 grams per deciliters (g/dL) in males and 12 to 16 g/dL in females. The Hct is the percentage of RBCs in whole blood: 40% to 54% for men and 38% to 48% for women.

White Blood Cells

Most inflammatory processes (e.g., rheumatic fever; endocarditis, MI) that produce necrotic tissue within the heart muscle increase the WBC count. The normal WBC level for both men and women is 5000 to 10,000 cells/mm3.

Assessing Blood Coagulation Values

Coagulation studies are ordered to determine effectiveness of blood clotting. Anticoagulants, most notably heparin, warfarin, and platelet inhibitory agents (e.g., GPIIb/IIIa agents, aspirin), are administered to decrease MI extention and to reduce the incidence of reocclusion after successful coronary artery reperfusion, as well as for management of patients with unstable angina and non-ST-segment elevation acute MI. patients who have stasis of blood, valvular heart disease, atrial fibrillation, or a history of thrombosis are at risk of developing a thrombus and usually requires anticoagulation. Coagulation studies are required to guide dosage of antithrombotic drugs.

Prothrombin Time and International Normalized ratio

Most coagulation study results are reported as the length of time in seconds it takes for blood to form a clot in the laboratory test tube. The prothrombin time (PT) is also reported as an international normalized ratio (INR). The INR was developed by the world health organization (WHO) in an attempt to standardize PT results among clinical laboratories worldwide. The PT and INR are used to determine therapeutic dosage of warfarin (Coumadin) necessary to achieve anticoagulation.

Partial Thromboplastin Time and Activated Coagulation Time

The Partial Thromboplastin time (PTT) and activated partial thromboplastin time (aPTT) are used to measure the effectiveness of heparin administration. An additional test of heparin effect is the activated coagulation time (ACT). The ACT can be performed outside the laboratory setting in the cardiac catheterization laboratory, operating room, and specialized critical care units.

Evaluating Serum Lipid Profile

Four primary blood lipid levels are important in evaluating and individual's risk of developing CAD or experiencing progressive CAD: total cholesterol, low density lipoprotein, triglycerides, and high density lipoprotein (LDL) cholesterol when LDLs and triglycerides are elevated or HDLs are low, the patient is considered "at risk" for developing or having progressive CAD.

Total Cholesterol

Cholesterol is a fatlike substance (lipid) present in cell membrane, is a precursor of bile acids and steroids hormones, and is produced by the liver. The cholesterol level in the blood is determined partly by genetics and partly by acquired factors such as diets, calorie balance, and level of physical activity. Cholesterol in excess amounts (greater than 200 mg/dL) in the serum fosters the progression of atherosclerosis.

Low Density Lipoproteins

About 60% to 70% of the total serum cholesterol is transported in the bloodstream complexed as LDL cholesterol. Both the LDL cholesterol and total serum cholesterol levels are directly correlated with risk for CAD, and high levels of each are significant predicators of future MI. LDL cholesterol is the major atherogenic lipoprotein and thus is the primary target for cholesterol lowering efforts.

Very-Low-Density Lipoproteins and Triglycerides

The-very-low-density lipoproteins (WLDLs) contain 10% to 15% of the total serum cholesterol along with most of the triglycerides in fasting serum. Elevated triglyceride levels are also often associated with reduced HDL cholesterol levels.

High-Density Lipoproteins

HDL cholesterol particles carry 20% to 30% of the total serum cholesterol. A low HDL cholesterol level (less than 35mg/dL) is another independent, significant risk factor for coronary artery disease. A high HDL cholesterol level protects against atherosclerotic CAD.

Nursing Management

The nursing management of a patient undergoing a diagnostic procedure involves a variety of interventions. Nursing priorities are directed toward (1) preparing the patient psychologically and physically for the procedures, (2) obtaining informed consent, (3) monitoring the patient's physiologic responses, and (4) assessing the patient after the procedure.

Preparing the patient includes teaching about the procedure, answering questions, and ensuring that the patient is informed about the diagnostic procedure. If the procedure is invasive the medical professional that will perform procedure must discuss risks, benefits, and potential complications with the patient to ensure that informed consent is obtained. The critical care nurse

may assist with transport or positioning of the patient for the procedure. Monitoring the patient's responses during diagnostic procedures includes observing for signs of pain, anxiety, and hemorrhage and monitoring vital signs. Assessing the patient after the procedure includes monitoring for complications and medicating the patient for any postprocedural anxiety, pain, or discomfort. Evidence of bleeding or chest pain should be immediately reported to the physician and emergency measures undertaken to maintain circulation and increase myocardial oxygen supply.

In addition to **Lead I** which was shown above, there are several other leads that provide different views of the electrical activity. Their polarities and measurements are all manipulated by the ECG machine. The other leads are shown in the following figures.

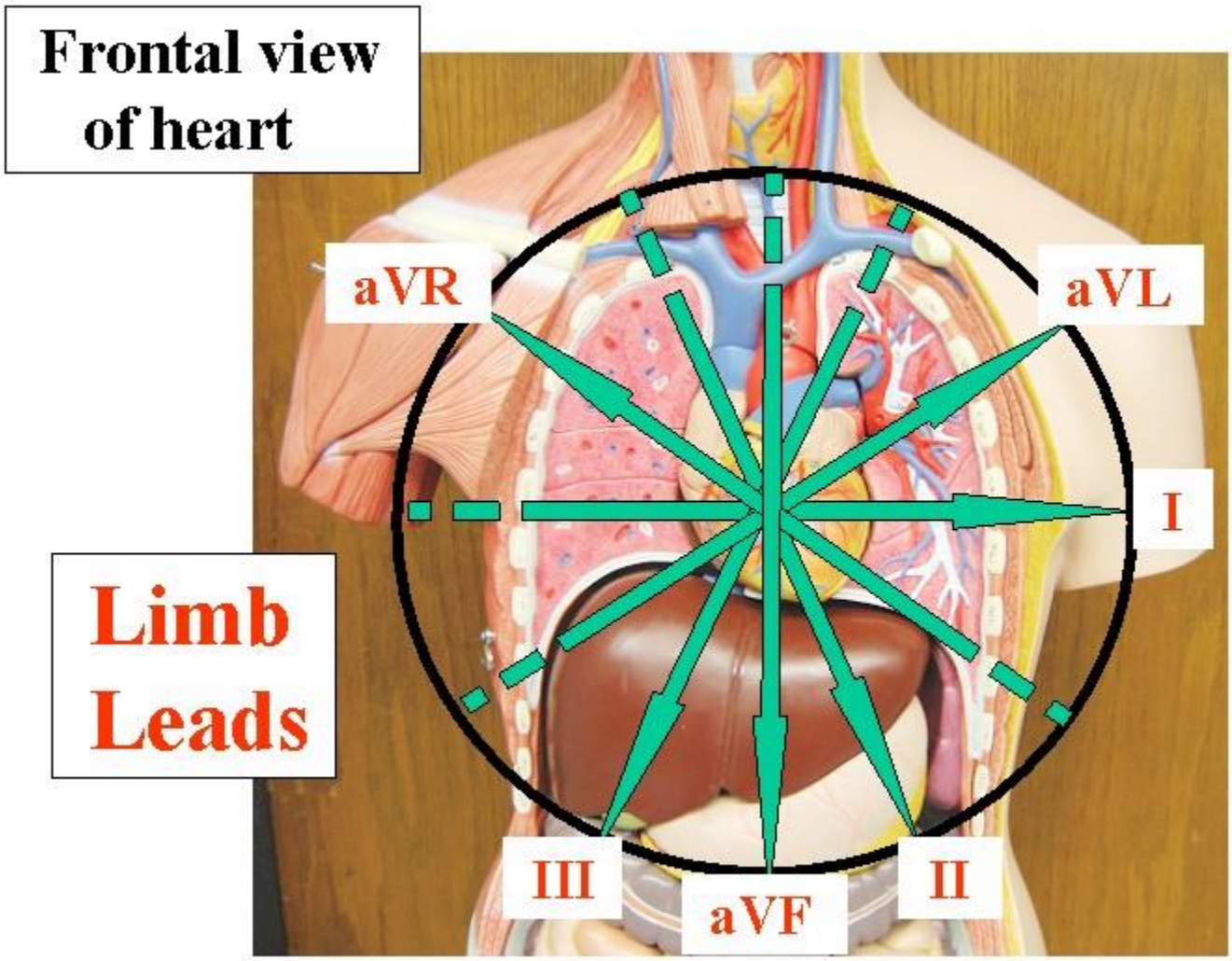
Note that even though there are "12 leads", **there are only 10 actual contacts on the body**. There are 9 recording leads (3 on the limbs [right arm, left arm, and left leg] plus 6 on the chest over various parts of the heart) and a 10th neutral or ground lead attached to the right leg. The remaining 3 leads representing the limbs are called "augmented" leads, which are derived from vectored combinations of the other 3 actual limb leads and provide different angles of view.

The various limb leads together provide a frontal view of the heart. (Always keep in mind that left and right orientation are relative to the subject, not the observer.)

The remaining 6 leads are located across the chest and provide a cross-sectional view of the heart. They go by several names: "precordial" ("in front of the heart"), "pericardial" ("around the heart"), or simply the "chest" leads.

In summary: the 12 standard leads are

Limb leads –



I, from the right arm (-) toward the left arm (+)
II, from the right arm toward the left leg
III, from the left arm toward the left leg
(taken together, these three form the classic "Einthoven's triangle")

aVR, augmented lead toward the right (arm)
aVL, augmented lead toward the left (arm)
aVF, augmented lead toward the foot
(note: aVR is approximately opposite of I and should essentially mirror the shape of I vertically)

Chest leads –

V1 through V6, starting over the right atrium with V1, and placed in a semi-circle of positions leftwards, to the left side of the left ventricle. **V1** and **V2**, on the right and left sides respectively, are placed just off the sternum at the 4th intercostal spaces (the space between the 4th and 5th

ribs, which can be felt through the skin) and the others travel around to **V6** under the armpit, as shown in the diagram.

[Detailed instructions for placing the leads:

Limb leads: Arms (RA, LA) anywhere from the upper arm to the wrists
Legs (RL, LL) anywhere from the thigh to the ankles
(positions of limb leads or their distances from heart are not critical, as long as they are more than 10 cm [4 inches] from the heart)

Chest leads:V1 to the right of the sternum, next to it, in the space between ribs 4 and 5
V2 as V1 but to the left of the sternum
V3 halfway between V2 and V4 (see next one)
V4 below the middle of the clavicle, between ribs 5 and 6
V5 left of V4, halfway between V4 and V6 (see next one)
V6 on same horizontal line with V4 and V5, below middle of armpit]

The normal progression of muscular contractions, hence, electrical activity, travels from the upper right part of the atria downward and leftwards to the ventricles, with the left ventricle being the strongest. This leads to a topic of ECG interpretation involving the electrical "axis" of the heart, which is mostly beyond this introduction. However, for a general picture: The pattern on any one lead, and all of the leads taken together, represents a view of the sum of all of the vectors from contractions of the different muscle fibers in the various parts of the heart. The total picture for any given wave (P, R, T) is the heart's electrical axis, which normally is from the upper, posterior right side of the heart (in the right atrium) to the lower point of the ventricles in a left direction. For example, the normal axis should generally be a combination toward the left (that is, upward in lead I) and toward the feet (that is, tracing upward also in aVF). Further details of the axis are determined by the direction of the traces in the other leads.

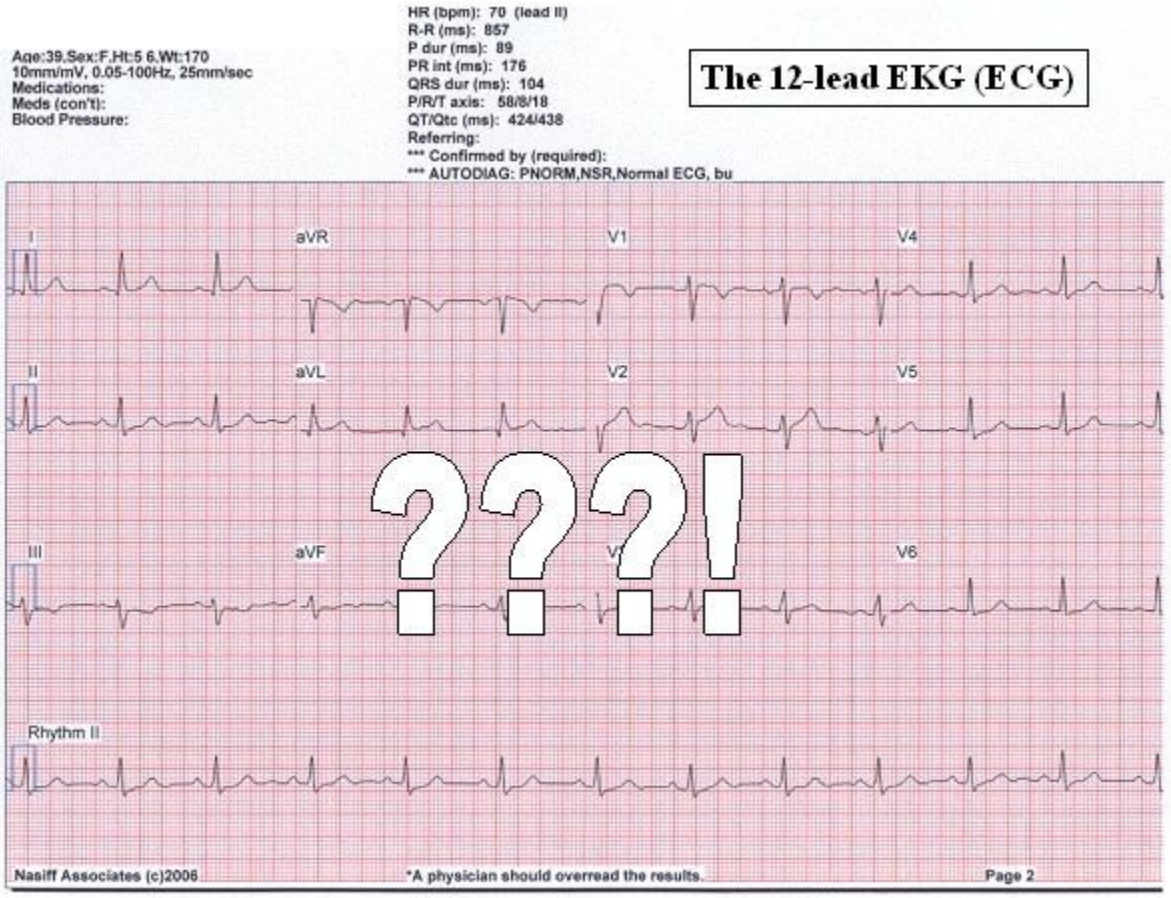
Various combinations of limb leads and chest leads taken together provide a **three-dimensional view** into the electrical activity and workings of the heart for anyone who knows how to read an ECG. A heart attack and resulting damaged or dead portions of the heart, for example, can greatly affect the summed vectors, hence, the axes of the various waves.

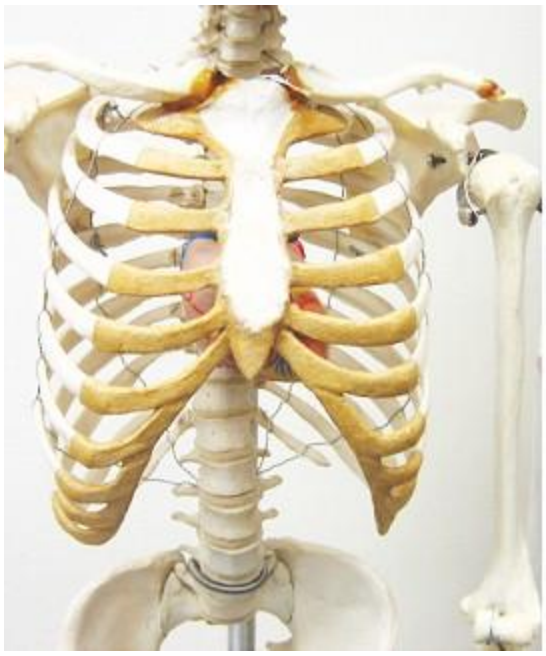
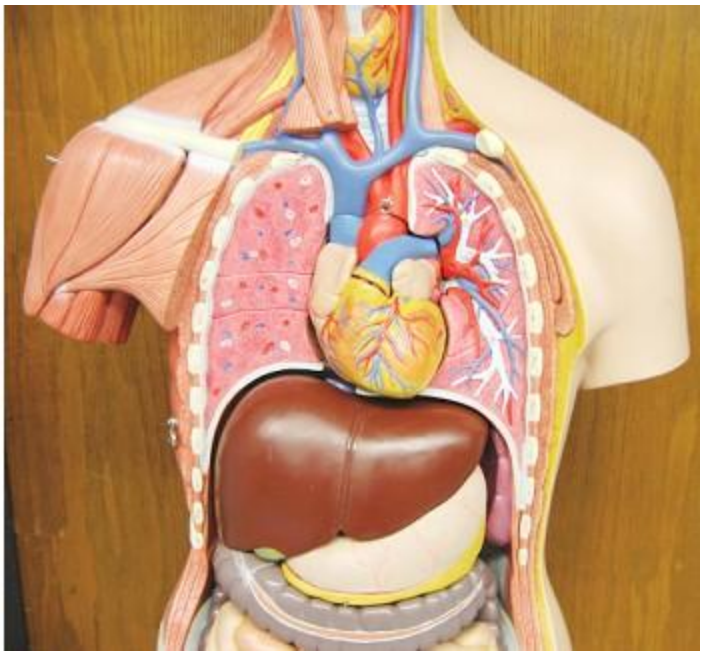
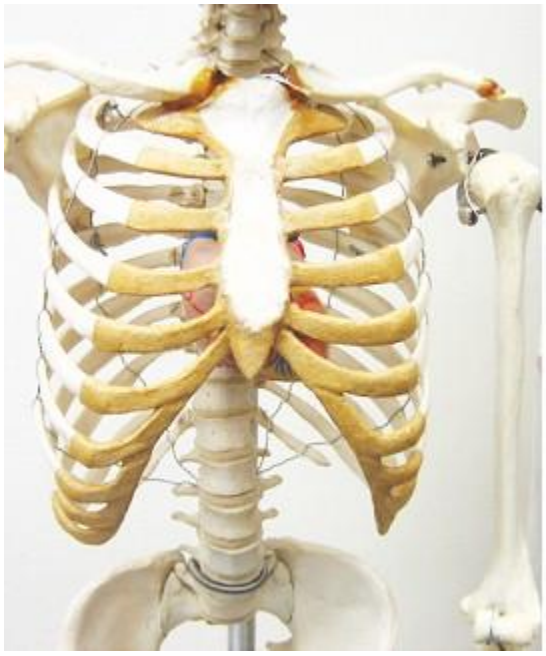
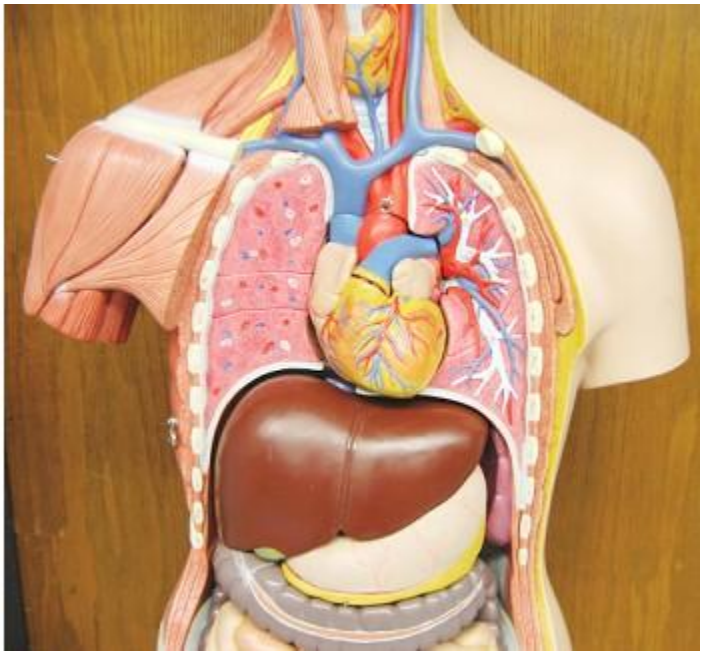
Various abnormalities including heart attacks, arrhythmia's, congenital problems, and a host of diseases and factors that affect the heart will cause sometimes major and sometimes subtle changes to the ECG patterns, which can be interpreted by a trained, experienced observer.

Now we can return to a standard 12-lead ECG print and begin to make sense of it. It shows each of the 12 leads in their own segments on the page. Given even a rudimentary understanding, hopefully as provided above, you can interpret some of the variations among the different leads, as shown in the following figures. The figures also illustrate some of the other components of a typical ECG, including a rhythm trace, calibration boxes, etc. This is an example of a "normal" ECG.

Below you will find some selected videos to help you visually understand lead placement.

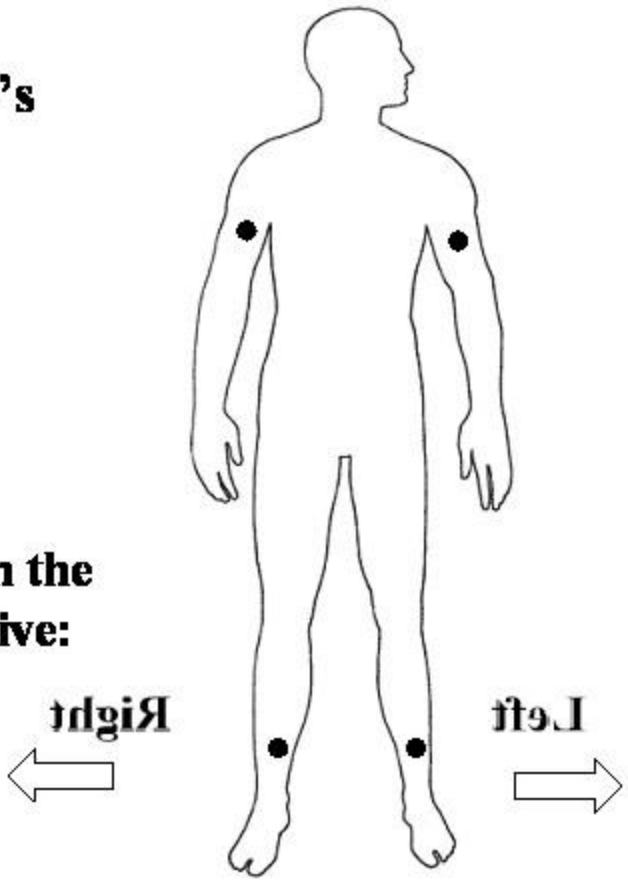
To help further understand this and get some hands-on experience, see the section on, as well as consider doing some exercises as described in the next section.



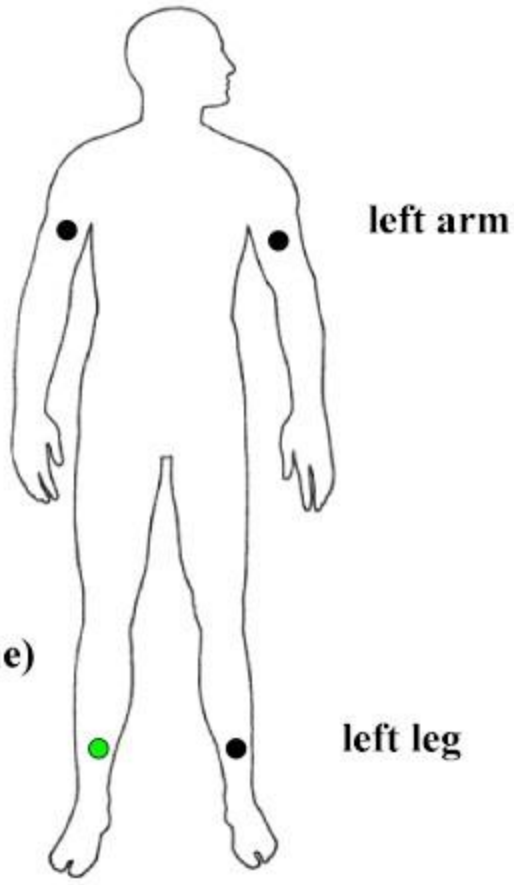





Sensing the heart's electrical activity via electrodes
(contacts placed on the surface of the body)

Note: anatomical orientation is from the subject's perspective:



The basic four limb electrodes:



electrical polarity:

- neutral or ground
- negative
- positive

(manipulated by the EKG machine)

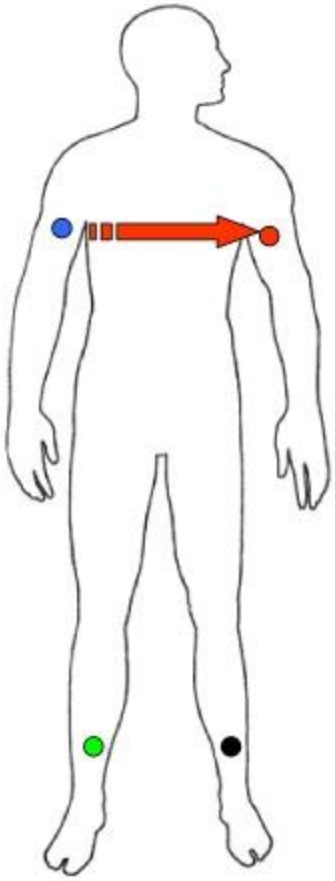
Lead I (toward left)

right arm

left arm

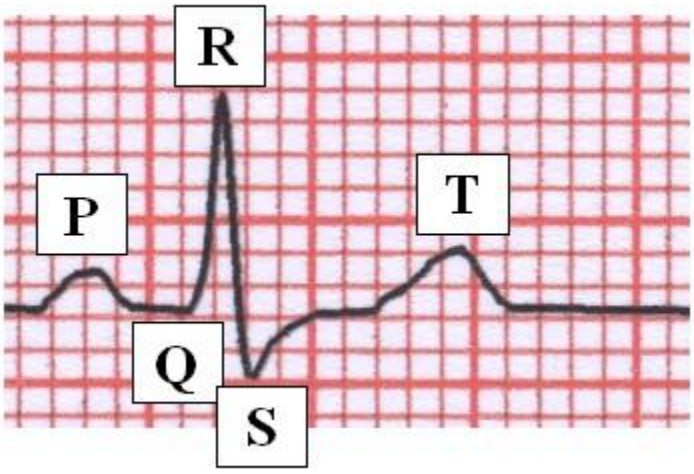
electrical polarity:

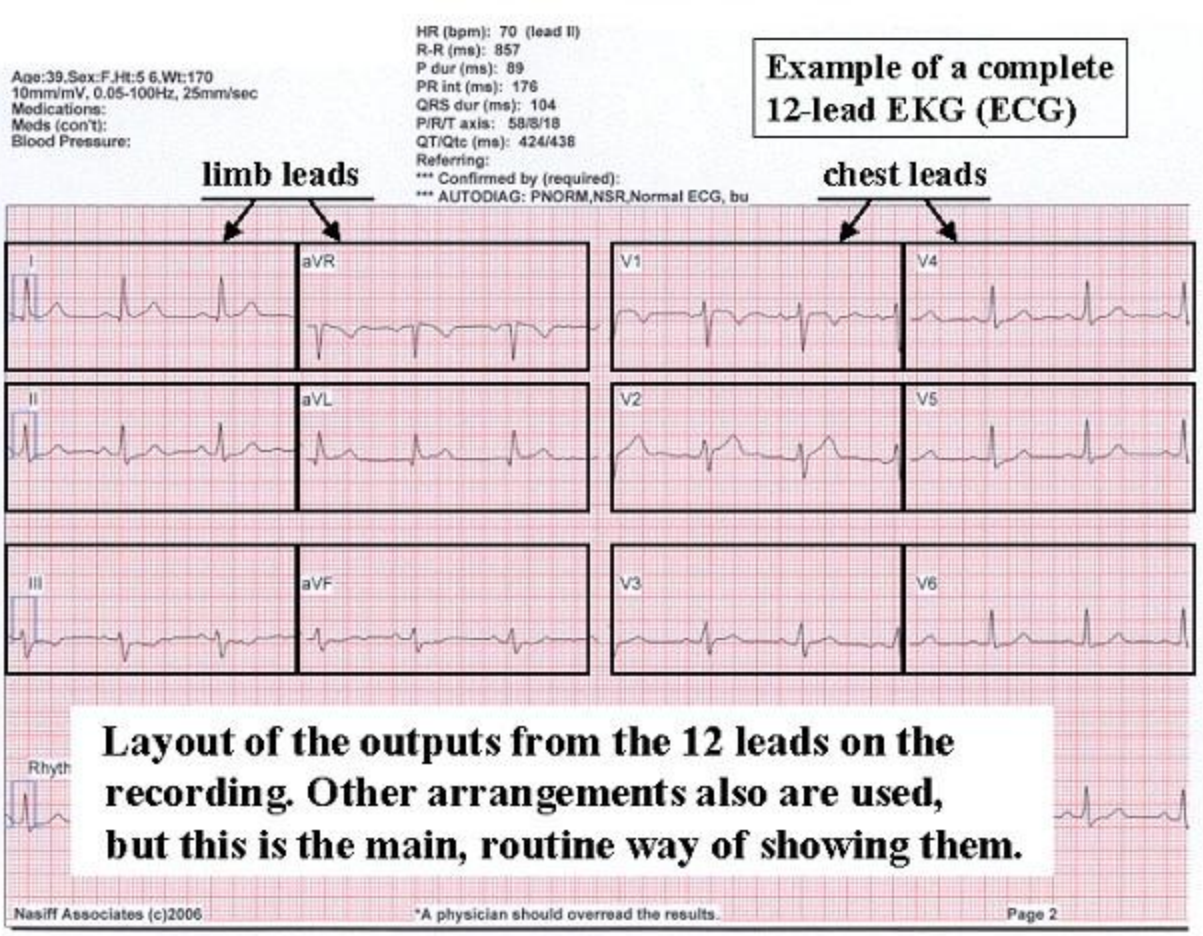
- neutral or ground
- negative
- positive

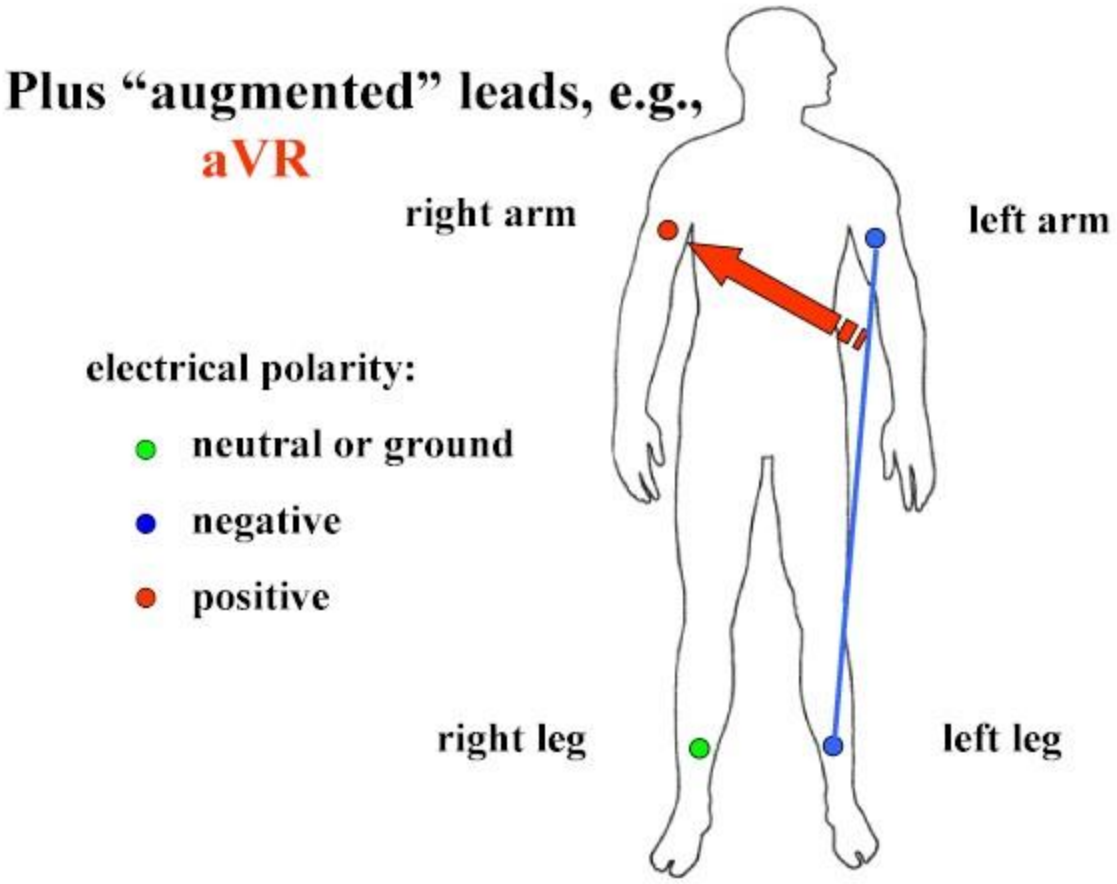


right leg

left leg




Example of a complete 12-lead EKG (ECG)
Age:39,Sex:F,Ht:5 6,Wt:170
10mm/mV, 0.05-100Hz, 25mm/sec
Medications:
Meds (con't):
Blood Pressure:
HR (bpm): 70 (lead II)
R-R (ms): 857
P dur (ms): 89
PR int (ms): 176
QRS dur (ms): 104
P/R/T axis: 58/8/18
QT/QTc (ms): 424/438
Referring:
*** Confirmed by (required):
*** AUTODIAG: PNORM,NSR,Normal ECG, bu
limb leads
chest leads
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
Rhyth
Layout of the outputs from the 12 leads on the recording. Other arrangements also are used, but this is the main, routine way of showing them.
Nasiff Associates (c)2006
*A physician should overread the results.
Page 2



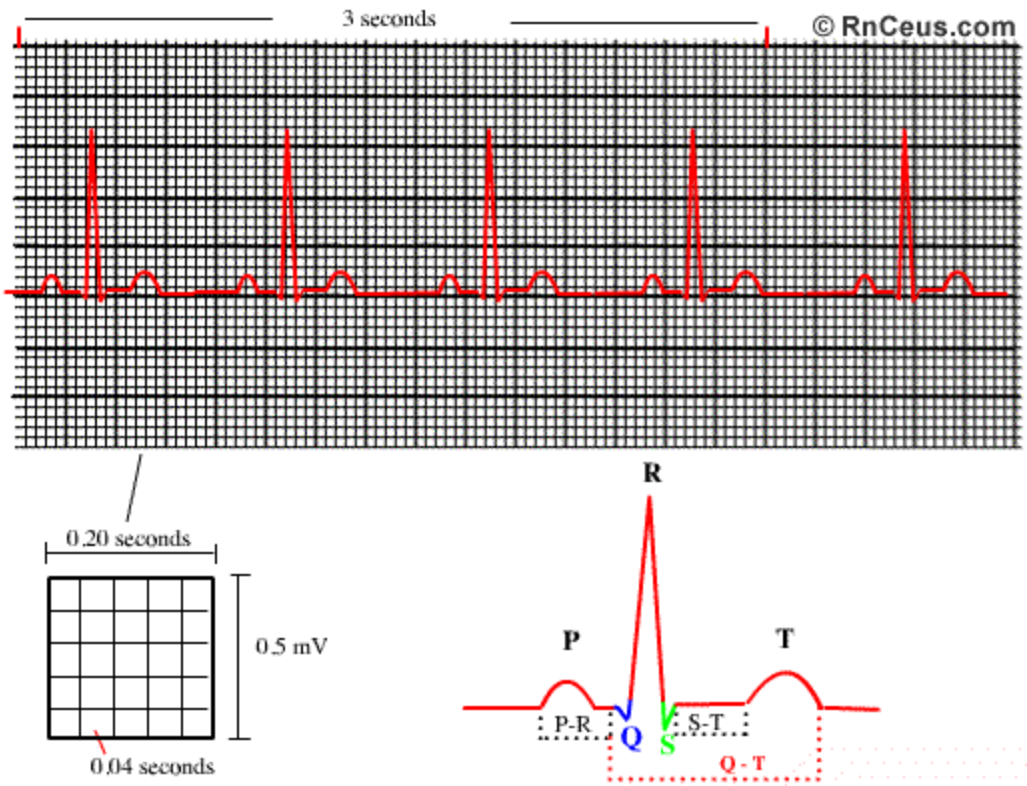
How to Read an EKG Strip

EKG paper is a grid where time is measured along the horizontal axis.

- Each small square is 1 mm in length and represents 0.04 seconds.
- Each larger square is 5 mm in length and represents 0.2 seconds.

Voltage is measured along the vertical axis.

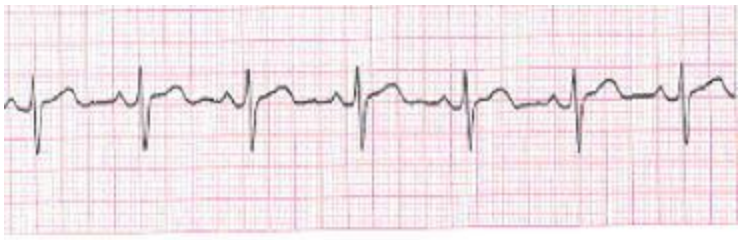
- 10 mm is equal to 1mV in voltage.
- The diagram below illustrates the configuration of EKG graph paper and where to measure the components of the EKG wave form

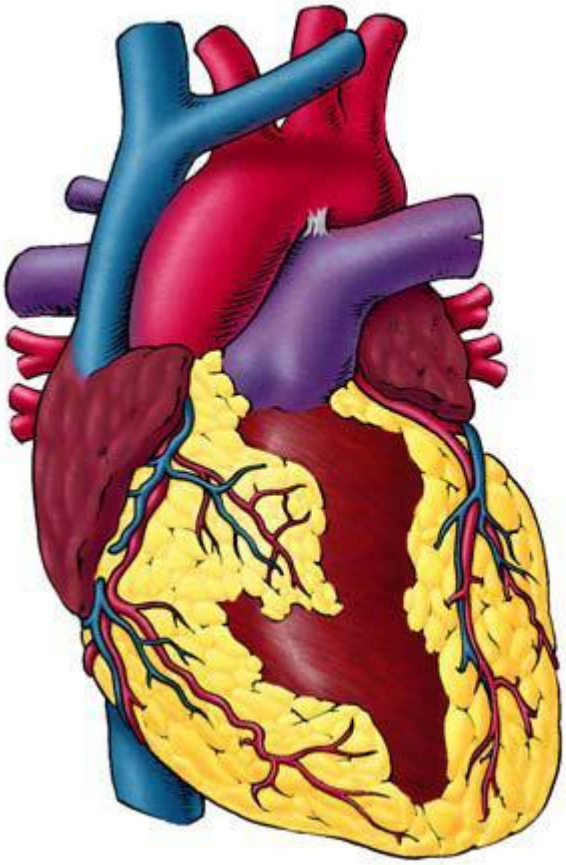


Heart rate can be easily calculated from the EKG strip:

- When the rhythm is regular, the heart rate is 300 divided by the number of large squares between the QRS complexes.
    - For example, if there are 4 large squares between regular QRS complexes, the heart rate is 75 (300/4=75).
- The second method can be used with an irregular rhythm to estimate the rate. Count the number of R waves in a 6 second strip and multiply by 10.
    - For example, if there are 7 R waves in a 6 second strip, the heart rate is 70 (7×10=70).
    -
    - ***Understanding ECG's***

This is a EKG Strip





Let’s look a the heart.

Tracing the blood flow is as follows;

1. Blood enters the superior and inferior vena cava and flows into the right atrium

2. The right atrium contracts and the blood flows through the right atrioventriular valve or also called the (Tricuspid Valve) into the right ventricle.

3. Next the right ventricle contracts and blood flows through the pulmonic valve into the pulmonary artery and travels to the lungs where the blood picks up oxygen.

4. Now the oxygenated blood flows back to the heart via the pulmonary vein into the left atrium.

5. The left atrium contracts and freshly oxygenated blood flows through the left atrioventricular valve, or also called the (bicuspid valve) into the left ventricle.

6. The left ventricle contracts and pushes the fresh blood through the semi lunar valve or the aortic valve into the aorta through the rest of the body.

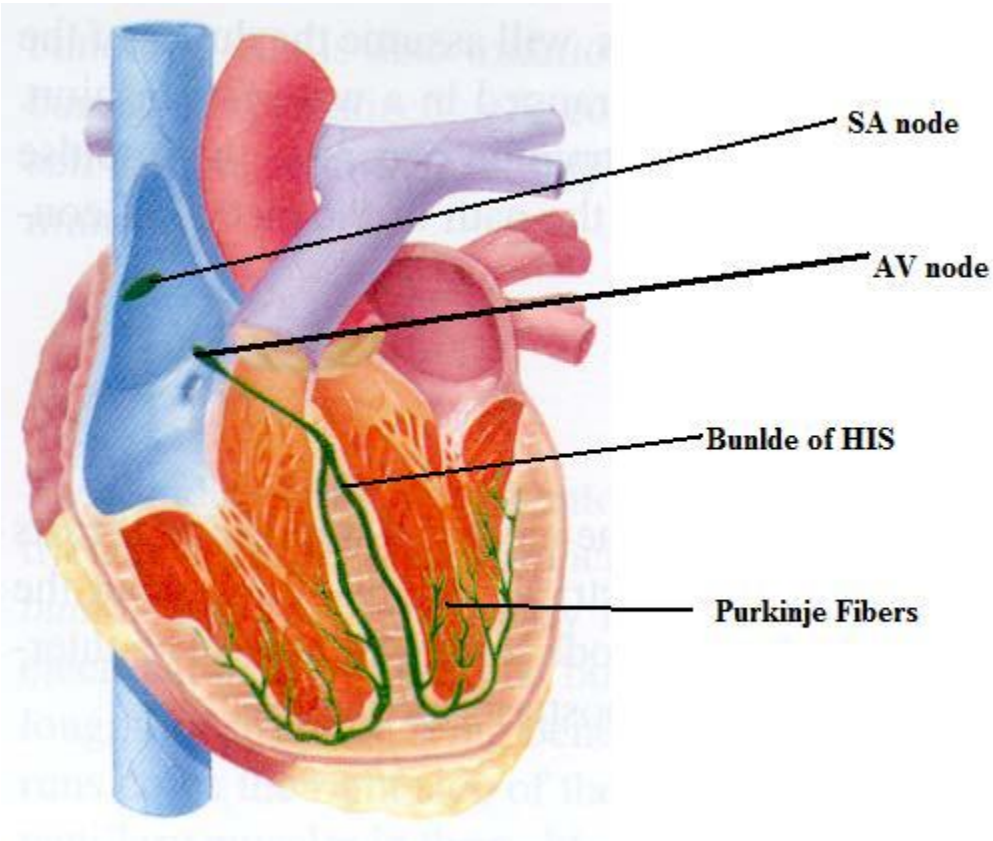
Objectives
1. Describe the basic approach to interpretation of ECG strips

2. Explain the five steps used in interpretation of ECG strips

3. Explain how to calculate heart rate, PRI, and QRS complex, given a 6 6-second strip

4. Identify different types of ECG strips pertaining to ACLS

The Electrical Conduction System



SA node: Fastest rate of automaticity automaticity. .
"Primary" pacemaker of the heart.
– Rate: 60 to 100 bpm

AV node: Has a delay which allows for atrial contraction and a more filling of the ventricles.
– Rate: 40 40-60 bpm

Bundle of His: Has the ability to self self-initiate electrical activity
– Rate: 40 40-60 bpm

Purkinje Fibers: Network of fibers that carry an electrical impulses directly to ventricular muscle cells.
– Rate: 20 20-40 bpm

Information Obtainable from ECG Rhythm Strip Analysis

1. Look /and measure the heart rate, is there one "Yes"

2. Check for Rhythm/regularlity

3. Check for Impulse conduction time intervals

4. Abnormal conduction pathways

5. Pumping action

6. Cardiac output

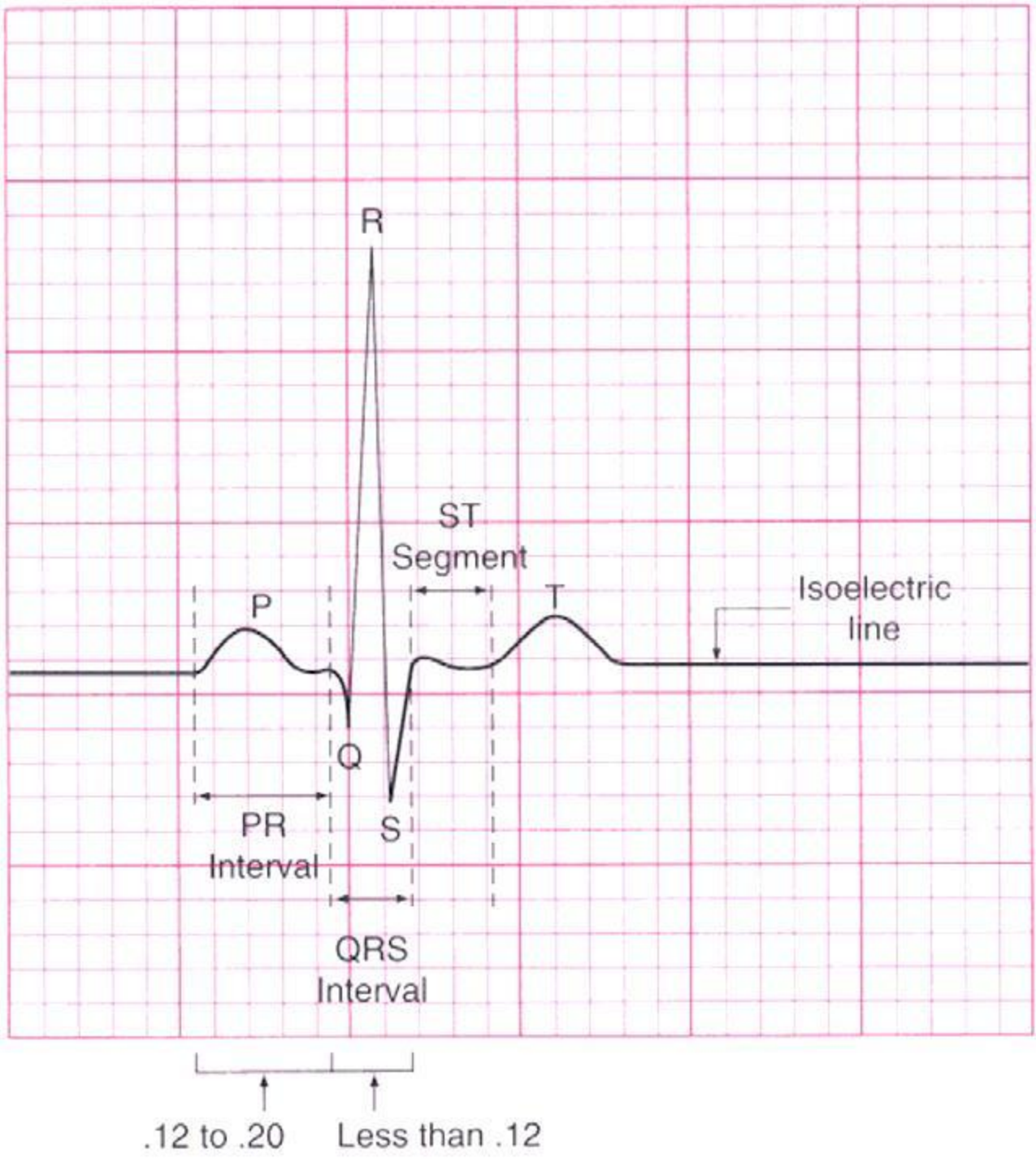
7. Blood pressure

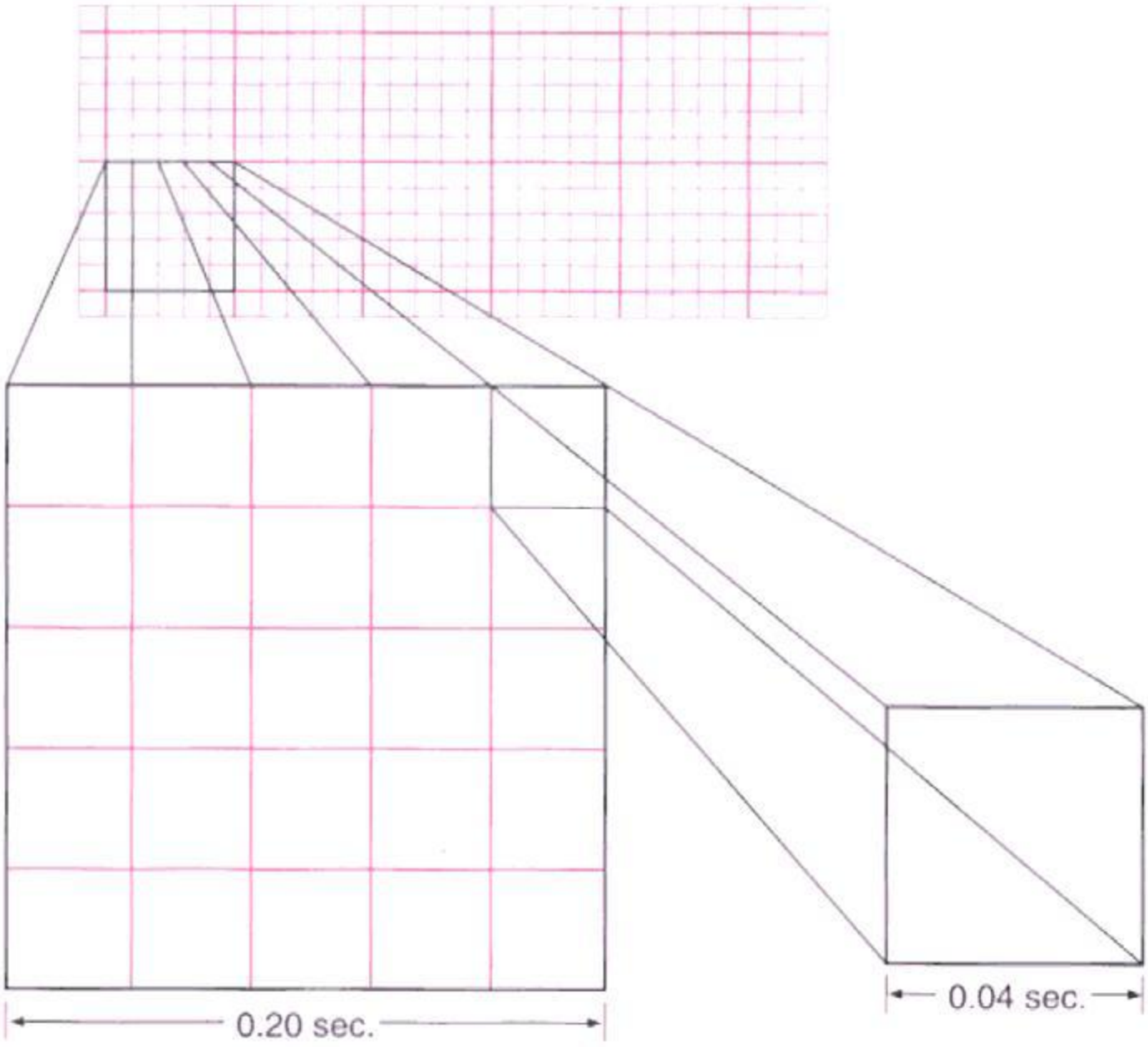
8. Cardiac muscle hypertophy

THE ELCETROCARDIOGRAM

A. defines the graphic representation of the electrical activity of the heart

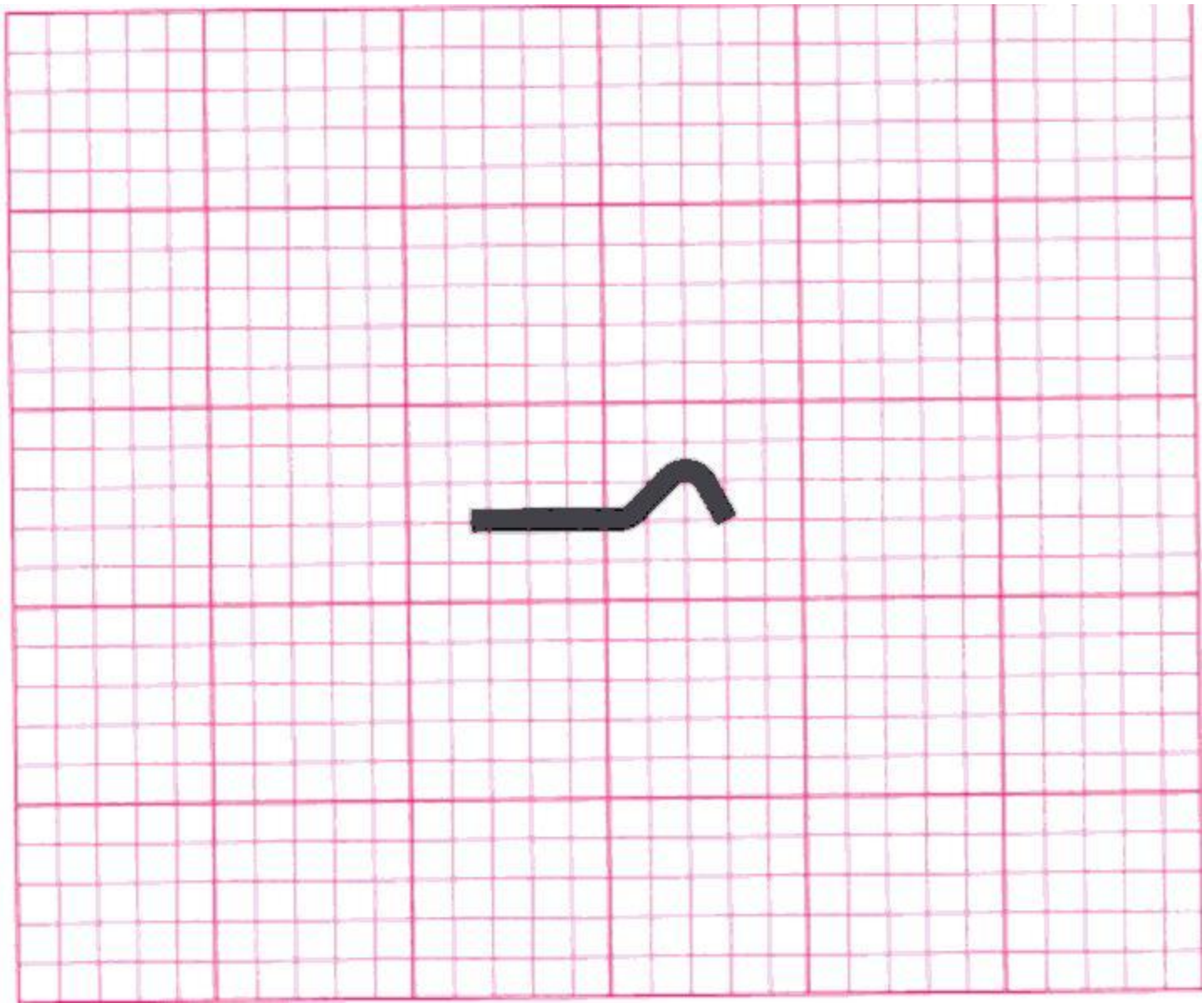
B. The printed record of the electrical activity of the heart is called a rhythm strip or an ECG strip.


R
ST
Segment
P
T
Isoelectric
line
Q
S
PR
Interval
QRS
Interval
.12 to .20
Less than .12

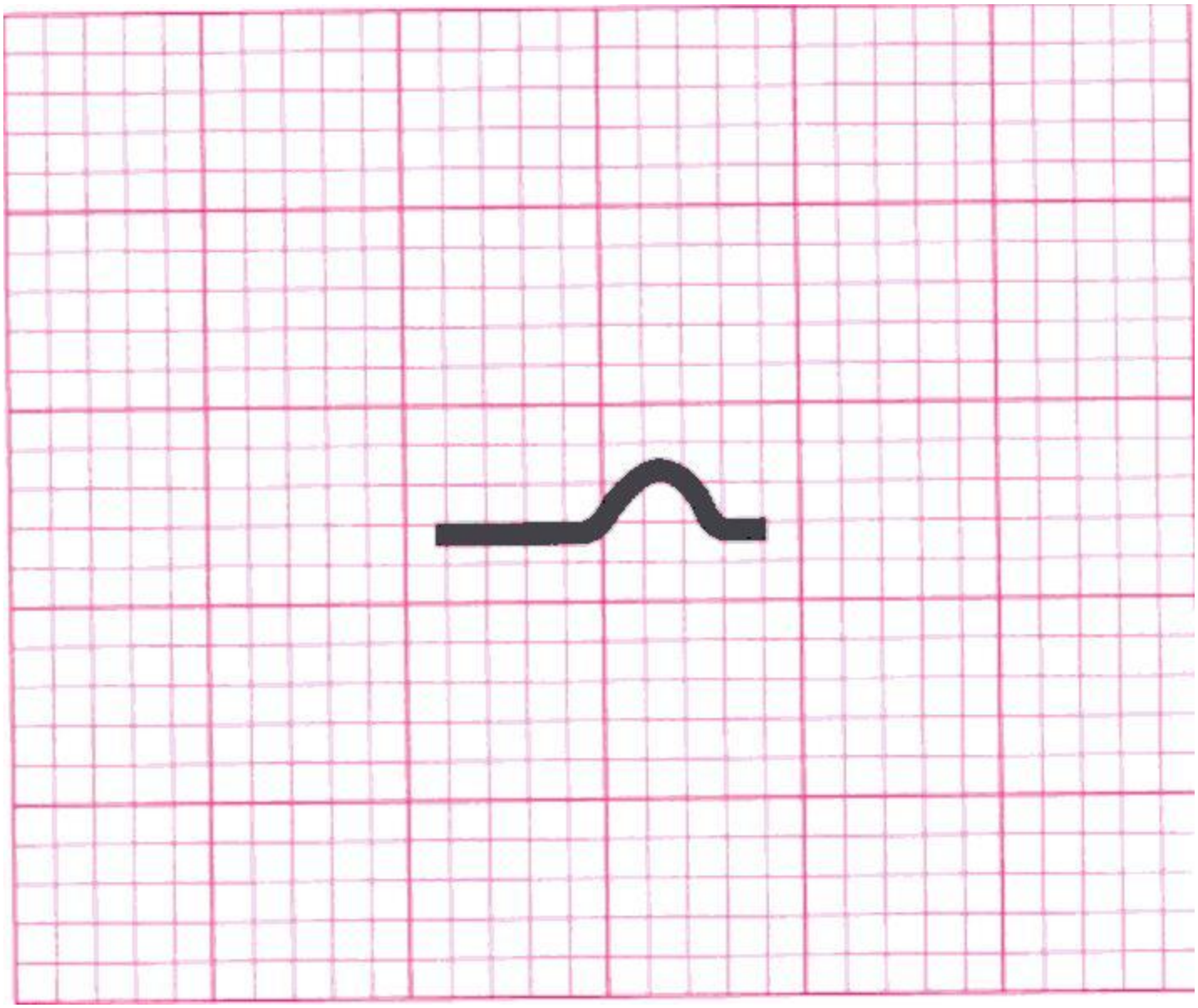


Breakdown of an ECG

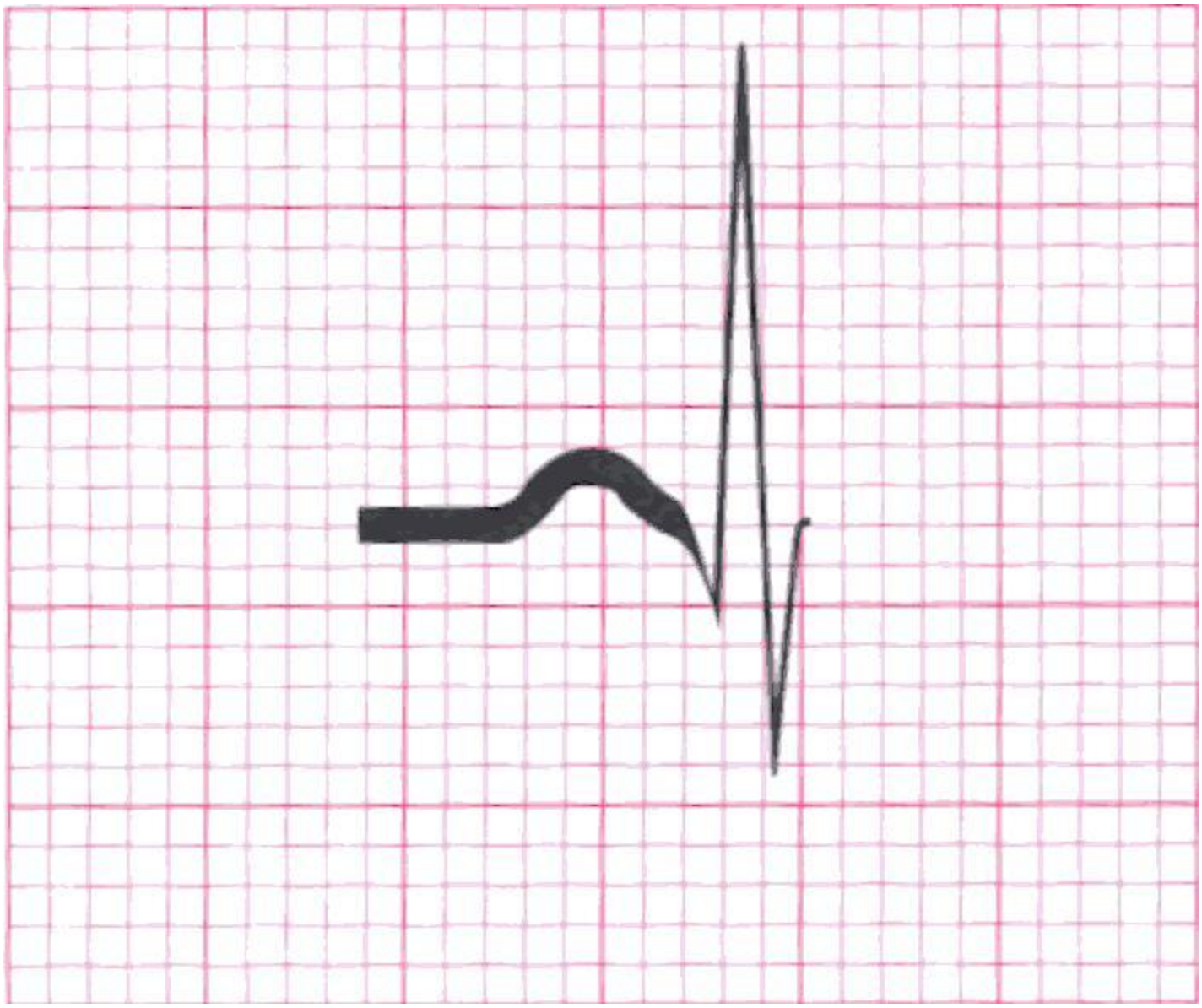
P-Wave
– SA node fires, sends the electrical impulse outward to stimulate both atria and manifests as a P
P-wave.
– Approximately 0.10 seconds in length



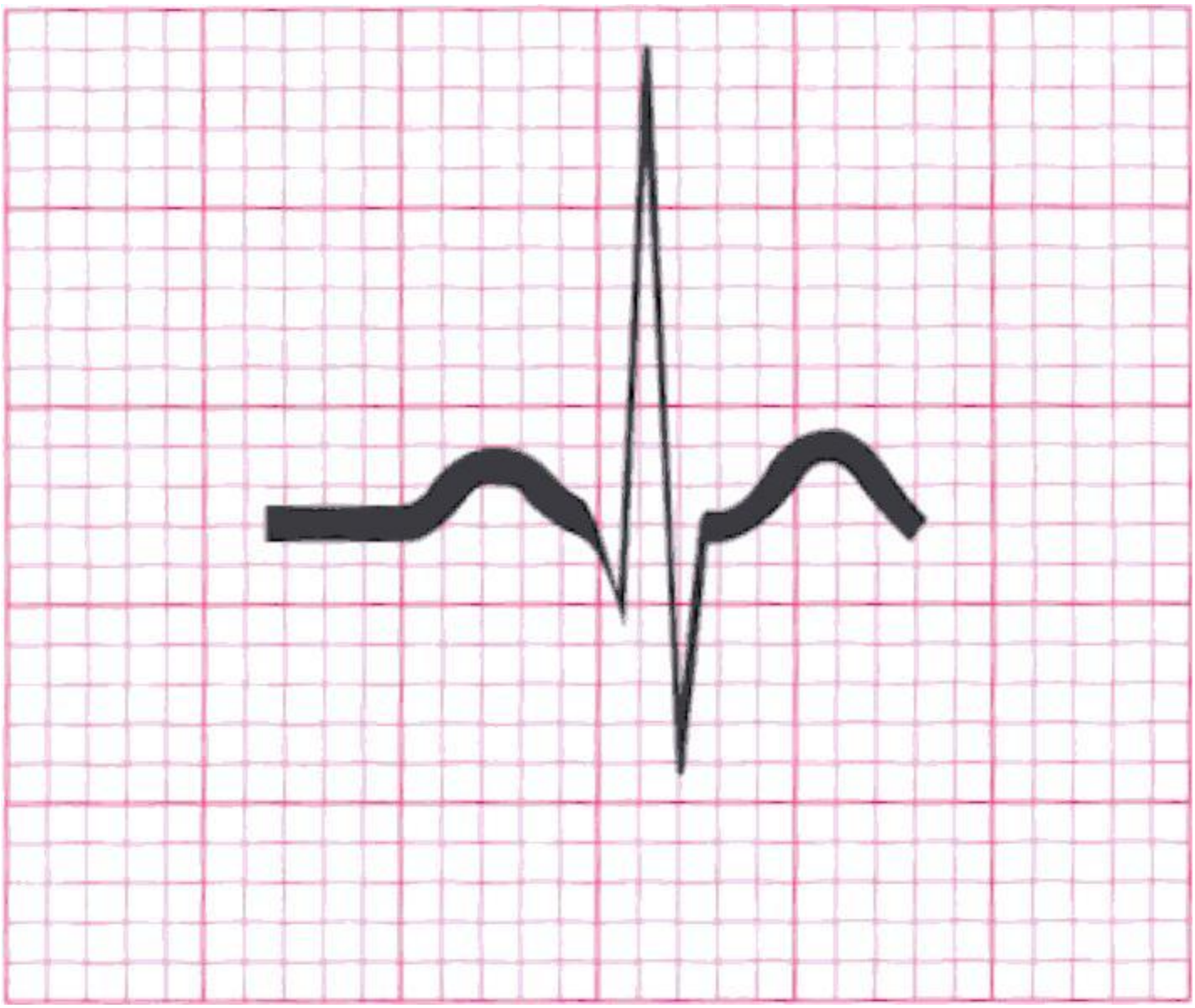
PR Interval (PRI) Time which impulse travels from the SA node to the atria and downward to the ventricles



QRS Complex Impulse from the Bundle of HIS throughout the ventricular muscles– Measures less than 0.12 seconds or less than 3 small squares on the ECG paper (see below)



T-Wave: Ventricular repolarization, meaning no associated activity of the ventricular muscle–
Resting phase of the cardiac cycle



Here are the steps to begin to interpret the ECG rhythm

Step 1: HEART RATE

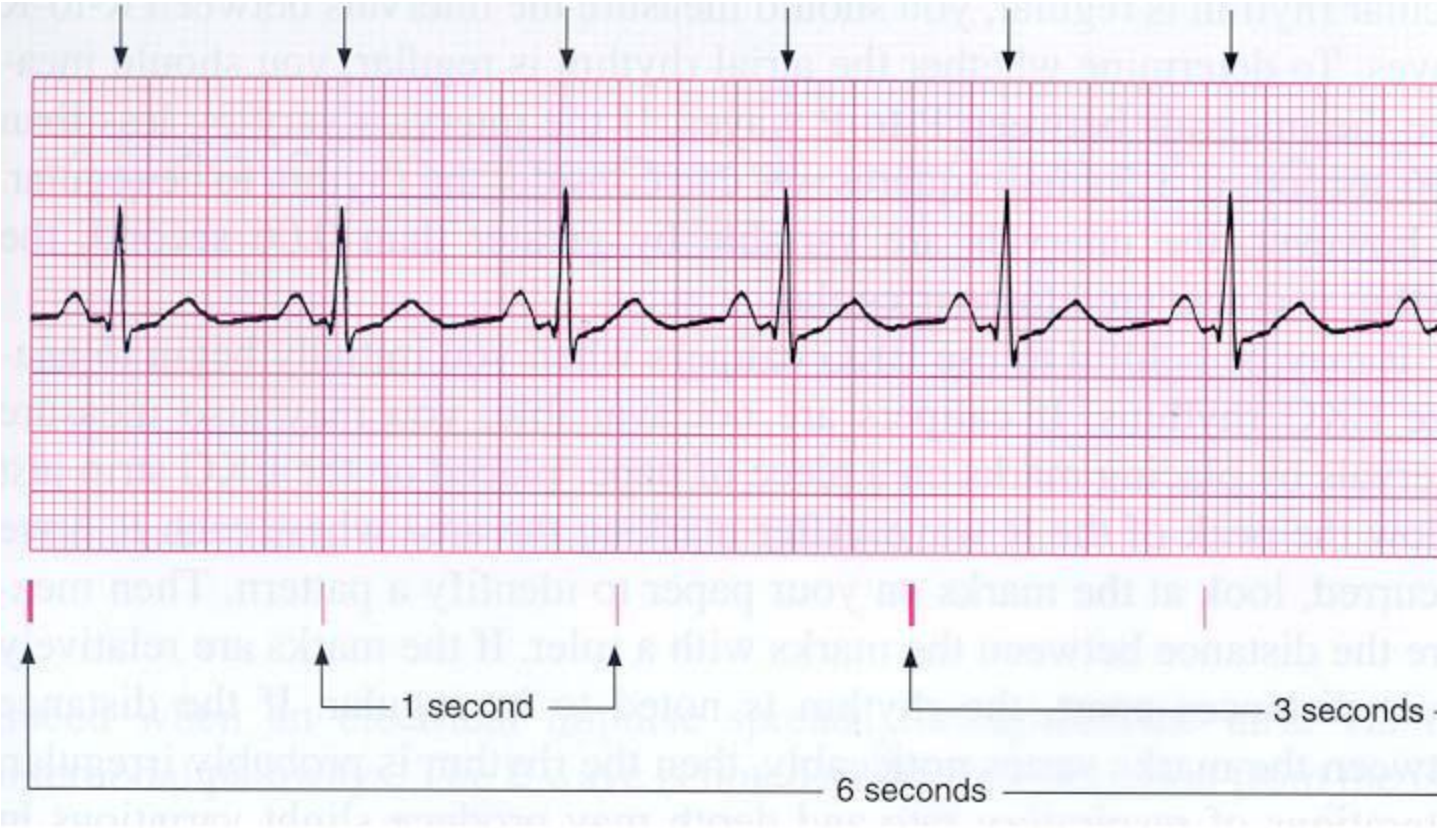
Step 2: HEART RHYTHM

Step 3: P- WAVE

Step 4: PR, or PRI interval

Step 5: QRS COMPLEX

Let's practice the next strip below



Asystole Treatment

There is no evidence to date that routine use of any vasopressor at any stage during management of pulseless VT, VF or asystole increase rtes of survival to hospital discharge. But there is evidence that the use of vasopressor favor initial resuscitation with ROSC.
Vasopressors optimize cardiac output and blood pressure. The vasopressors used during cardiac arrest are:
1. Epinephrine: 1mg IV/IO (repeat every 3-5 minutes)
2. Vasopressin: 1 dose of 40 U IV/IO may replace either the first or second dose of epinephrine.
3. If IV/IO access cannot be established or is delayed, give epinephrine 1:1000, 2 to 2.5 mg diluted in 5-10 mL of water or normal saline and injected directly into the ET tube. Remember, the ET route of drug administration results in variable and unpredictable drug absorption and blood levels.

Epinephrine: Although epinephrine has been used for year in resuscitation. There is little data to show that it improves outcome in humans. No studies have found improved rates of survival to hospital discharge or neurologic outcome when initial or escalating high-dose epinephrine was compared with standard doses. Therefore, the routine use of high dose or escalating doses of epinephrine cannot be recommended.

Repeat epinephrine 1 mg IV/IO every 3 to 5 minutes during cardiac arrest.
Vasopressin: Vasopressin is a nonadrenergic peripheral vasoconstrictor that causes coronary and renal vasoconstriction. Because the efficacy of vasopressin is not different than that of

epinephrine in cardiac arrest, a single dose of vasopressin (40 U IV/IO) may replace either the first or second close of epinephrine.

Antiarrhythmic Agents; When VF/pulseless VT persists after a total of 2 or 3 shocks, separated by cycles or CPR and administration of a vasopressor, consider use of one of the following antiarrhythmics;
a. Amiodarone
b. Lidocain
c. Magnesium Sulfate
These agent can also be used for maintenance therapy in the postresuscitation period.
Amiodarone: Consider amiodarone for treatment of VF or pulseless VT unresponsive to shock delivery, CPR, and vasopressor. Although there is no evidence that giving any antiarrhythmic routinely during cardiac arrest increases rates of survival to hospital discharge, amiodarone has been shown to increase rates of survival to hospital admission (ie., short-term survival compared with placebo or lidocaine.]

Amiodarone is a complex drug that affects sodium, potassium, and calcium channels. It also has alpha adrenergic and beta adrenergic blocking properties. Amiodarone is available in vial and prefilled syringes. During cardiac arrest consider amiodarone 300mg IV/IO push for the first dose. If VF/pulseless VT persists, consider giving a second dose of 150 mg IV/IO in 3 to 5 minutes.
Lidocaine is an alternative antiarrhythmic of long standing and widespread familiarity. However, it has no proven short term or long term efficacy in cardiac arrest. Lidocaine is included as an alternative to amiodarone in setting where amiodarone is not available.
Give lidocaine in a dose of 1-1.5 mg/kg IV/IO. Repeat if indicated at 0.5 to 0.75 mg/kg IV/IO over 5-10 minute intervals to a maximum of 3 doses or 3mg/kg. If no IV/IO access is available, then the dose for ET administration is 2-4 mg/kg.

Magnesium Sulfate: IV magnesium may terminate or prevent recurrent torsades de pointes in patients who have a prolonged QT interval during normal sinus rhythm. When VF/pulseless VT cardiac arrest is associated with torsades de pointes, give magnesium sulfate at a loading dose or 1-2 g IV/IO diluted in 10mL D5W given over 5-20 minutes. If a prearrest 12 lead ECG is available for review, check the QT interval for prolongation. Remember that pulseless VT is treated with an immediate high energy shock, whereas magnesium is an adjunctive agent used to prevent recurrent or treat persistent VT associated with torsades de pointes.
Magnesium slufate is also indicated for patients with known or suspected low serum magnesium, such as patients with alcoholism or other condiditon associated with malnutrition or hyomagnesemic states.
Frequent causes of PEA (H’s and T’s)

Frequent causes of PEA (H’s and T’s)

CONDITION	EKG TRACING	H &P CLUES	TREATMENT	
HYPOVOLEMIA	NARROW COMPLEX/RAPID RATE	HISTORY/FLAT NECK VEINS	VOLUME INFUSION	

HYPOXIA	**SLOW RATE**	**CYANOSIS, BLOOD GASES, AIRWAY PROBLEMS**	**OXYGENATION, VENTILATION**	
HYDROGEN ION ACIDOSIS]	**SAMLLER AMPLITUDE QRS COMPLEXES**	**HX OF DIABETES, BICARBONATE RESPONSIVE PREEXISTING ACIDOSIS, RENAL FAILURE**	**SODIUM BICARBONATE HYPERVENTILATION**	
HYPERKALEMIA	**BOTH HYPERKALEMIA AND HYPOKALEMIA CAUSE WIDE COMPLEX QRS** **HIGH POTASSIUM ECT.** **T WAVES TLLER AND PEAKED** **P WAVES GET SMALLER** **QRS WIDENS** **SINE WAVE PEA**	**HX OF REANL FAILURE, DIABETES, RECENT DIALYSIS, DIALYSIS FISTURLAS, MEDICATIONS**	**SODIUM BICARBONATE** **GLUCOSE PLUS INSULIN** **CALCIUM CHLORIDE** **KAYEXALATE/SORBITAL** **DIALYSIS LONG TERM** **POSSIBLY ALBUTERAL**	
HYPOKALEMIA	**LOW POTASSIUM** **T WAVES FLATTEN** **PROMINENT U WAVES** **ORS WIDENS** **QT PROLONGS** **WIDE COMPLEX TACHYCARDIA**	**ABNORMAL LOSS OF POTASSIUM DIURETIC USE**	**HYPOKALEMIA** **RAPID BUT CONTROLLED INFUSION OF POTASSIUM** **ADD MAGNESIUM IF CARDIAC ARREST**	
HYPOTHERMIA	**J OR OSBORNE**	**HX OF**	**SEE HYPOTHERMIA**	

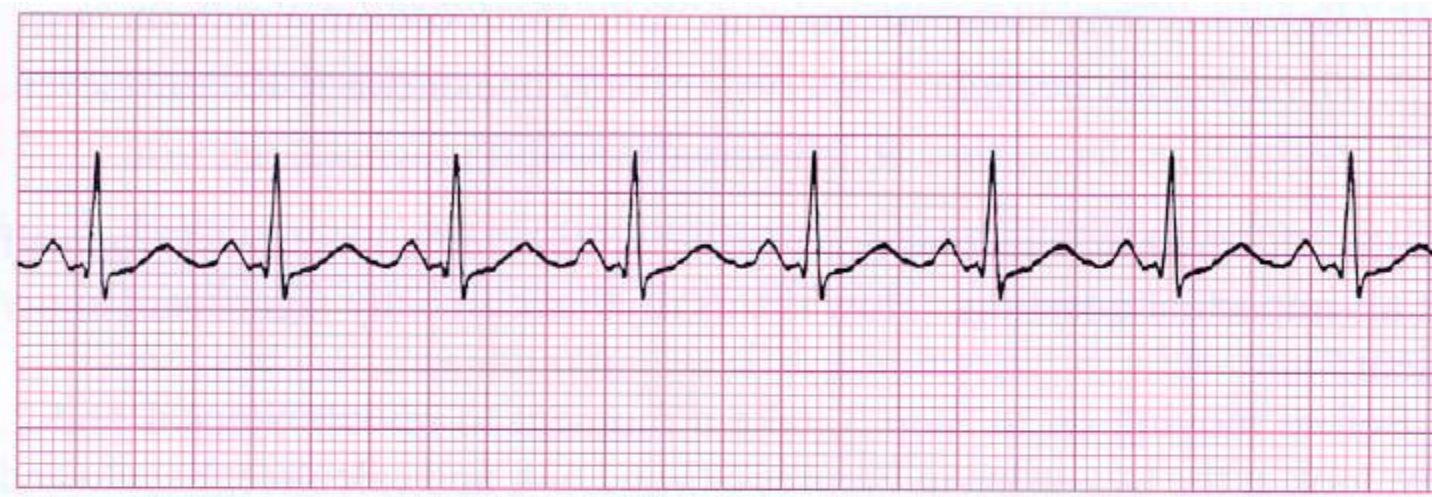
	WAVES	**EXPOSURE TO COLD, CENTRAL BODY TEMPERATURE**	**ALGORITHM**	
TABLET DRUG OVERDOSE, TRICYCLICS, DIGOXIN, BETA BLOCKERS, CALCIUM CHANNEL BLOCKERS	**VARIOUS EFFECTS ON ECG PREDOMINATELY PROLONGATION OF QT INTERVAL**	**BRADYCARDIA, EMPTY BOTTLES AT THE SCENE, PUPILS, NEUROLOGIC EXAM**	**DRUG SCREENS, INTUBATION, LAVAGE, ACTIVATED CHARCOAL, LACTULOSE PER LOCAL PROTOCOLS, SPECIFIC ANTIDOTES AND AGENTS PER TOXIDROME**	

TABLET DRUG OVERDOSE, TRICYCLICS, DIGOXIN, BETA BLOCKERS, CALCIUM CHANNEL BLOCKERS	**VARIOUS EFFECTS ON ECG PREDOMINATELY PROLONGATION OF QT INTERVAL**	**BRADYCARDIA, EMPTY BOTTLES AT THE SCENE, PUPILS, NEUROLOGIC EXAM**	**DRUG SCREENS, INTUBATION, LAVAGE, ACTIVATED CHARCOAL, LACTULOSE PER LOCAL PROTOCOLS, SPECIFIC ANTIDOTES AND AGENTS PER TOXIDROME**	
CARDICA TAMPONADE	**NARROW COMPLEX/RAPID RATE**	**HX, NO PULSE FELT WITH CPR, VEIN DISTENTION**	**PERI CARDIO CENTESIS**	
TENSION PNEUMONTHORAX	**NARROW COMPLEX SLOW RATE HYPOXIA**	**HX, NO PULSE FELT WITH CPR, NECK VEIN DISTENTION, UNEQUAL BREATH SOUNDS, DIFFICULT TO VENTILATE PATIENT**	**NEEDLE DECOMPRESSION**	
THROMBOSIS HEART, ACUTE MASSIVE MI	**ABNORMAL 12 LEAD ECG** **Q WAVES** **ST SEGMENT**	**HX CARDIAC MARKERS**	**FIBRINOLYTIC AGESNT**	

	CHANGES **T WAVES INVERSIONS**			
THROMBOSIS LUNGS, MASSIVE PULMONARY EMBOLISM	**NARROW COMPLEX RAPID RATE**	**HX NO PULSE FELT WITH CPR, DISTENDED NECK VEINS PRIOR POSITIVE TEST FOR DVT OR PE**	**SURGICAL EMBOLECTOMY, FIBRINOLYTICS**	

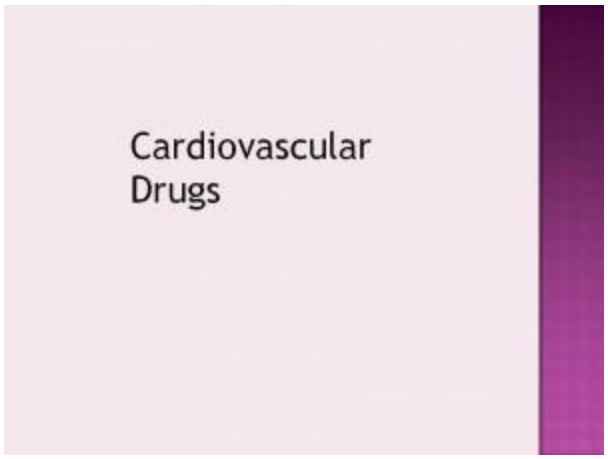
Look at the strip. To calculate the heart rate you can use the 6-Second Method: Have a six second strip, count the
QRS complexes and multiple by 10.

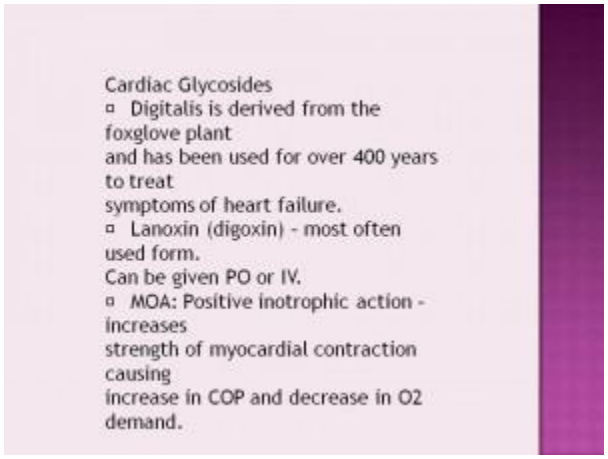
QUESTION 1. FOR THE STRIP BELOW TRY TO CALCULATE THE HEART RHYTHM



What is the heart rhythm on this strip?

Answer: _____





Cardiac Glycosides

Digitalis is derived from the foxglove plant and has been used for over 400 years to treat symptoms of heart failure.

Lanoxin (digoxin) – most often used form.

Can be given PO or IV.

MOA: Positive inotrophic action – increases strength of myocardial contraction causing increase in COP and decrease in O2 demand.

Lanoxin

MOA: 1. Positive inotrophic action – increases strength of myocardial contraction causing increase in COP and decrease in O2 demand. 2. Negative chronotropic action – decreases HR by decreasing impulse formation in the SA node. It indirectly stimulates the vagus nerve. Together these actions lead to a decrease in compensatory tachycardia (sympathetic nervous system).

3. Negative dronotrophic action – slowed conduction of impulses through the AV node

HEART RHYTHM;

Heart rhythms are classified as regular or irregular. Calculating the heart rhythm involves establishing a pattern of QRS complexes as they occur.

Measure ventricular rhythm by measuring the interval between R- to -R waves and atrial rhythm by measuring the P-to-P waves

Interval > than 0.06 seconds implies irregular

Now let's look at the P-WAVE

There are 5 questions that you should ask as you interpret a strip.

1. Are P-Waves present?

2. Are P-Waves occurring regularly?

3. Is there a P-Wave for each ORS compex?

4. Are the P-Waves smooth, rounded, and upright in appearance, or are they inverted?

5. Do all P-Waves look similar?

The PR or PRI Interval

The normal length of the PRI is 0.12 to 0.20 seconds or (3-5 small squares)

There are 3 questions to ask:

1. Are the PR/PRI intervals greater than 0.02 seconds?

2. Are the PR/PRI's less than 0.12 seconds?

3. Are the PR/PRI's constant across the ECG strip?

THE QRS COMPLEX

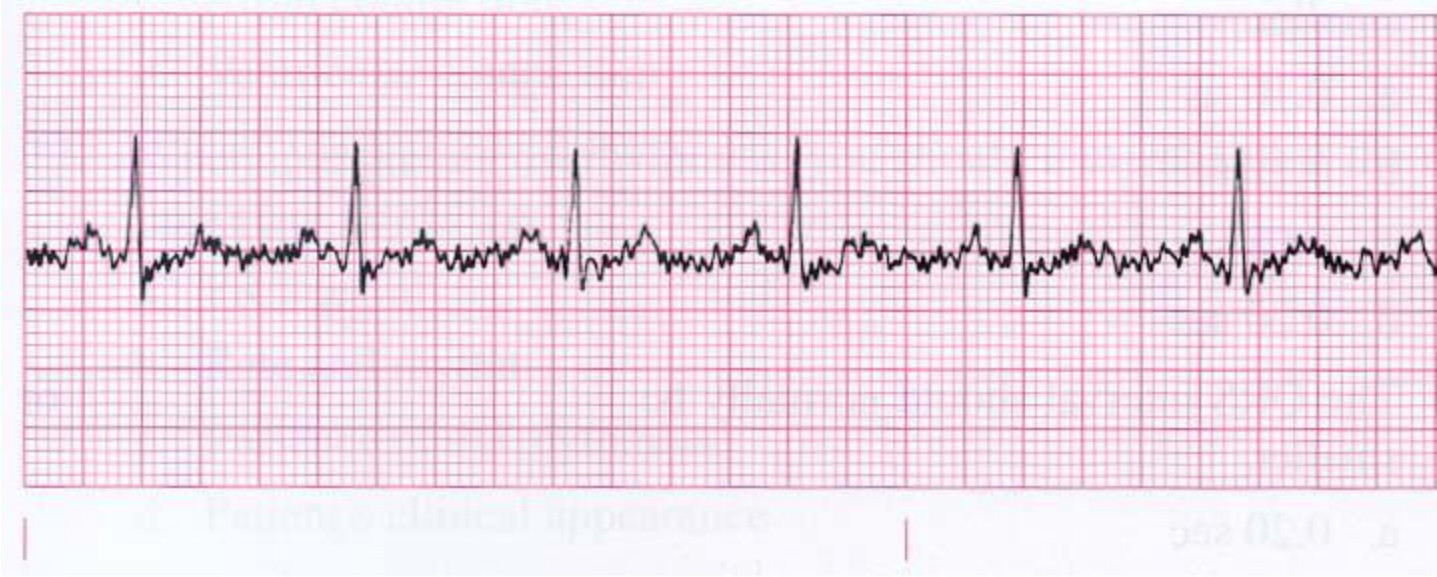
3 Questions to ask:

1. Are the ORS intervals greater than 0.12 seconds (wide)? If so, the complex may be ventricular in origin.

2. Are the ORS intervals less than 0.12 seconds (narrow)? If so, the complex is most likely suprventricular in origin.

3. Are the ORS complexes similar in appearance across the ECG strip?

NOW LET'S IDENTIFY YOUR FIRST RHYTHM STRIP



STEP 1: HEART RATE

STEP 2: HEART RHYTHM

STEP 3: P-WAVE

STEP 4: PR

STEP 5: QRS COMPLEX

ANSWER: If this strip seems confusing, don't worry. This is called Artifact. You will see this if the following reasons are present;

patient movement

loose or defective electrodes

improper grounding

faulty ECG apparatus

PATIENT ASSESSMENT IS CRITICAL!!!!

ALRIGHT, ENOUGH PLAYING, LET'S TAKE A LOOK AT THE TYPES OF RHYTHMS and interpretation

RATE:

Bradycardia = rate of < 60 bpm

Normal = rate of 60-100 bpm

Tachycardia = rate of > 100-160 bpm

WHERE ITS COMING FROM:

Sinus: SA node

Atrial; SA node fails, impulse comes from the atria (internodal or the AV node)

Ventricular: SA node of AV junction fails, ventricles will shoulder responsibility of pacing the heart.

SINUS RHYTHMS (these rhythms originate in the sino atrial node)

1. Normal Sinus Rhythm (NSR)

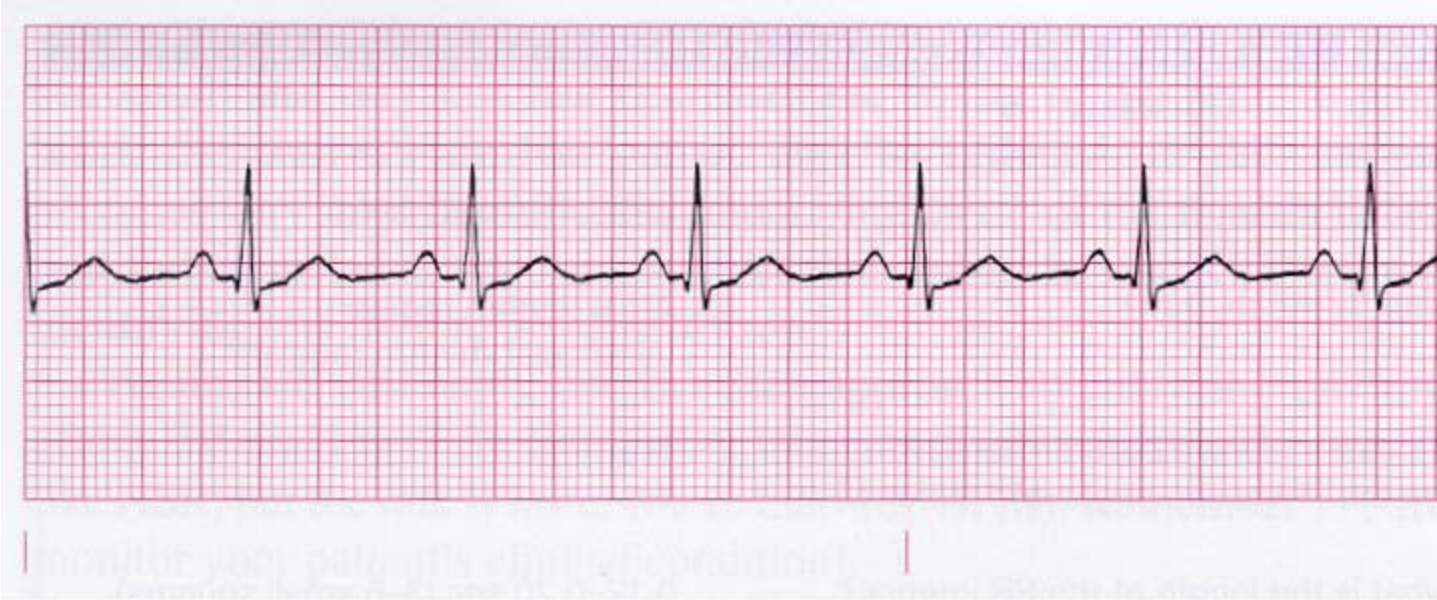
2. Sinus Bradycardia

3. Sinus Tachycardia

TABLE 7-3 NORMAL SINUS RHYTHM

Question 1–5

What is the rate?	60–100 BPM
What is the rhythm?	Atrial rhythm regular Ventricular rhythm regular
Is there a P wave before each QRS?	Yes
Are the P waves upright and uniform?	Yes
What is the length of the PR interval?	0.12–0.20 sec (3–5 small squares)
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is . . . ?	Less than 0.12 sec (3 small squares)

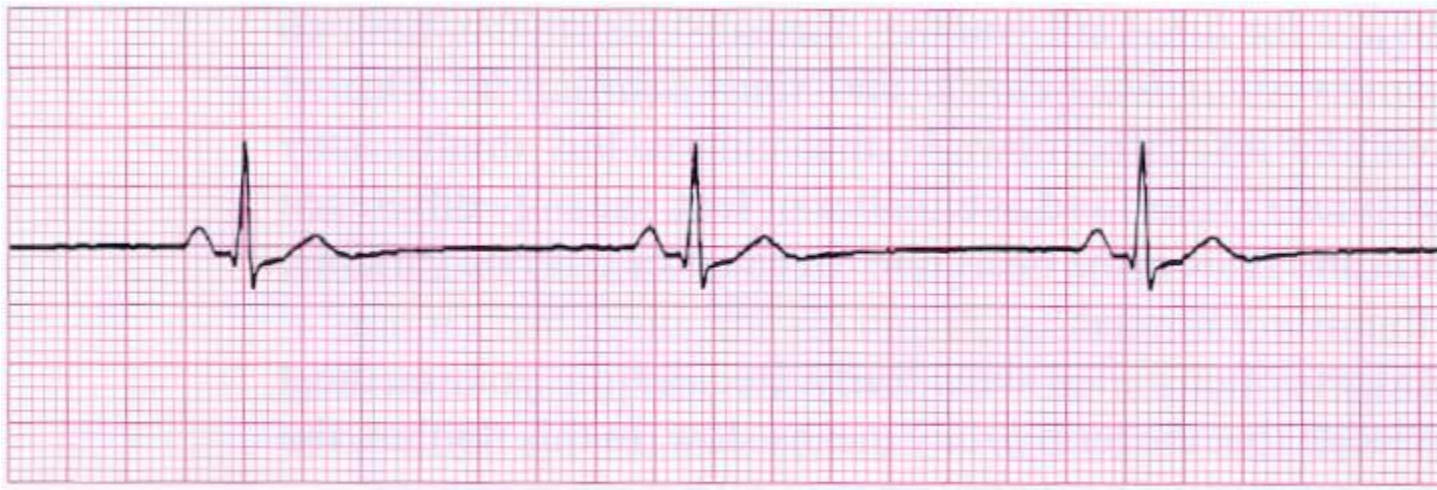


SINUS BRADYCARDIA RHYTHM

TABLE 7-4 SINUS BRADYCARDIA RHYTHM

Questions 1–5

What is the rate?	LESS THAN 60 BPM
What is the rhythm?	Atrial rhythm regular Ventricular rhythm regular
Is there a P wave before each QRS?	Yes
Are the P waves upright and uniform?	Yes
What is the length of the PR interval?	0.12–0.20 sec (3–5 small squares)
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is . . . ?	Less than 0.12 sec (3 small squares)

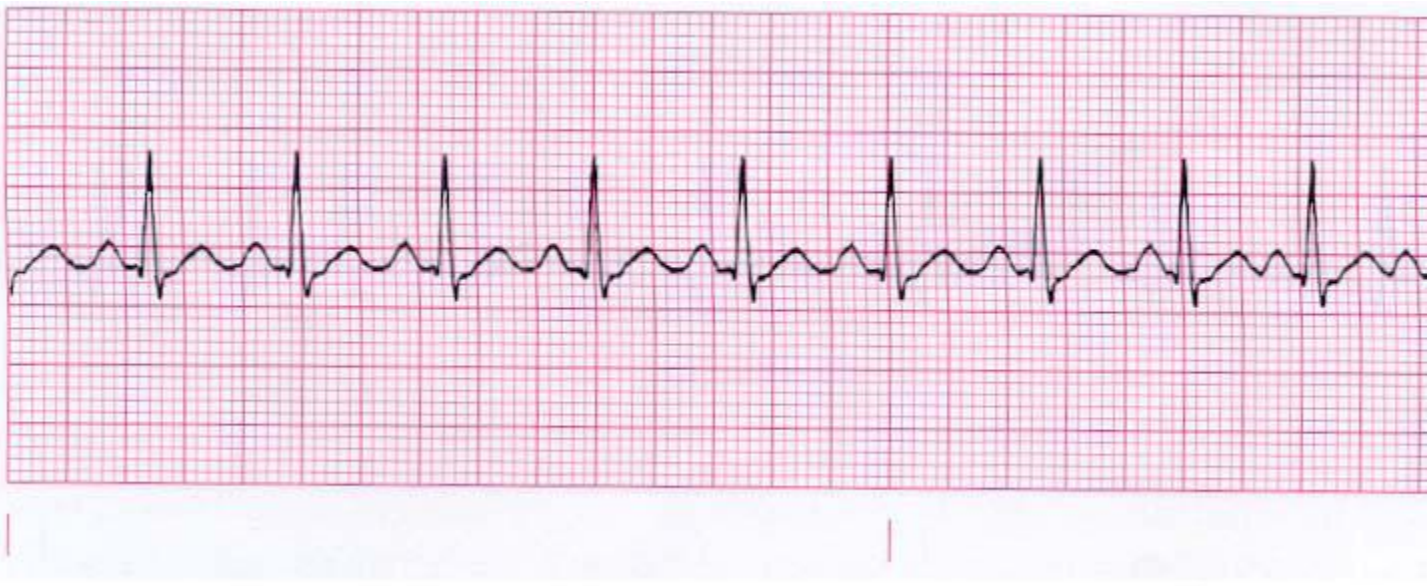


SINUS TACHYCARDIA RHYTHM

TABLE 7-5 SINUS TACHYCARDIA RHYTHM

Questions 1–5

What is the rate?	100–160 BPM
What is the rhythm?	Atrial rhythm regular Ventricular rhythm regular
Is there a P wave before each QRS?	Yes
Are the P waves upright and uniform?	Yes
What is the length of the PR interval?	0.12–0.20 sec (3–5 small squares)
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is . . . ?	Less than 0.12 sec (3 small squares)



ATRIAL RHYTHMS

SA node fails to generate an impulse, the atrial tissue or areas in the internodal pathways may initiate an impulse.

These are called atrial dysrhythmias

Generally not considered life-threatening or lethal careful and deliberate patient assessment must be continuous.

TYPES OF ATRIAL RHYTHMS

Atrial Flutter

Atrial Fibrillation

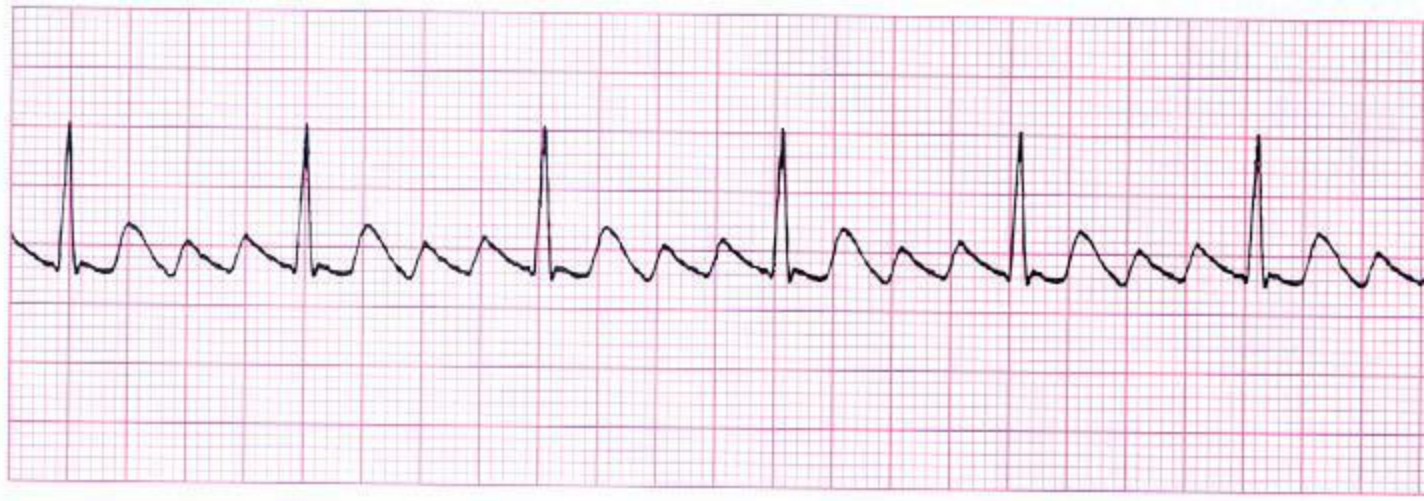
Supraventricular Tachycardia

ATRIAL FLUTTER TABLE

TABLE 8-3 ATRIAL FLUTTER

Questions 1–5

What is the rate?	Atrial—250–300 BPM Ventricular—variable
What is the rhythm?	Atrial—regular Ventricular—regular or irregular
Is there a P wave before each QRS? Are the P waves upright and uniform?	Normal P waves are absent; replaced by F waves (sawtooth)
What is the length of the PR interval?	Not measurable
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is?	Usually less than 0.12 sec (3 small squares)



ATRIAL FIBRILLATION

TABLE 8-4 ATRIAL FIBRILLATION

Questions 1–5

What is the rate?	Atrial—350–400 BPM Ventricular—variable
What is the rhythm?	Irregularly irregular
Is there a P wave before each QRS? Are the P waves upright and uniform?	Normal P waves are absent; replaced by f waves
What is the length of the PR interval?	Not discernable
Do all the QRS complexes look alike? The length of the QRS complexes is . . . ?	Yes Usually less than 0.12 sec

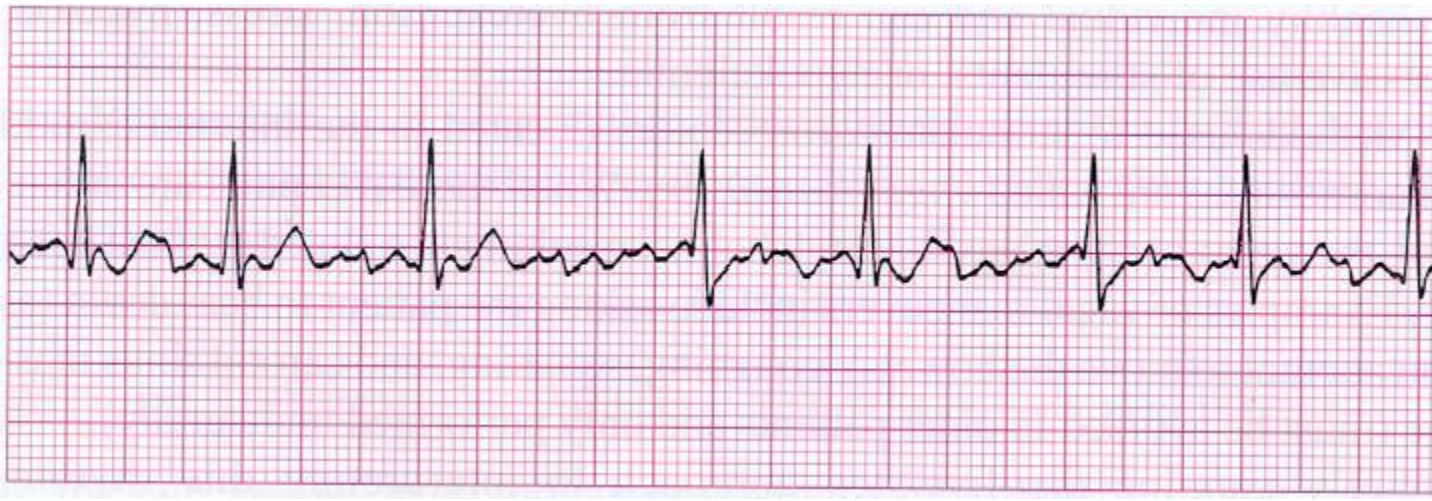
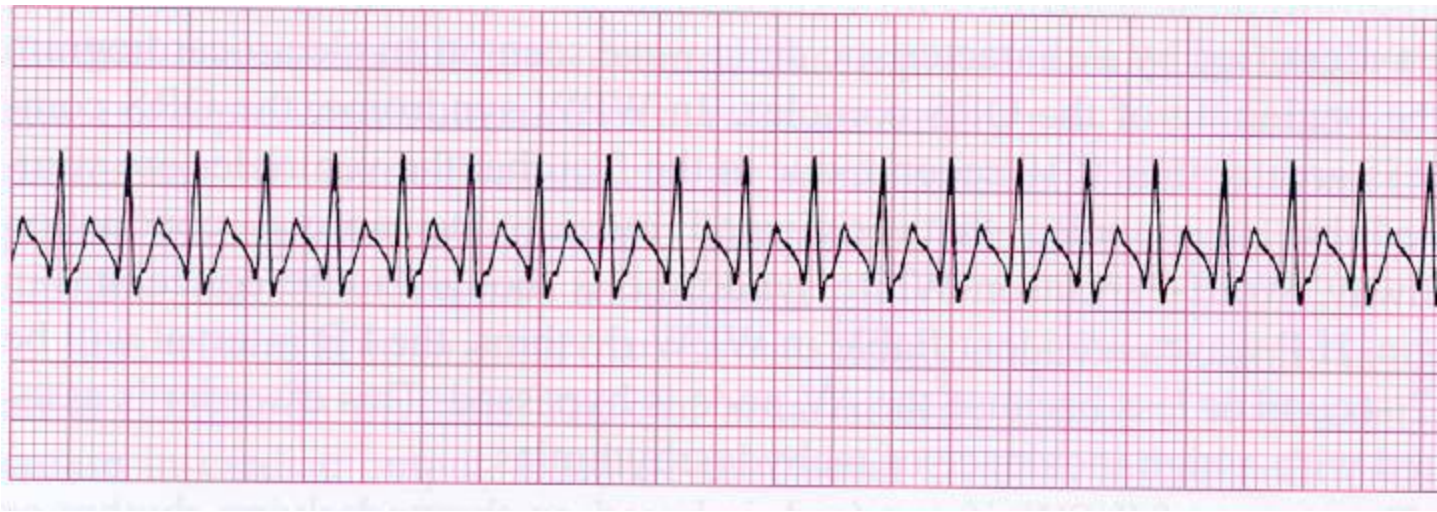


TABLE 8-5 SUPRAVENTRICULAR TACHYCARDIA

Questions 1–5

What is the rate?	Atrial—150–250 BPM Ventricular—150–250 BPM
What is the rhythm?	Regular
Is there a P wave before each QRS? Are the P waves upright and uniform?	Usually not discernable, especially at the high-rate range
What is the length of the PR interval?	Usually not discernable
Do all the QRS complexes look alike? The length of the QRS complexes is . . . ?	Yes Usually less than 0.12 sec



NOW LET US LOOK AT VENTRICULAR RHYTHMS

THE SA node or the AV junctional tissue fails to initiate an electrical impulse, the ventricles will shoulder the responsibility of pacing the heart.

This group of rhythms are called ventricular dysrhythmias.

An electrical impulse can be instigated from any pacemaker cell in the ventricles, incuding the bundle branches or the fibers of the Purkinje fibers.

TYPES OF VENTRICULAR RHYTHMS

1. PREMATURE VENTRICULAR COMPLEXES

2. VENTRICULAR TACHYCARDIA

3. TORSADES de POINTES

4. VENTRICULAR FIBRILLALTION

5. ASYSTOLE (FLAT LINE)

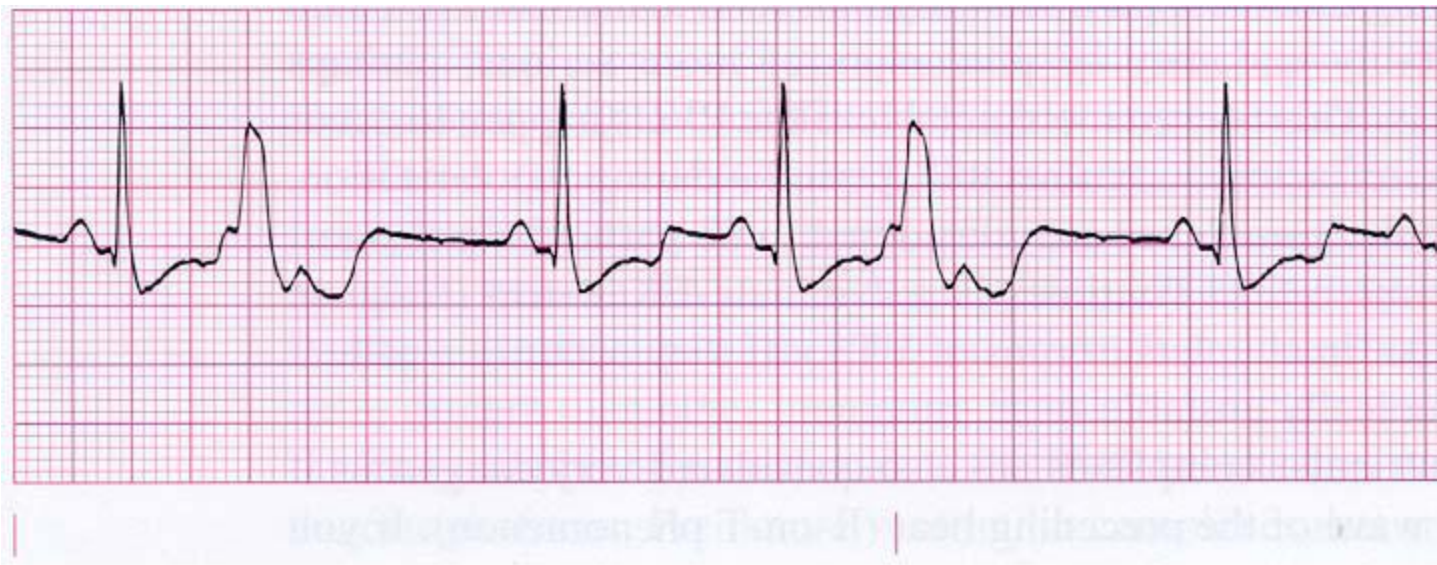
6. PULSELESS ELECTRICAL ACTIVITY (PEA)

PVC'S

TABLE 10-3 PREMATURE VENTRICULAR COMPLEXES

Questions 1–5

What is the rate?	Dependent on rate of underlying rhythm and number of PVCs
What is the rhythm?	Occasionally irregular; regular if interpolated PVC
Is there a P wave before each QRS? Are the P waves upright and uniform?	No P waves associated with PVC; P waves of underlying rhythm may be present
What is the length of the PR interval?	PRI not present with PVCs
The length of the QRS complexes is . . . ? What do the QRS complexes look like?	Greater than or equal to 0.12 sec (3 small squares); usually wide and bizarre



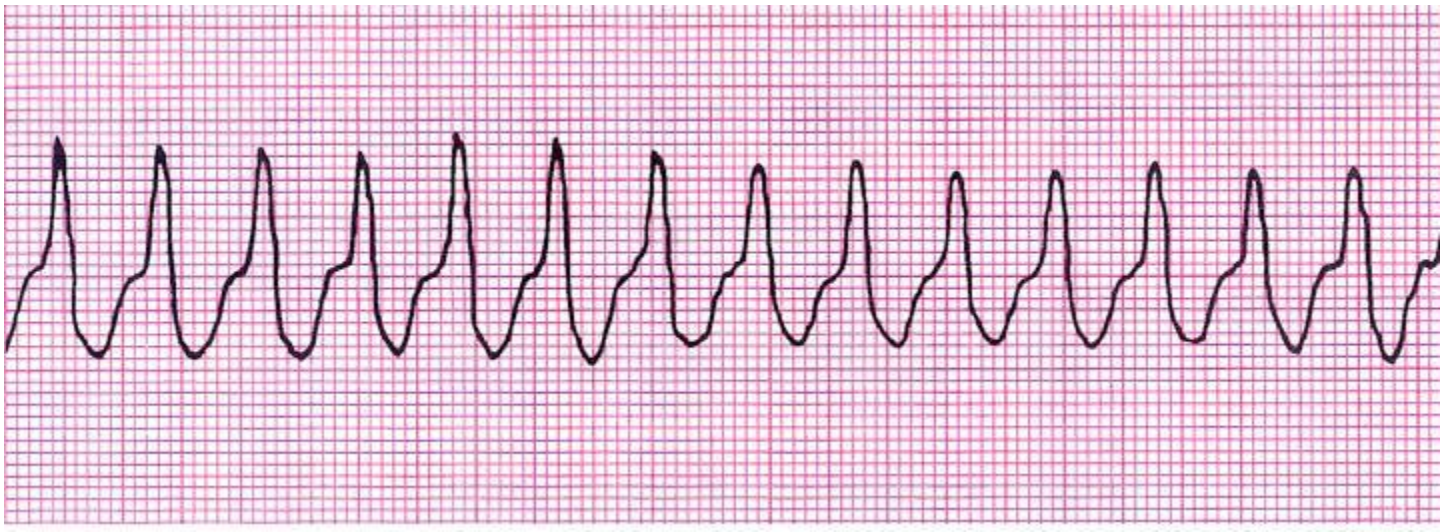
PVC STRIP

NOW LET'S LOOK AT VENTRICULAR TACHYCARDIA

TABLE 10-5 VENTRICULAR TACHYCARDIA RHYTHM

Questions 1–5

What is the rate?	100–250 BPM
What is the rhythm?	Atrial rhythm not distingui Ventricular rhythm usually
Is there a P wave before each QRS?	May be present or absent associated with QRS co
What is the length of the PR interval?	None
Do all the QRS complexes look alike?	Yes (except in torsades rh bizarre QRS morphology
The length of the QRS complexes is . . . ?	Greater than 0.12 sec



NEXT WE WILL LOOK AT TORSADES DE POINTES

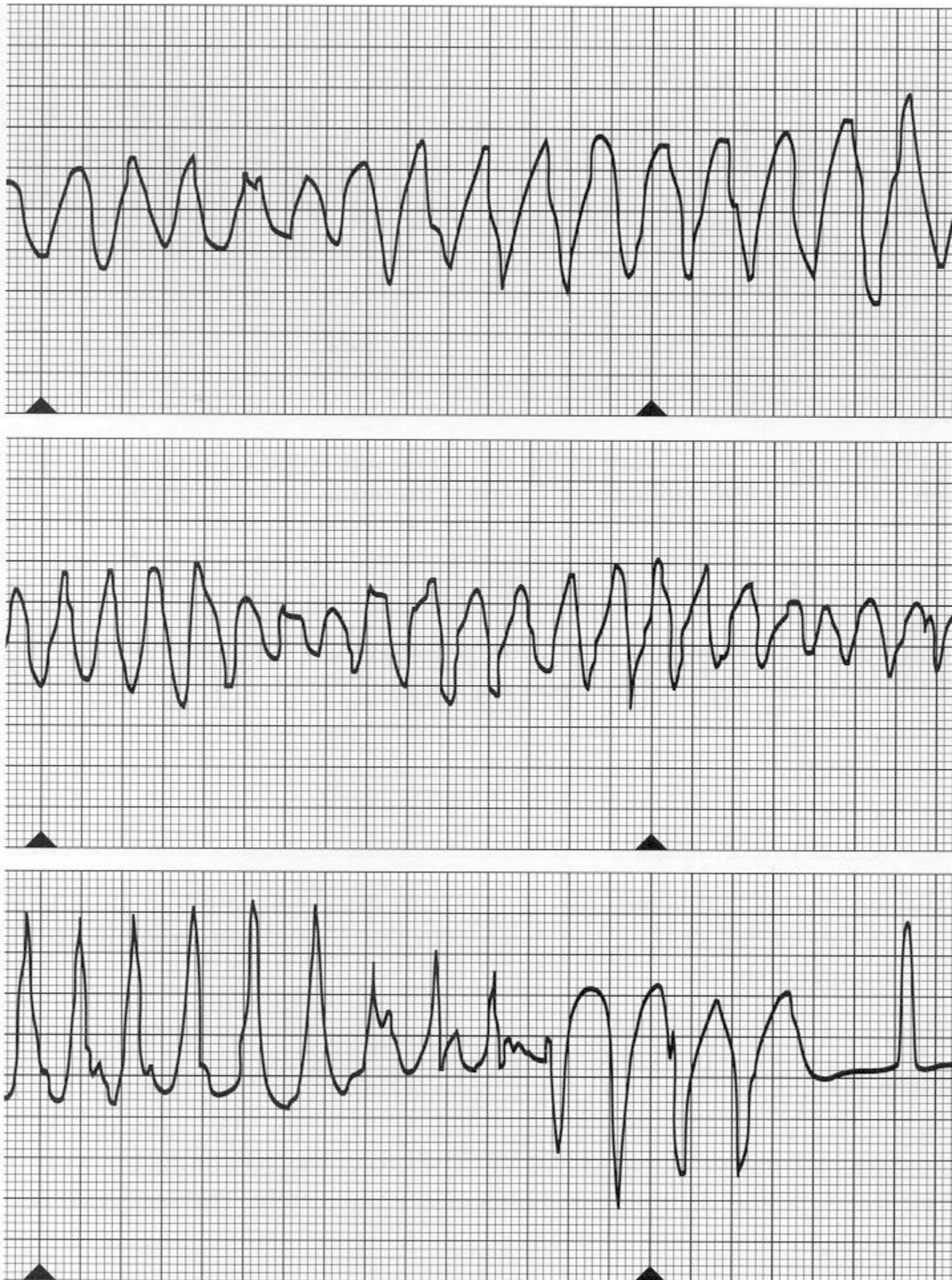
This is a French term that signifies the “twisting of the points”.

May wax and wane in amplitude and may “flip” or “twist” on its electrical axes.

Similar to ventricular tachycardia.

Caused by hypomagnesemia or by antiarrhythmic drugs.

BELOW IS AN EXAMPLE OF TORSADES de POINTES

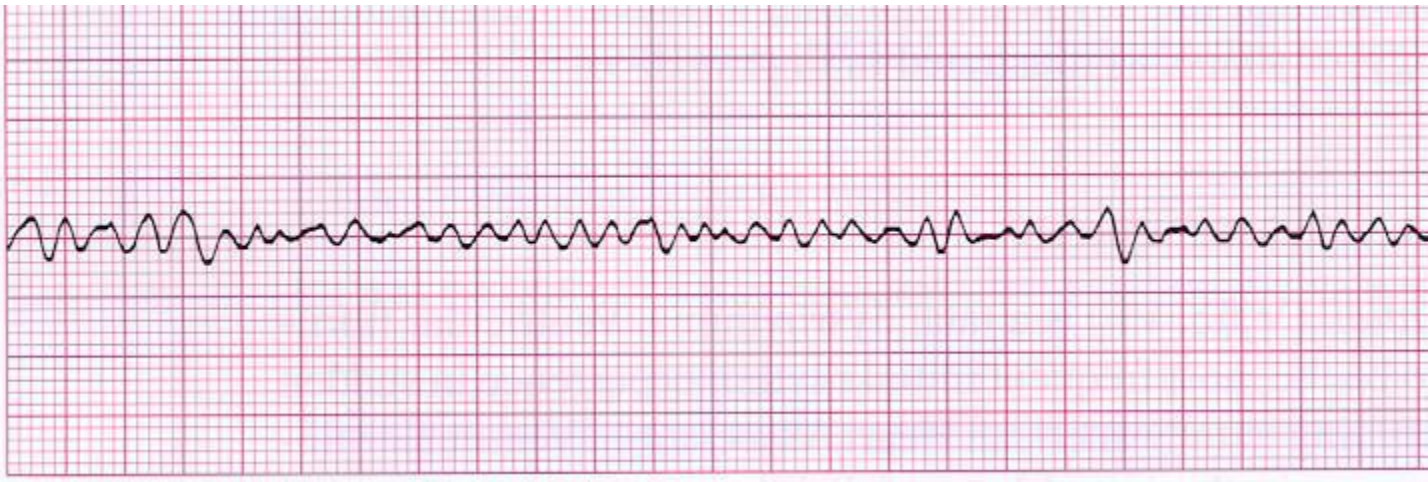


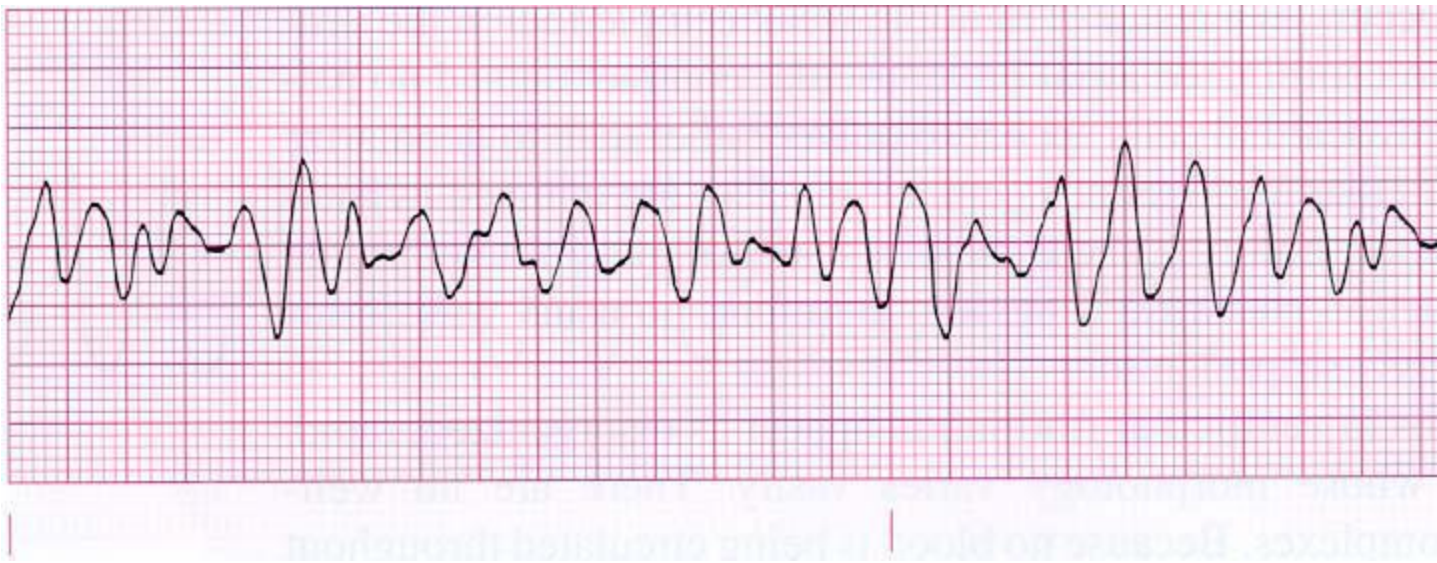
Next we will look at Ventricular Fibrillation

TABLE 10-6 VENTRICULAR FIBRILLATION

Questions 1–5

What is the rate?	Rate cannot be discerned
What is the rhythm?	Rapid, unorganized Rhythm not distinguishable
Is there a P wave before each QRS?	No
What is the length of the PR interval?	None present
Do all the QRS complexes look alike? The length of the QRS complexes is . . . ?	None present



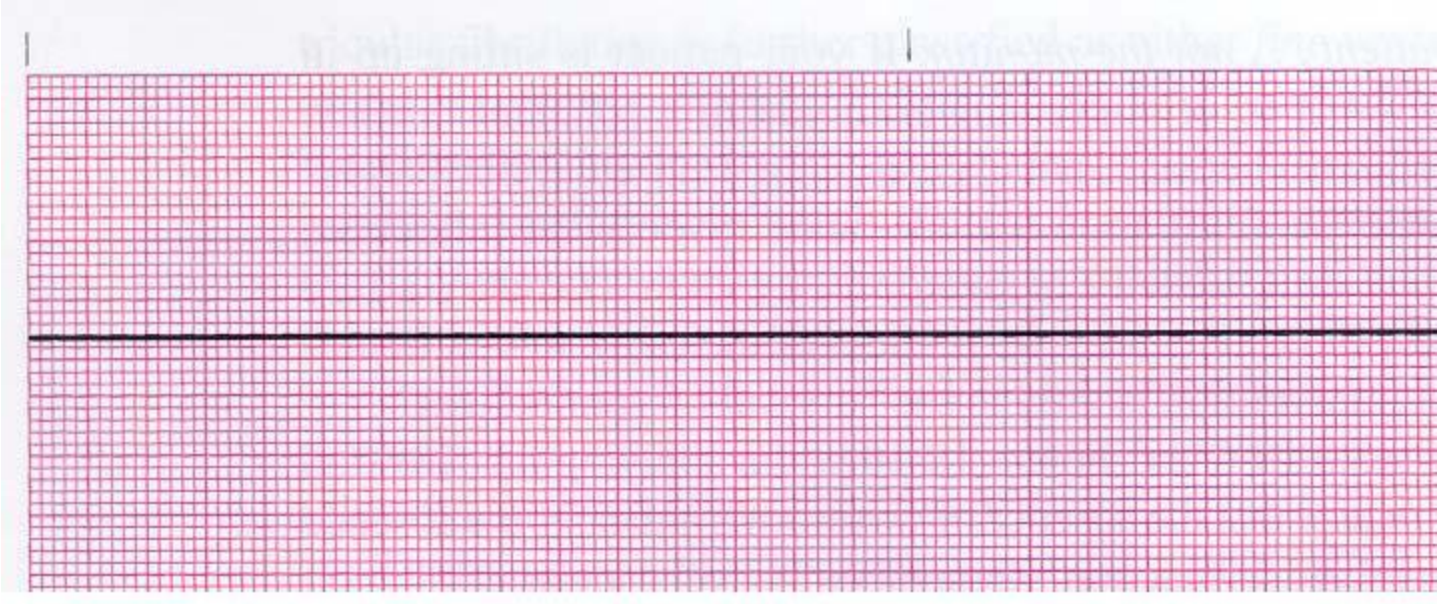


NEXT WE WILL LOOK AT ASYSTOLE OR FLAT LINE

TABLE 10-7 VENTRICULAR ASYSTOLE

Questions 1–5

What is the rate?	Absent
What is the rhythm?	Absent Rhythm not distinguishable
Is there a P wave before each QRS?	No
What is the length of the PR interval?	None present
Do all the QRS complexes look alike? The length of the QRS complexes is . . . ?	None present



PULSELESS ELECTRICAL ACTIVITY (PEA)

This type of activity is described as the absence of a palpable pulse and myocardial muscle activity with the presence of organized electrical activity (excluding VT and VF) on the cardiac monitor.

REMEMBER, THAT PEA IS NOT AN ACTUAL RHYTHM, IT REPRESENTS A CLINICAL CONDITION WHEREIN THE PATIENT IS CLINICALLY DEAD, DESPITE THE FACT THAT SOME TYPE OF ORGANIZED RHYTHMN APPEARS ON THE MONITOR.

Types of Heart blocks:

1. First Degree AV Block

2. Second Degree AV Block (Mobitz Type I) or Wenckebach

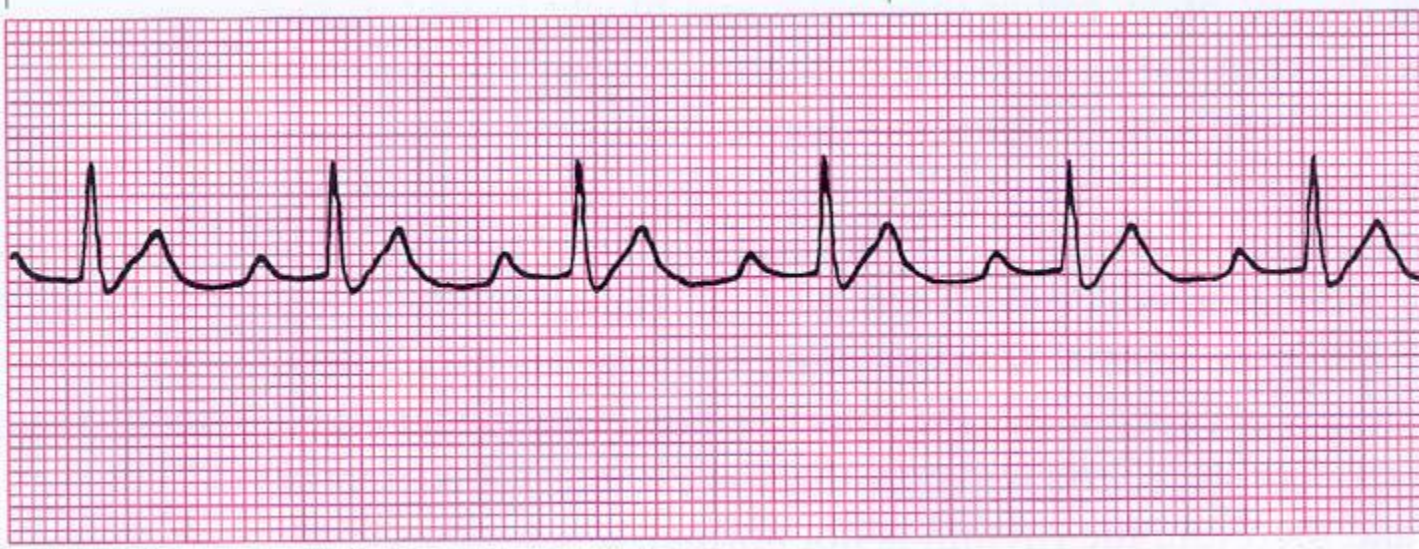
3. Second Degree AV Block (Mobitz Type II)

4. Third Degree AV Block (Complete)

TABLE 11-1 FIRST-DEGREE AV BLOCK

Questions 1–5

What is the rate?	Based on the rate of the under rhythm
What is the rhythm?	Usually regular
Is there a P wave before each QRS?	Yes
Are the P waves upright and uniform?	Yes
What is the length of the PR interval?	Greater than 0.20 sec (5 small squares)
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is . . . ?	Less than 0.12 sec (3 small sq

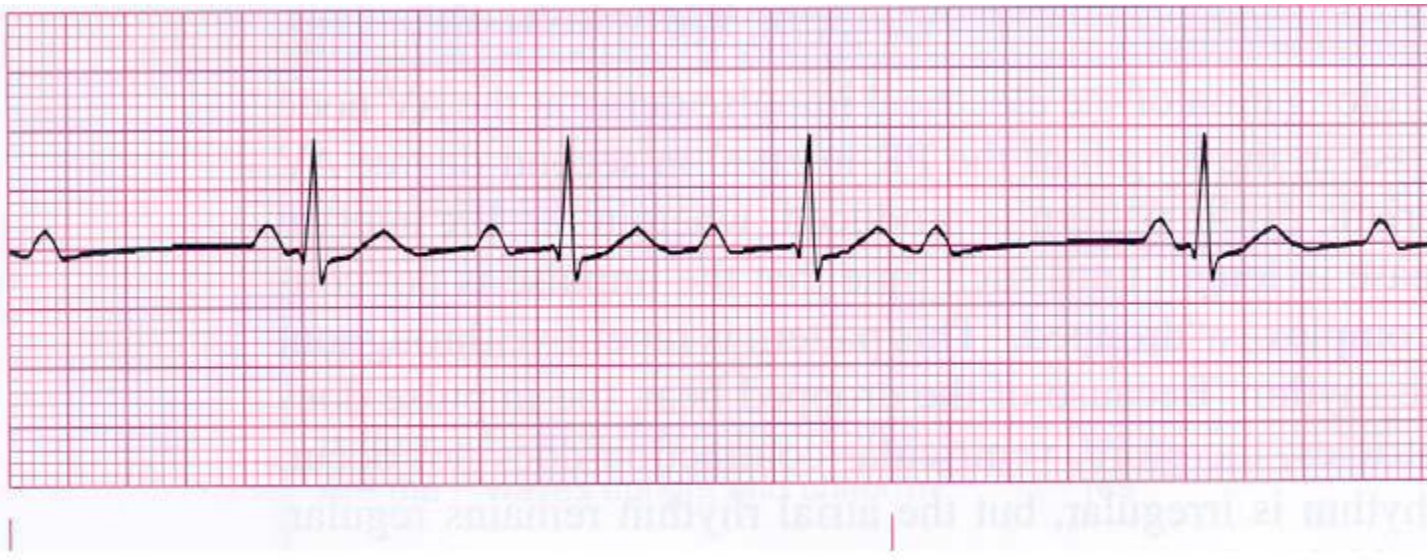


SECOND DEGREE AV BLOCK OR (MOBITZ TYPE I)/ALSO CALLED WENCKEBACH)

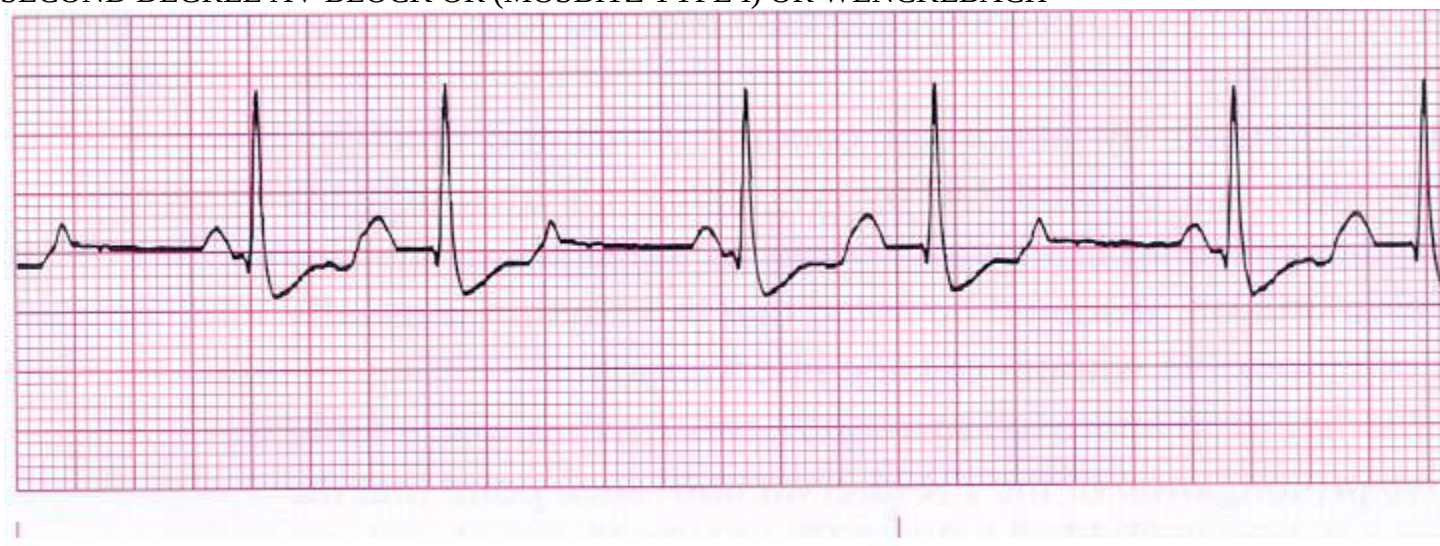
TABLE 11-2 SECOND-DEGREE BLOCK, MOBITZ TYPE I

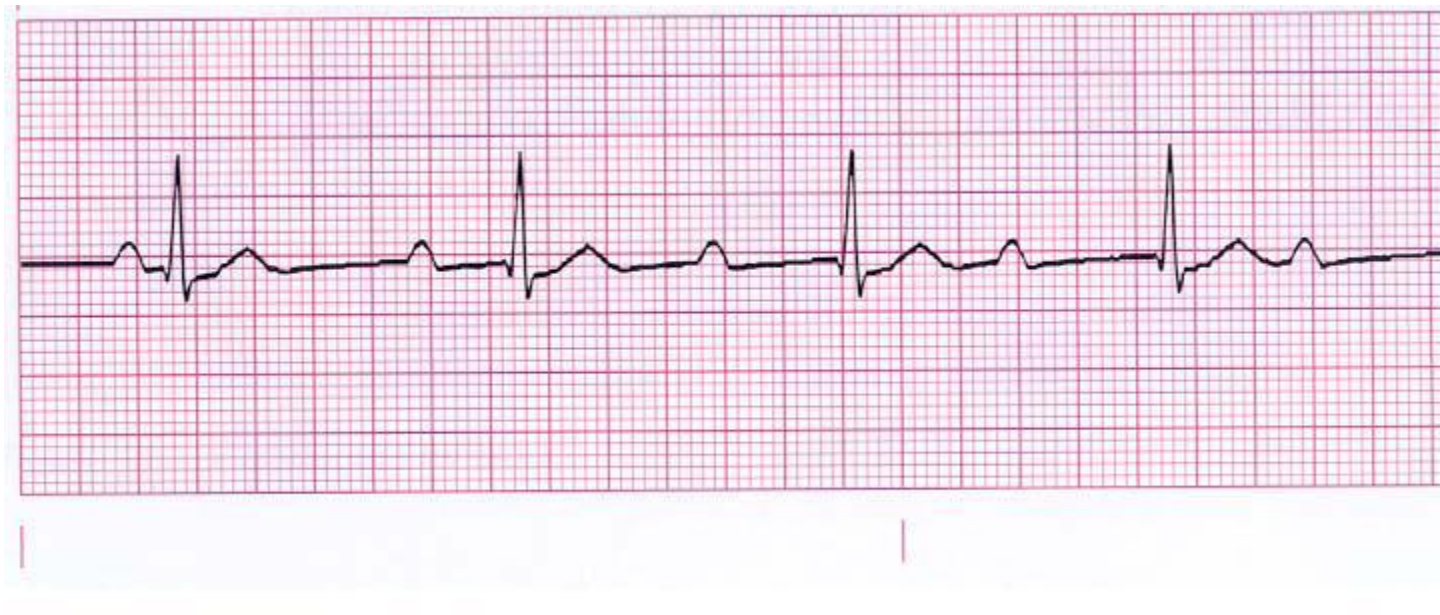
Questions 1–5

What is the rate?	Atrial unaffected Ventricular rate is usually slower than atrial
What is the rhythm?	Atrial rhythm regular Ventricular rhythm irregular
Is there a P wave before each QRS?	Yes
Are the P waves upright and uniform?	Yes, for conducted beats
What is the length of the PR interval?	Progressively prolongs until a QRS is not conducted
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is . . . ?	Less than 0.12 sec



SECOND DEGREE AV BLOCK OR (MOSBITZ TYPE I) OR WENCKEBACH



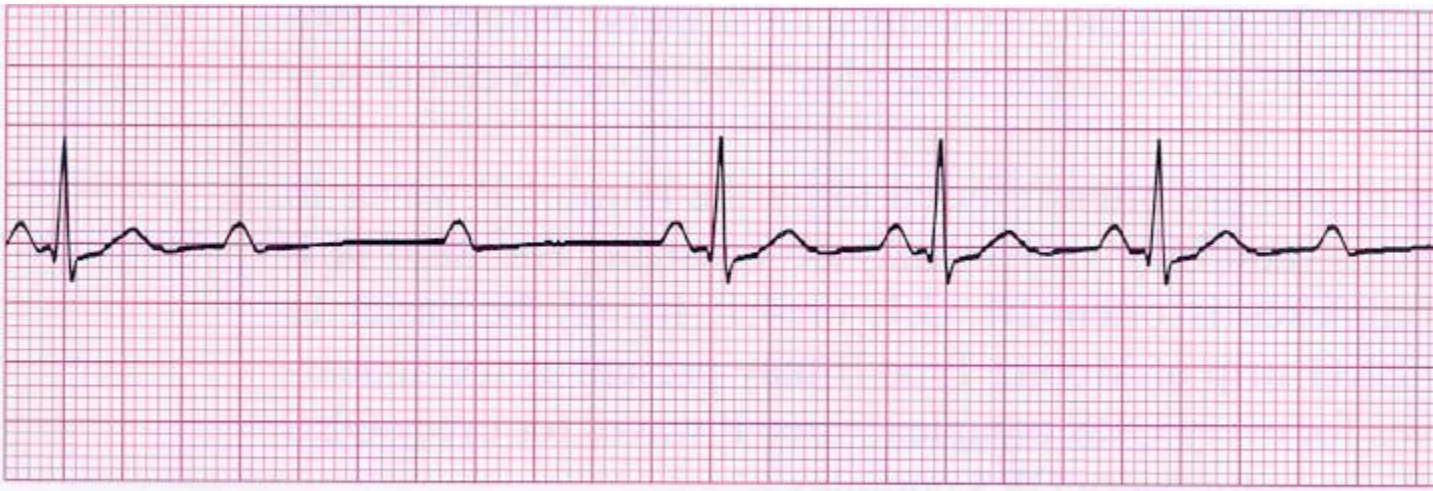


SECOND DEGREE AV BLOCK OR MOBITZ TYPE II

TABLE 11-3 SECOND-DEGREE BLOCK, TYPE MOBITZ II

Questions 1–5

What is the rate?	Atrial rate regular Ventricular rate may be bradyca
What is the rhythm?	Atrial rhythm regular Ventricular rhythm irregular
Is there a P wave before each QRS?	Yes; some P waves are not foll by a QRS complex
Are the P waves upright and uniform?	P waves are usually upright and uniform
What is the length of the PR interval?	Constant for conducted beats
Do all the QRS complexes look alike?	Yes; intermittently absent
The length of the QRS complexes is . . . ?	Greater than or equal to 0.12 se

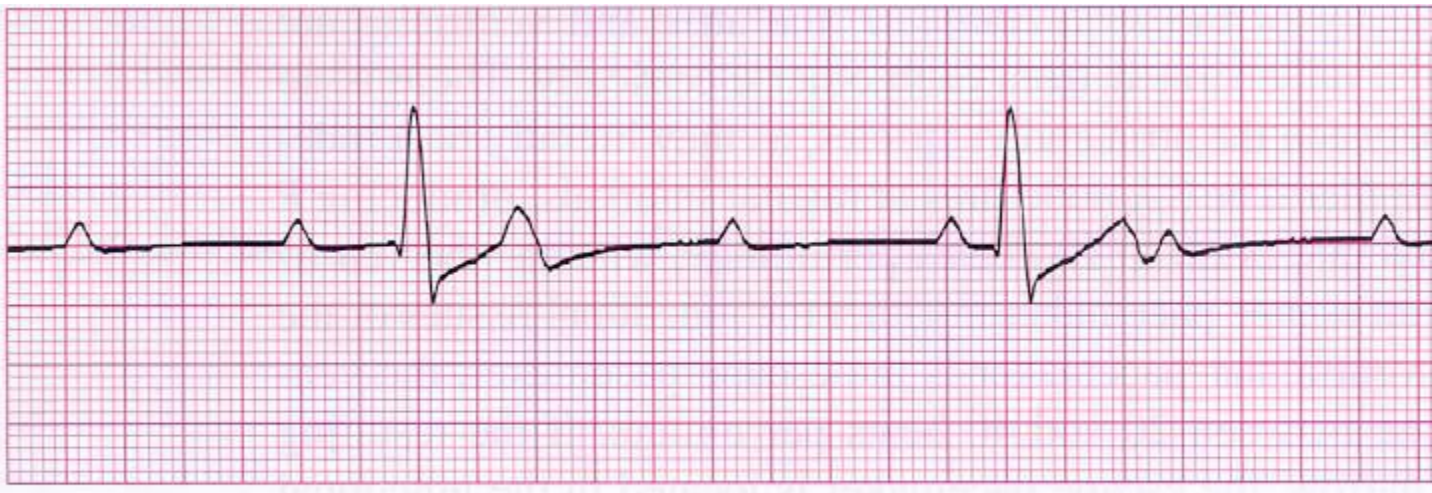


NOW LET US LOOK AT THIRD DEGREE AV BLOCK (COMPLETE)

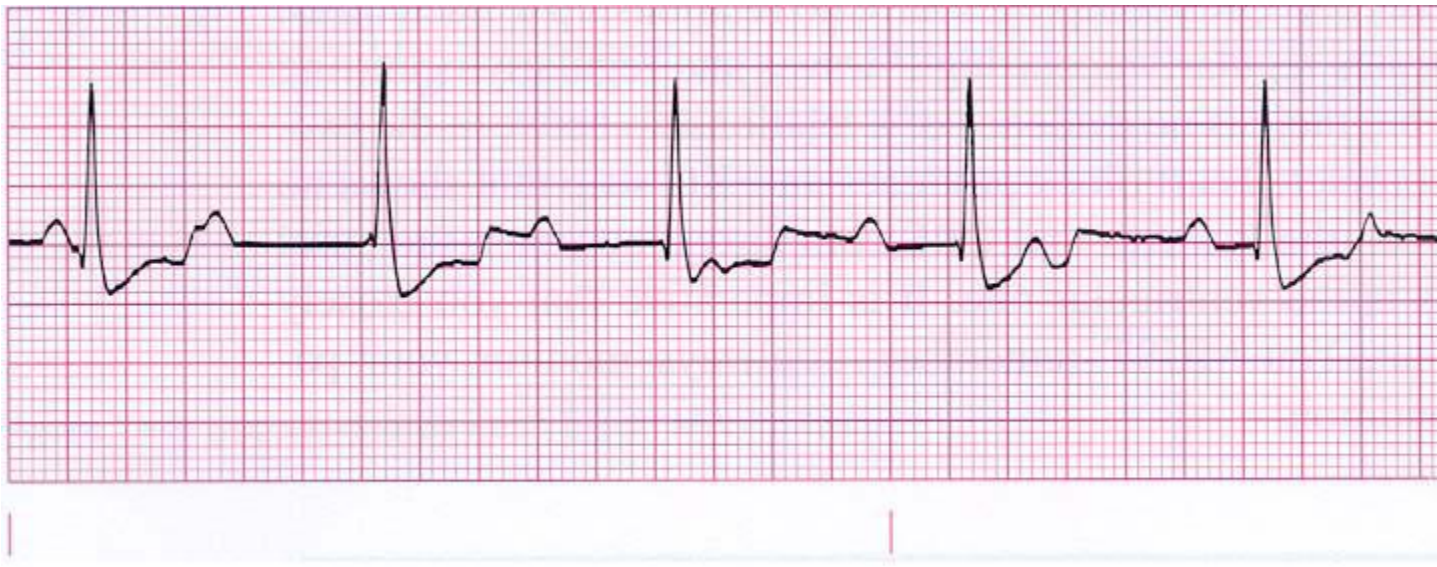
TABLE 11-4 THIRD-DEGREE (COMPLETE) HEART BLOCK

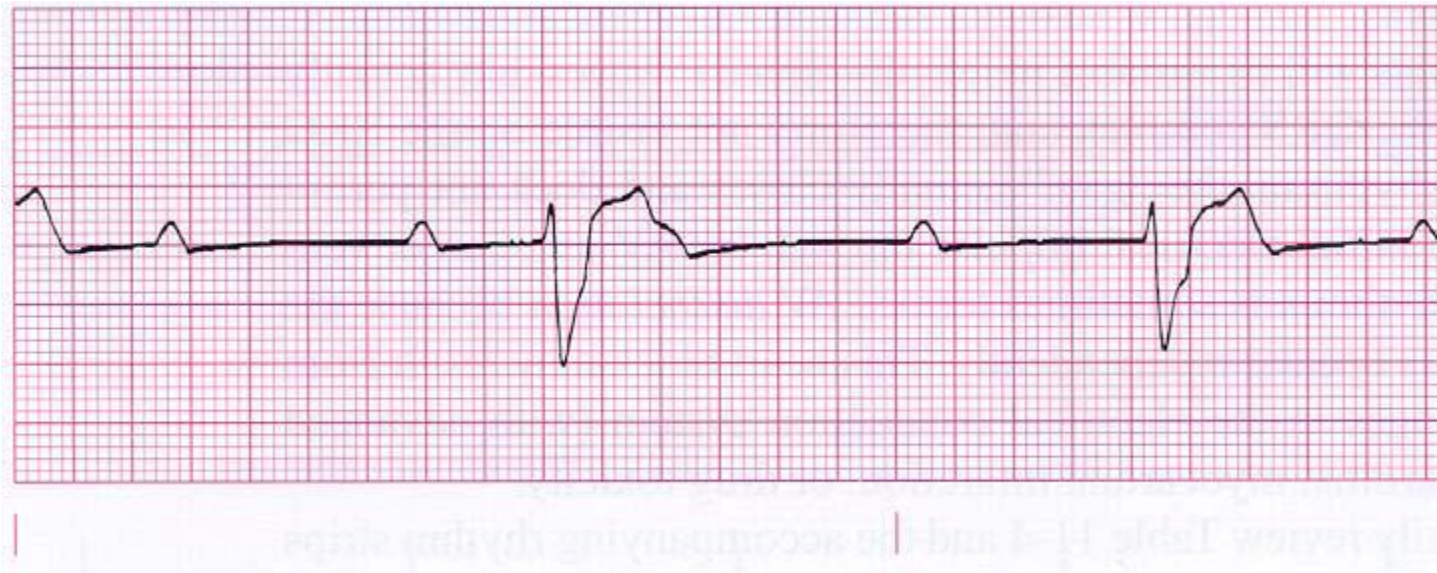
Questions 1–5

What is the rate?	Atrial rate usually 60 to 100 BPM Ventricular rate based on site of escape pacemaker
What is the rhythm?	Atrial rhythm regular Ventricular rhythm regular
Is there a P wave before each QRS?	No relationship to QRS complexes
Are the P waves upright and uniform?	Yes
What is the length of the PR interval?	Totally variable; no pattern
Do all the QRS complexes look alike?	Yes
The length of the QRS complexes is . . . ?	Based on site of escape pacemaker



NEXT WE WILL LOOK AT THE THIRD DEGREE AV BLOCK COMPLETE



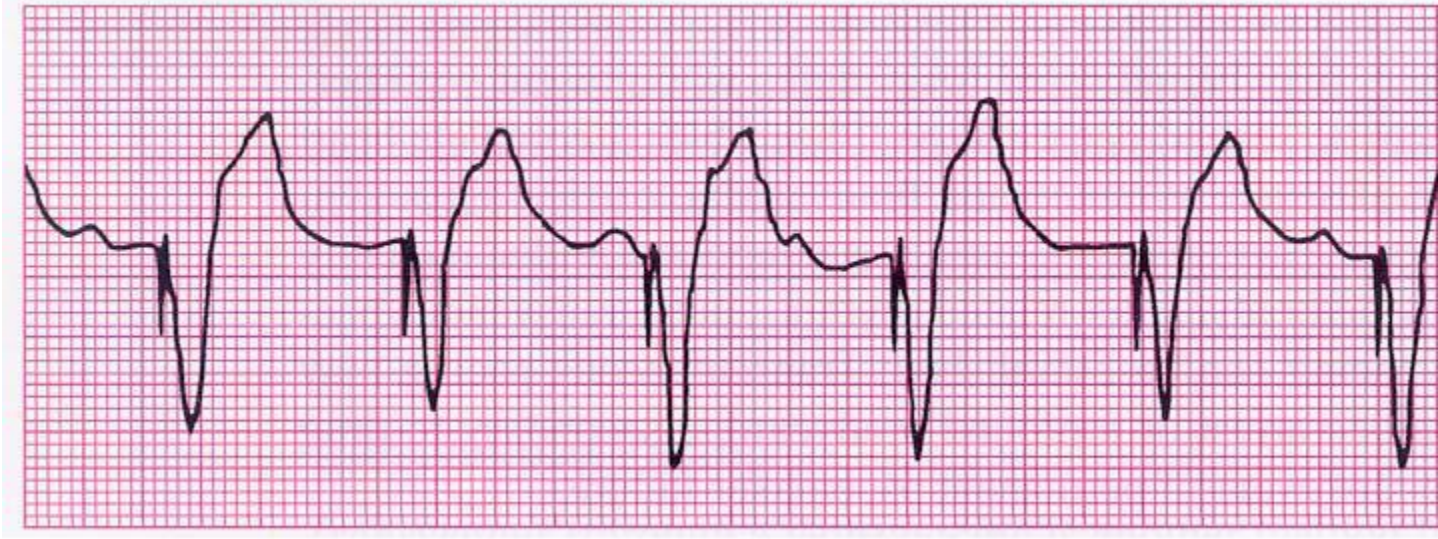


THE ARTIFICIAL PACEMAKER RHYTHM IS VERY IMPORTANT FOR PATIENT'S WHO HAVE A PACEMAKER

TABLE 12-1 ARTIFICIAL PACEMAKER RHYTHM

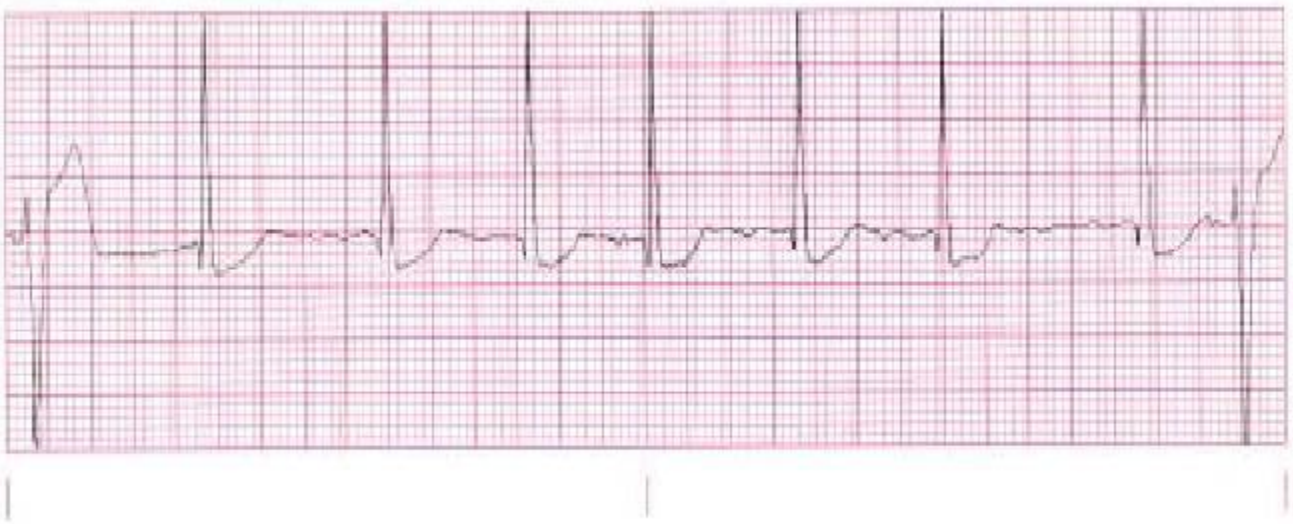
Questions 1–5

What is the rate?	Varies according to preset rate of pacemaker (usually 70 BPM)
What is the rhythm?	Regular if pacing is fixed, irregular if demand-paced
Is there a P wave before each QRS? Are the P waves upright and uniform?	May be absent or present, based type of artificial pacemaker
What is the length of the PR interval?	Variable, depending on type of artificial pacemaker
Do all the QRS complexes look alike? The length of the QRS complexes is . . . ?	Usually; greater than or equal to 0.12 sec; bizarre morphology; presence of spikes



READY TO DO SOME PRACTICE STRIPS? GOOD, LETS SEE HOW YOU DO

1.



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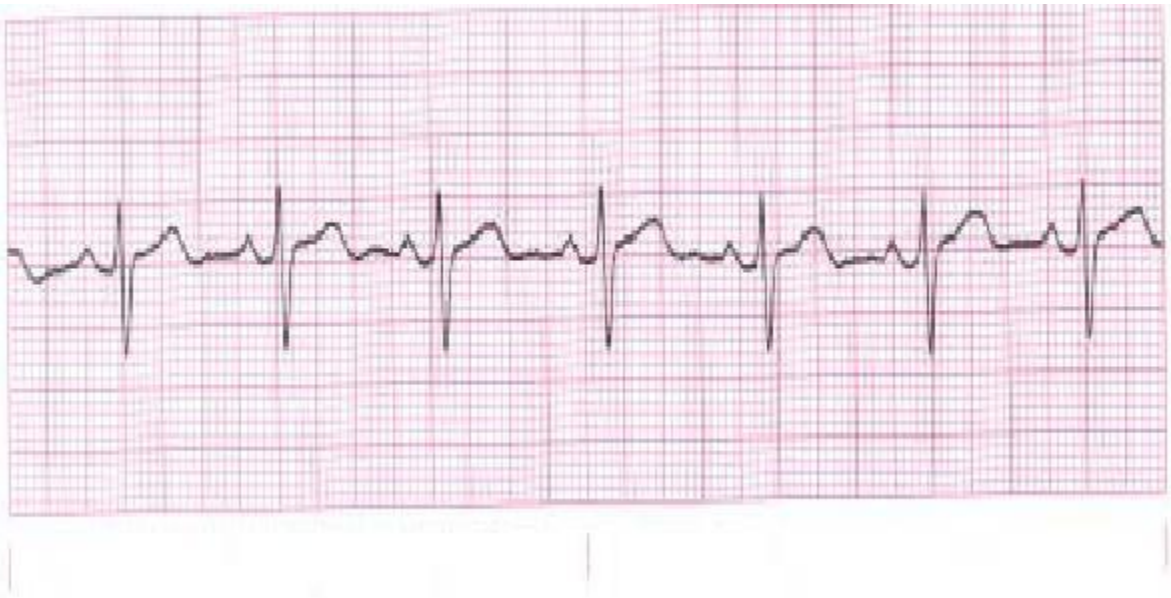

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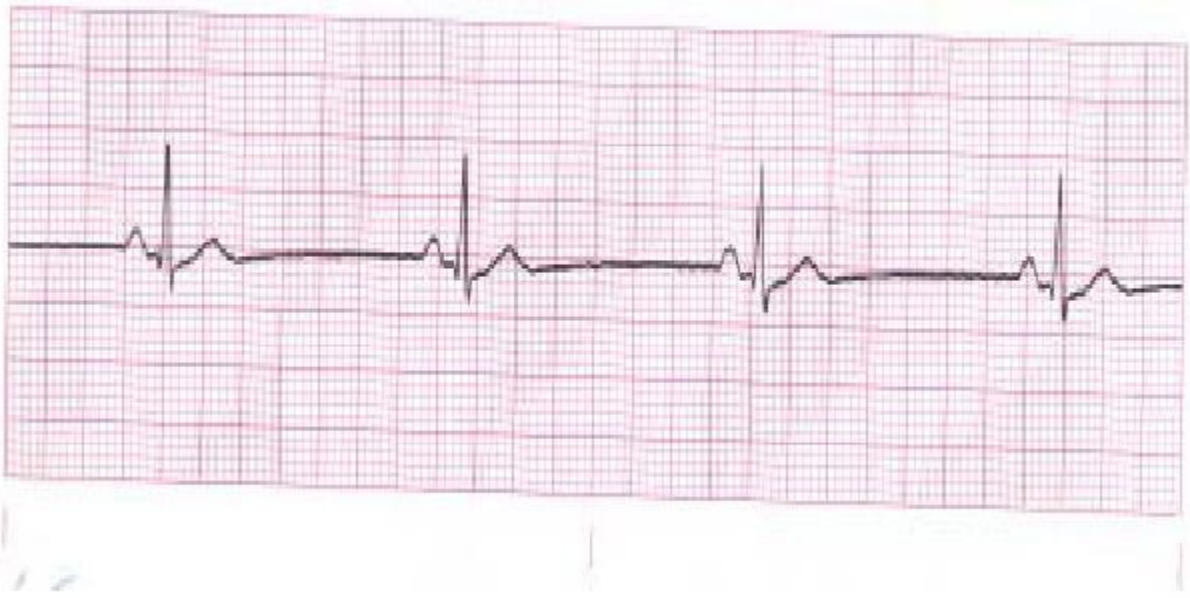

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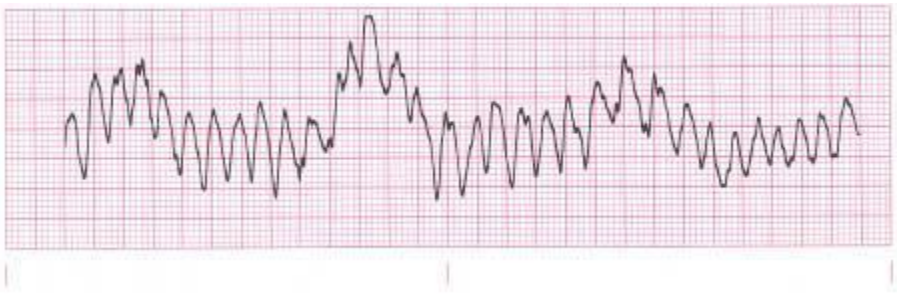
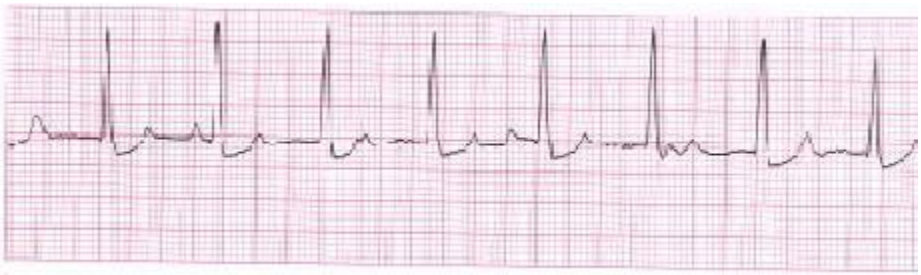
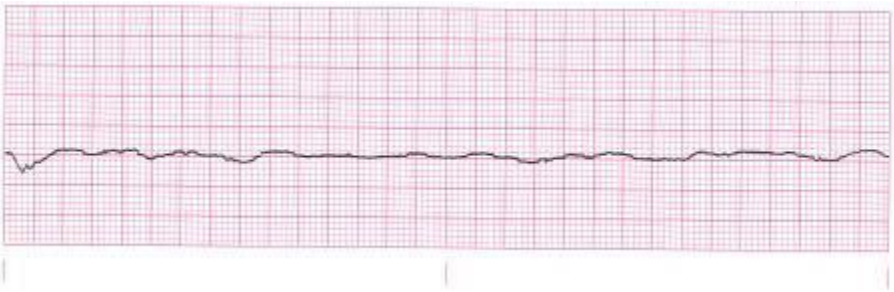
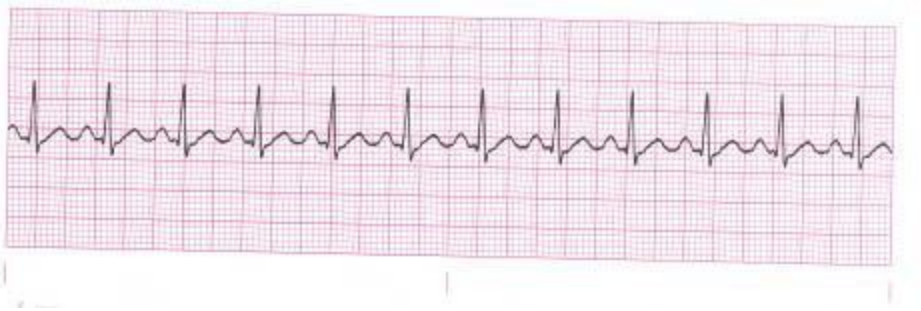


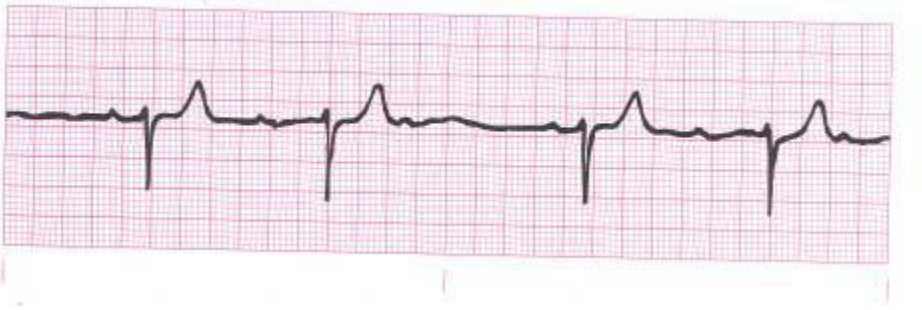
5.

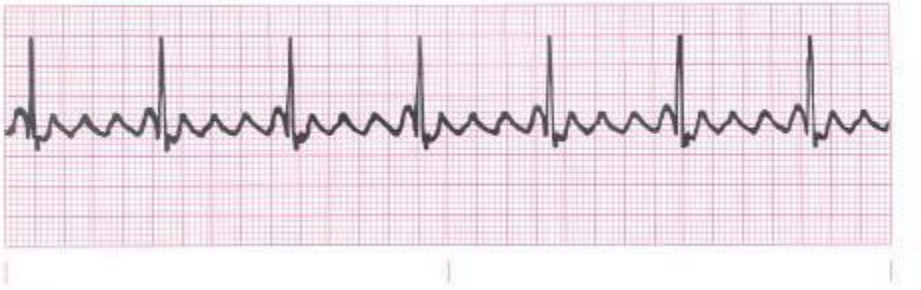


6.



7. 

8. 

9. 

Answers: 1. A Fib wtih PVC's

2. NSR 3. Sinus Bradycardia 4. Ventricular Fibrillation

5. Third degree heart block 6.Asytole

7. Sinus Tachycardia 8. Second Degree AV Block Mobitz Type I Wenckebach

9. Atrial Flutter

CET Study Guide (This is your study guide for National Certification)

Signs and Symptoms

Other Symptoms of Heart Rhythm Disorders

Heart rhythm disorders cause many different symptoms, or no symptoms at all. Sometimes, a person may feel an irregular heartbeat. Lightheadedness, fainting, chest pain and/or shortness of breath are signs that an arrhythmia may be serious.

You may have an arrhythmia without symptoms. The abnormal rhythm may be detected when you feel your pulse, or when your doctor listens to your heart or takes your blood pressure. It also may be diagnosed with a test called an electrocardiogram.

Palpitations are a common complaint that many people describe as a skipping, pounding, fluttering, flip-flopping, racing, or sudden stopping of the heartbeat. You may feel your heart speed up when you climb a flight of stairs or drink too much coffee. The rapid beating may last for seconds, minutes or even hours.

- A rapid heartbeat with other symptoms such as shortness of breath, chest pain or pressure, lightheadedness or fainting (syncope) may be due to a potentially life-threatening arrhythmia called ventricular tachycardia.

People who have any unexplained episode of fainting should see their doctor.

- Premature heartbeats occur when the heart's regular rhythm is interrupted by early or premature beats. It may feel as if the heart has skipped a beat. Usually it is not serious. If the beat arises from locations in the atria (upper chambers) it is called premature atrial beat. Premature ventricular beats arise from the ventricles (lower chambers). Sometimes they are called premature ventricular contractions, or PVCs.

- Lightheadedness or loss of consciousness. Lightheadedness is a common symptom that has many causes, including temporary conditions that are not serious. It is often described as feeling "far-away" or off balance. Individuals may feel as if they will faint or "black out." If the heart rate is too fast (tachycardia) or too slow (bradycardia) the blood supply to the brain may be reduced. If either arrhythmia lasts longer than six seconds, it can cause loss of consciousness, or fainting. The medical term for fainting is syncope.

Chest pain may be caused by rapid heart rhythms (tachycardias) that increase the oxygen needs of your heart. When the demand is too great, the heart cries out in pain. Pressure or aching in the chest and shortness of breath may accompany the fast heart rhythms. These are serious warning signs that should send you to a doctor. Chest pain also may be caused by coronary artery disease(CAD), a condition in which the blood supply to the heart is reduced because of clogged blood vessels. The medical term for this type of pain is called angina pectoris.

Angina may be the warning sign of a heart attack(myocardial infarction).

Chest pain due to cardiac causes may be associated with nausea, vomiting, sweating, shortness of breath, abnormal heart rhythms or weakness.

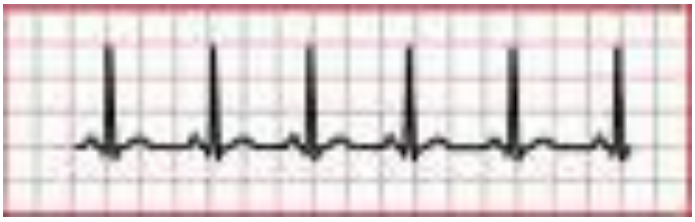
Shortness of Breath, or **dyspnea**, may be a sign of heart attack or of a slow or rapid heart rate that impairs the heart's ability to fill, causing blood to back up into the lungs. The most common arrhythmia associated with shortness of breath is atrial fib. It also may be a symptom of more serious ventricular arrhythmia's.

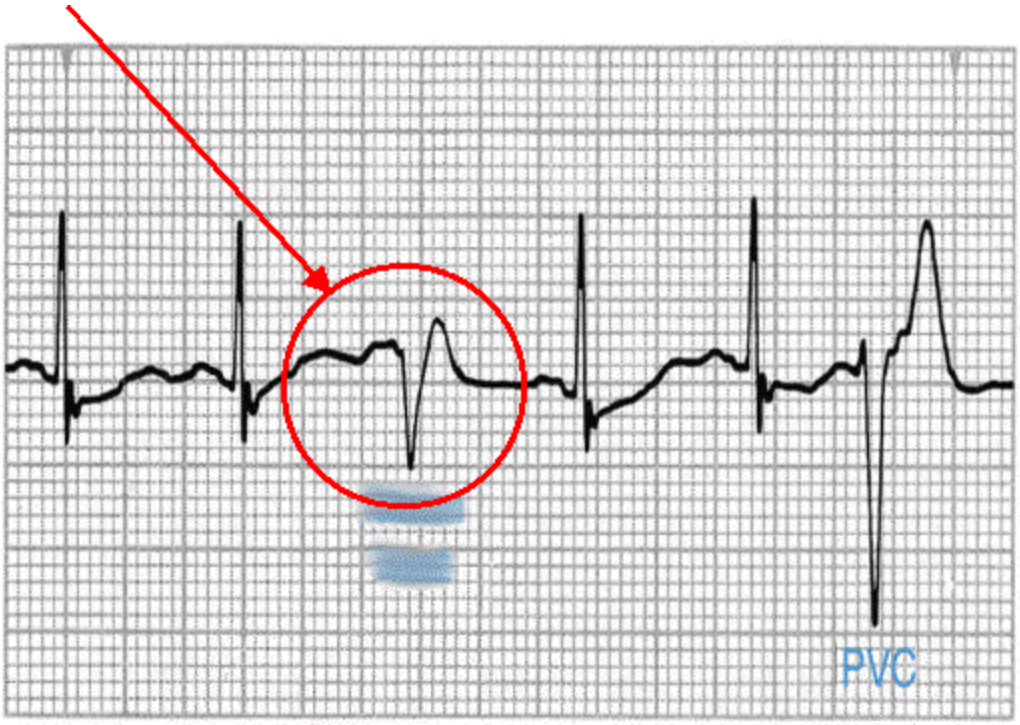
Fatigue is a common complaint with many causes, most of which are unrelated to the heart. It is normal to feel tired following hard work or exercise, sustained stress, anxiety or grief. Most illnesses can cause fatigue. Some medications and other medical treatments also may be a cause. People may feel tired when the heart rate is very slow (bradycardia) or rapid (tachycardia).

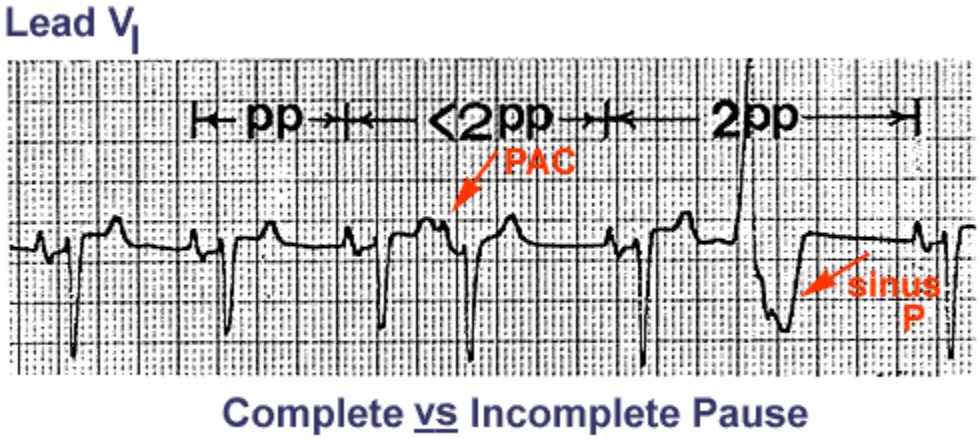
Name:_____

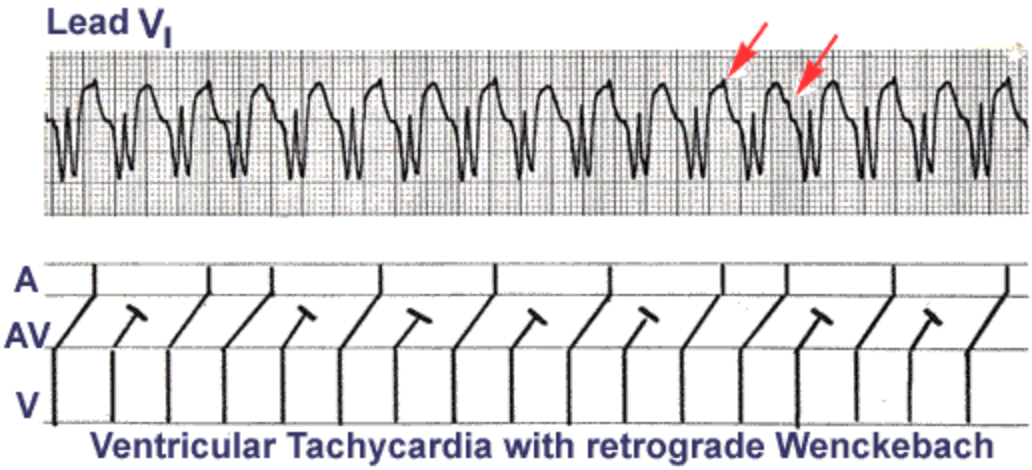
Cardiac Arrhythmia Test;

For the following strips identify the QRS, QT, Heart Rate, PR, ST, intervals, and whether or not the rhythm is normal or abnormal as well as the type of rhythm displayed. Try your best to identify the rhythm.

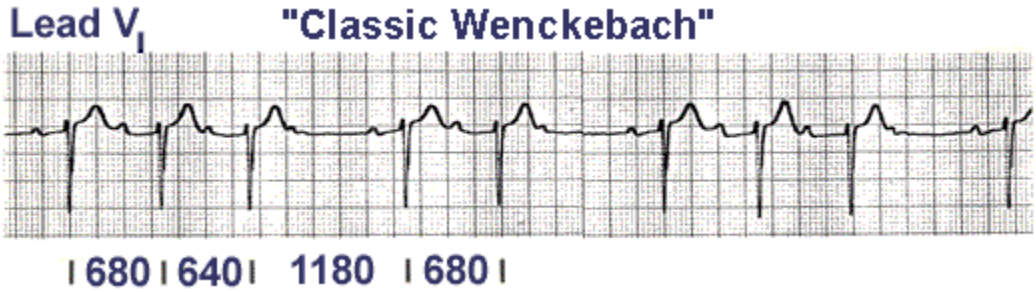
1. 

2. 
PVC

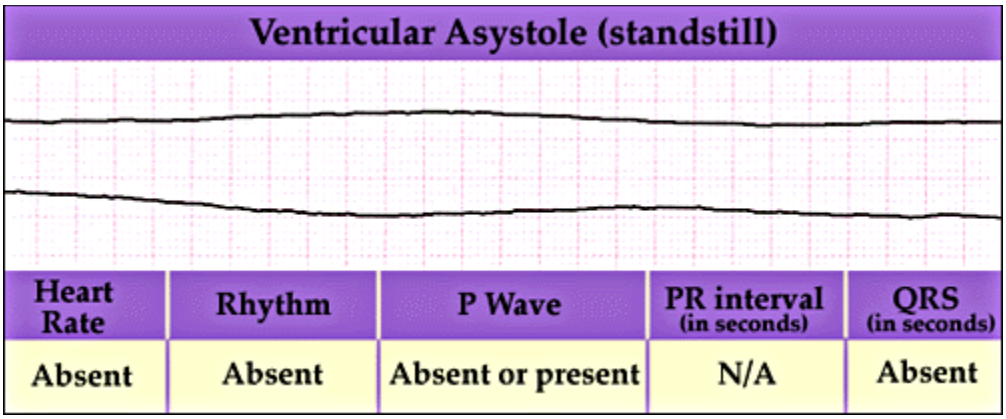
3. 
Lead V1
pp
<2pp
2pp
PAC
sinus P
Complete vs Incomplete Pause

4. 

(measure for proof)

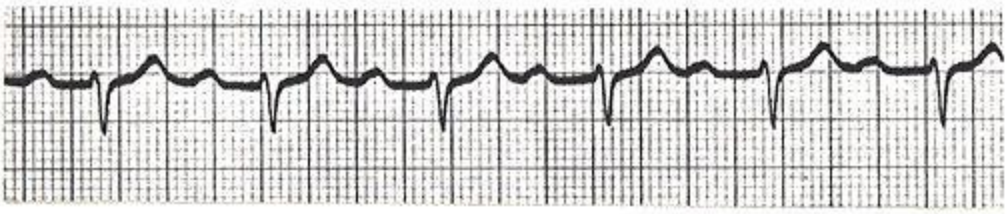
5. 

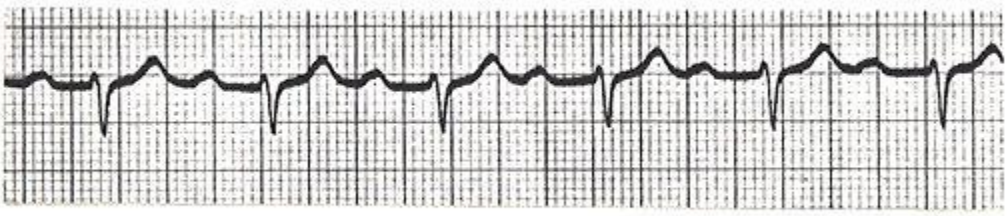
(prove with measurements)

6. 

Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
Absent	Absent	Absent or present	N/A	Absent

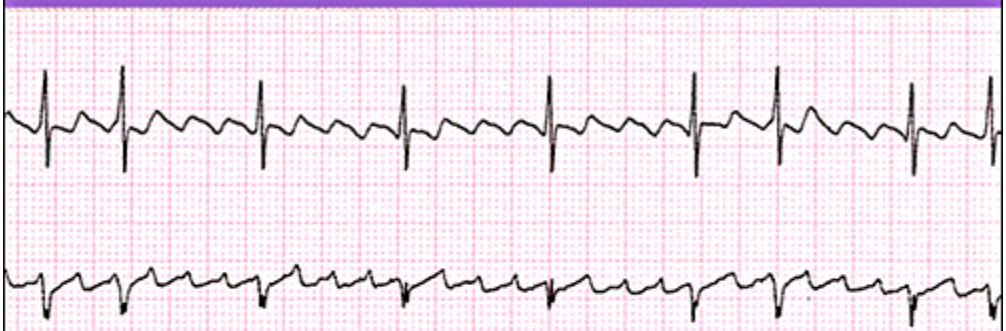
(explain what is happening on this strip and why)

7. 

8. 

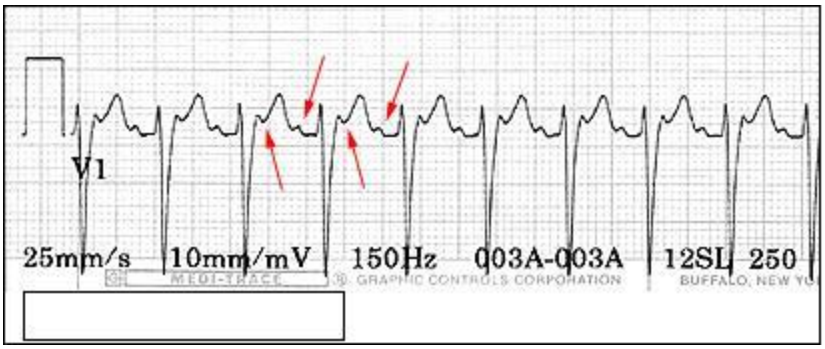
9.

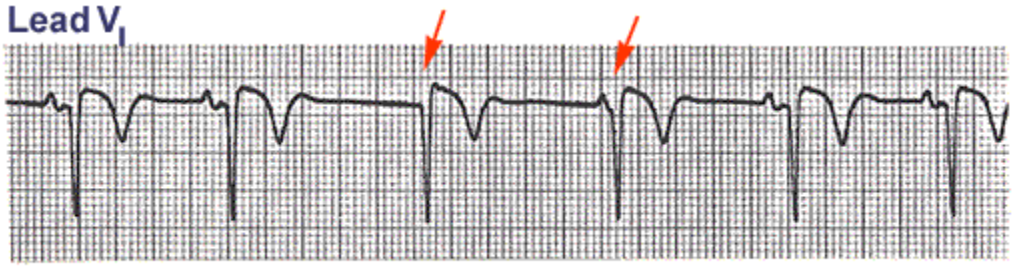
Atrial Flutter



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
A: 220-430 bpm V: <300 bpm	Regular or variable	Sawtoothed appearance	N/A	<.12

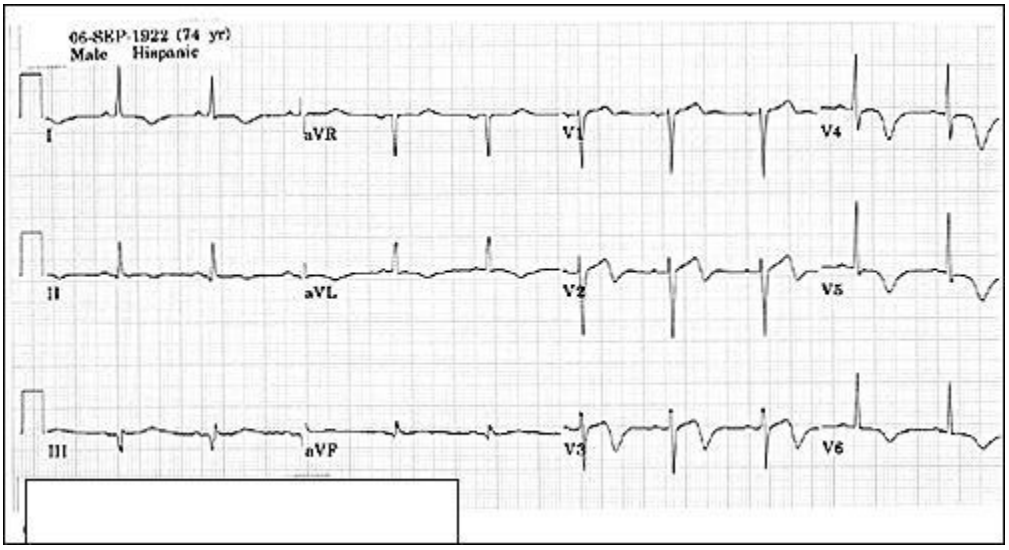
Describe how a person would feel when this cardiac rhythm is experienced. What is happening?

10. 

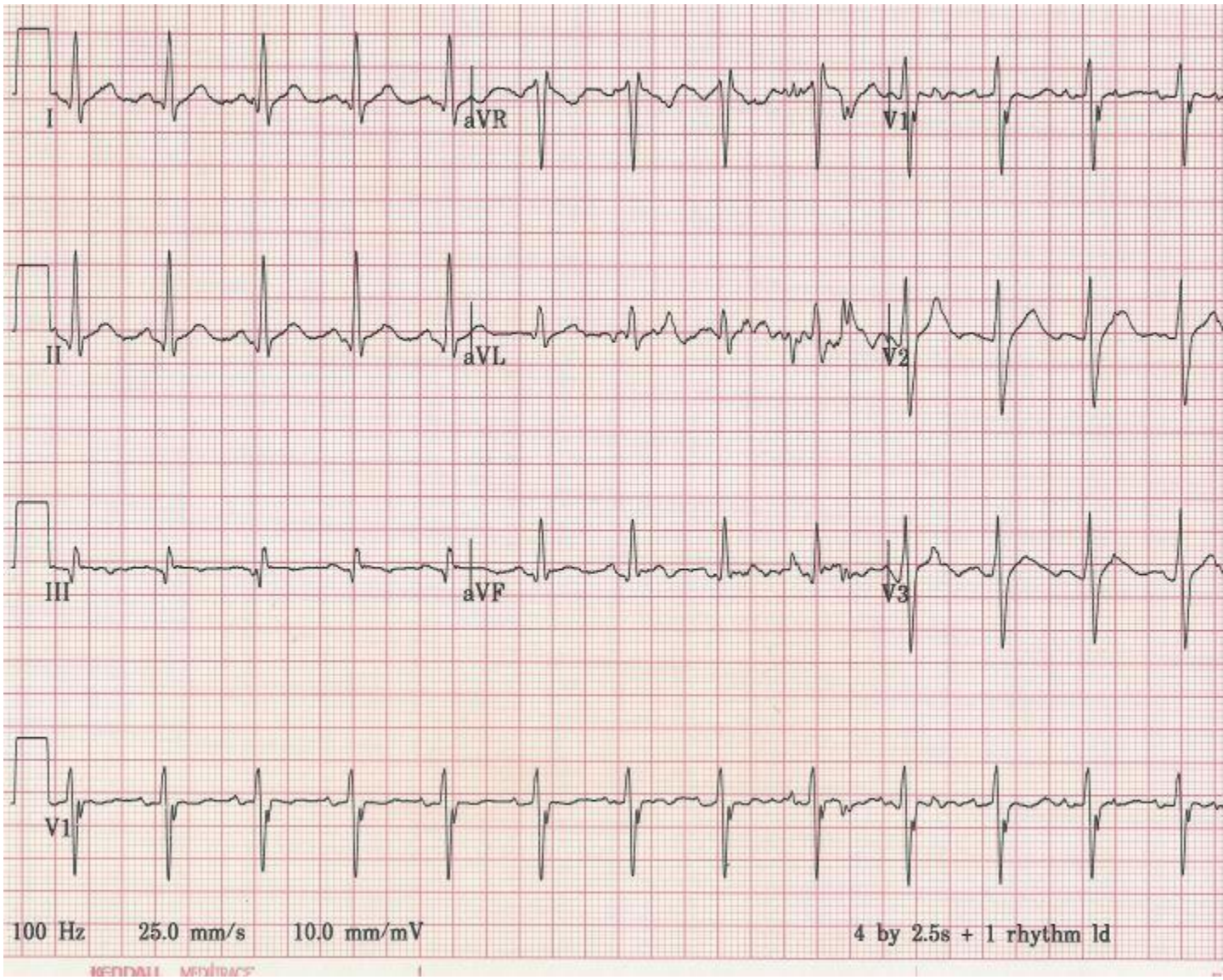
11. 

Incomplete AV dissociation due to sinus slowing (default) with junctional escapes (arrows)

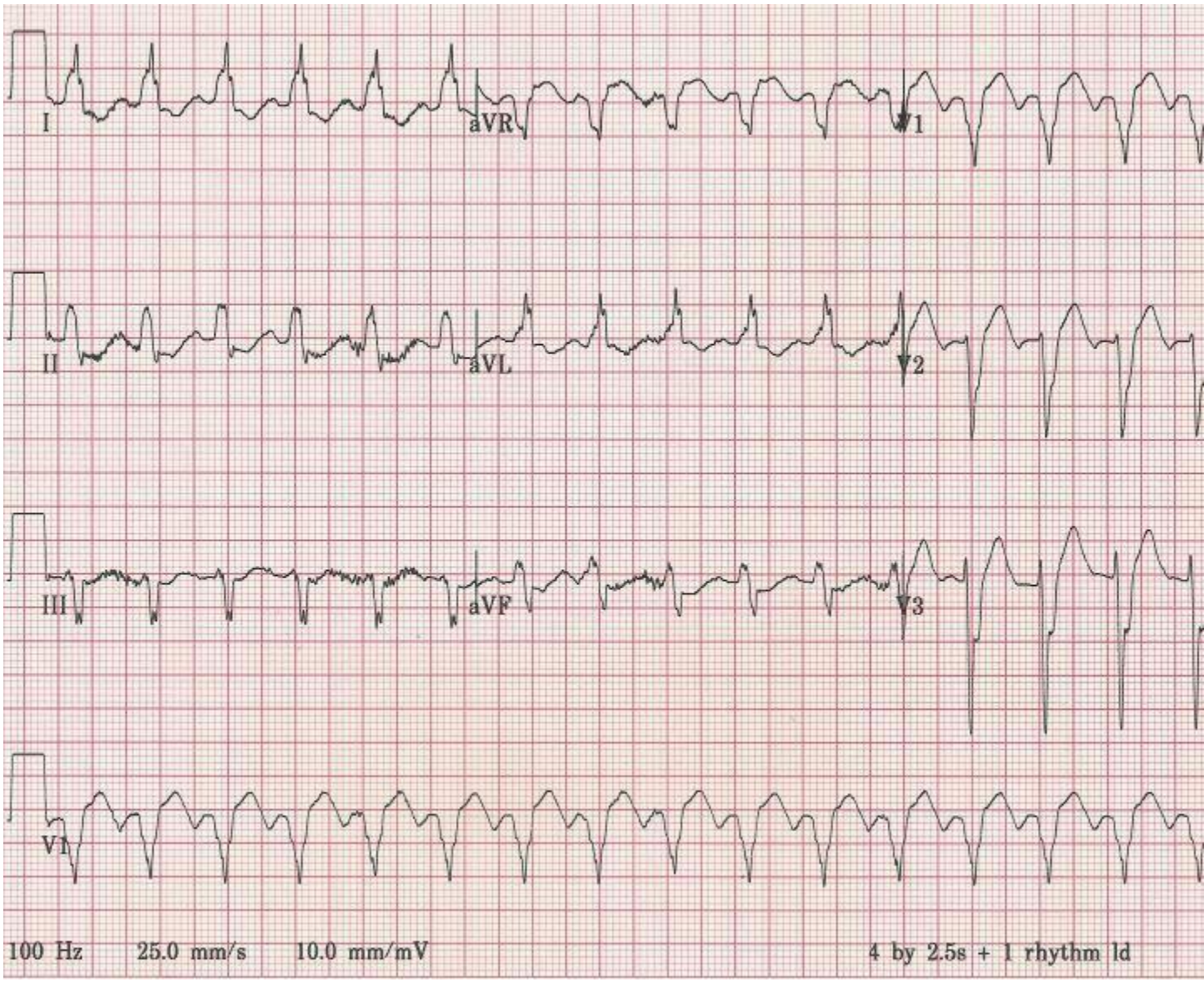
Name this rhythm

12. 

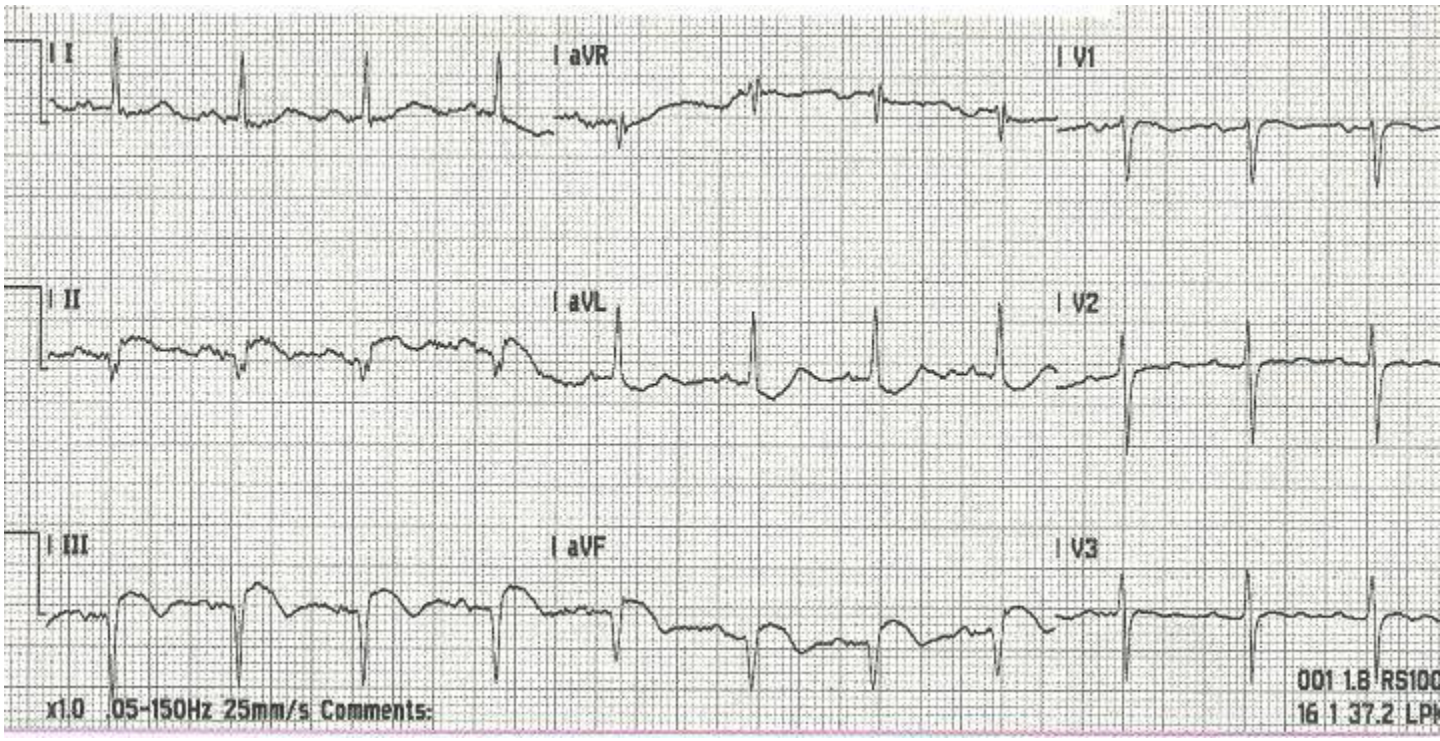
13.


I
aVR
V1
II
aVL
V2
III
aVF
V3
V1
100 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld

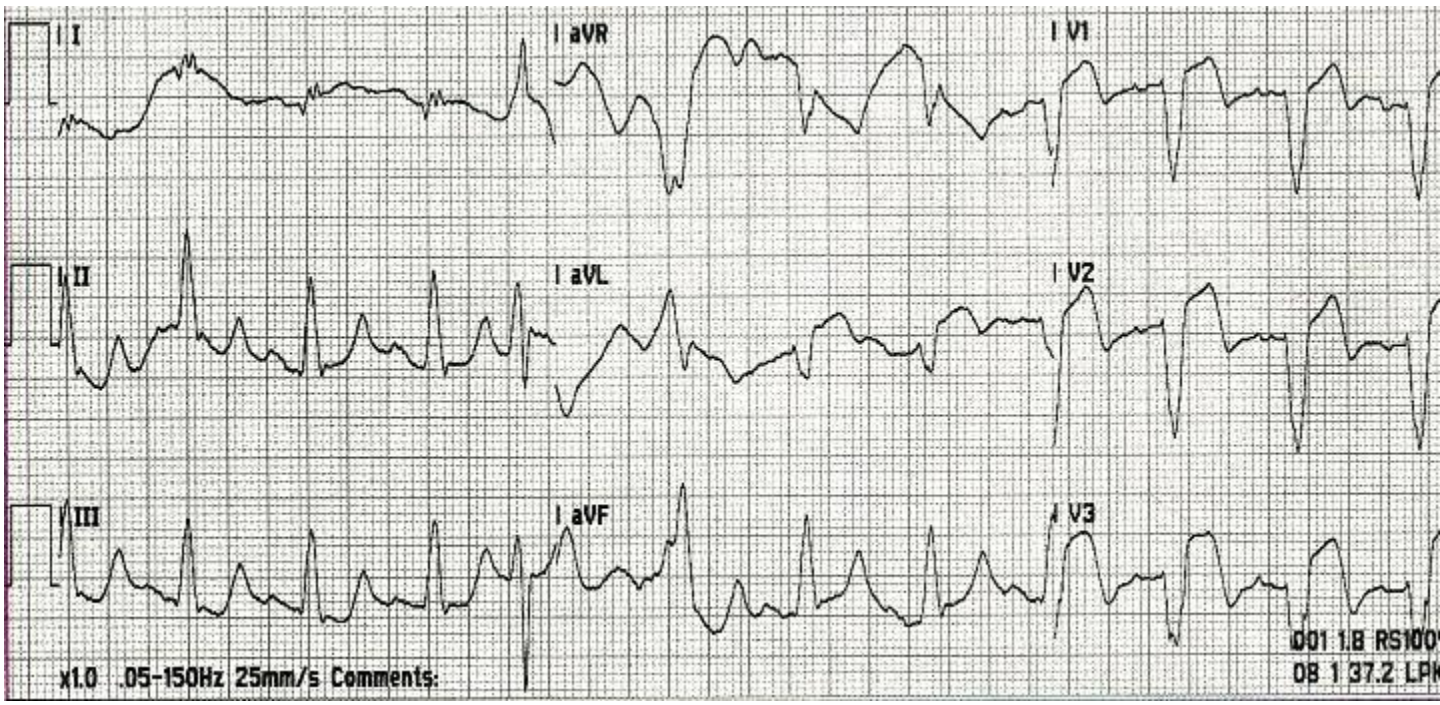
14.


I
aVR
V1
II
aVL
V2
III
aVF
V3
V1
100 Hz
25.0 mm/s
10.0 mm/mV
4 by 2.5s + 1 rhythm ld

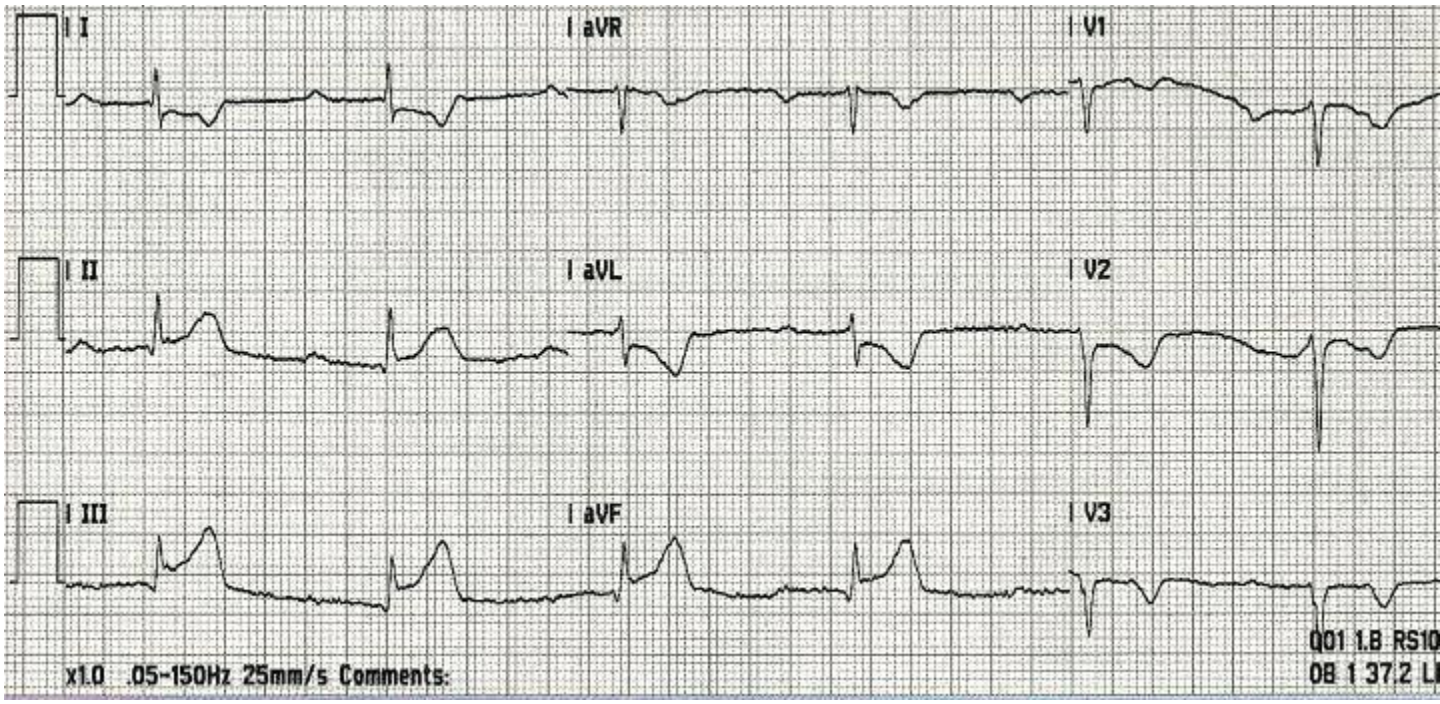
15.


I I
I aVR
I V1
I II
I aVL
I V2
I III
I aVF
I V3
x1.0 05-150Hz 25mm/s Comments:

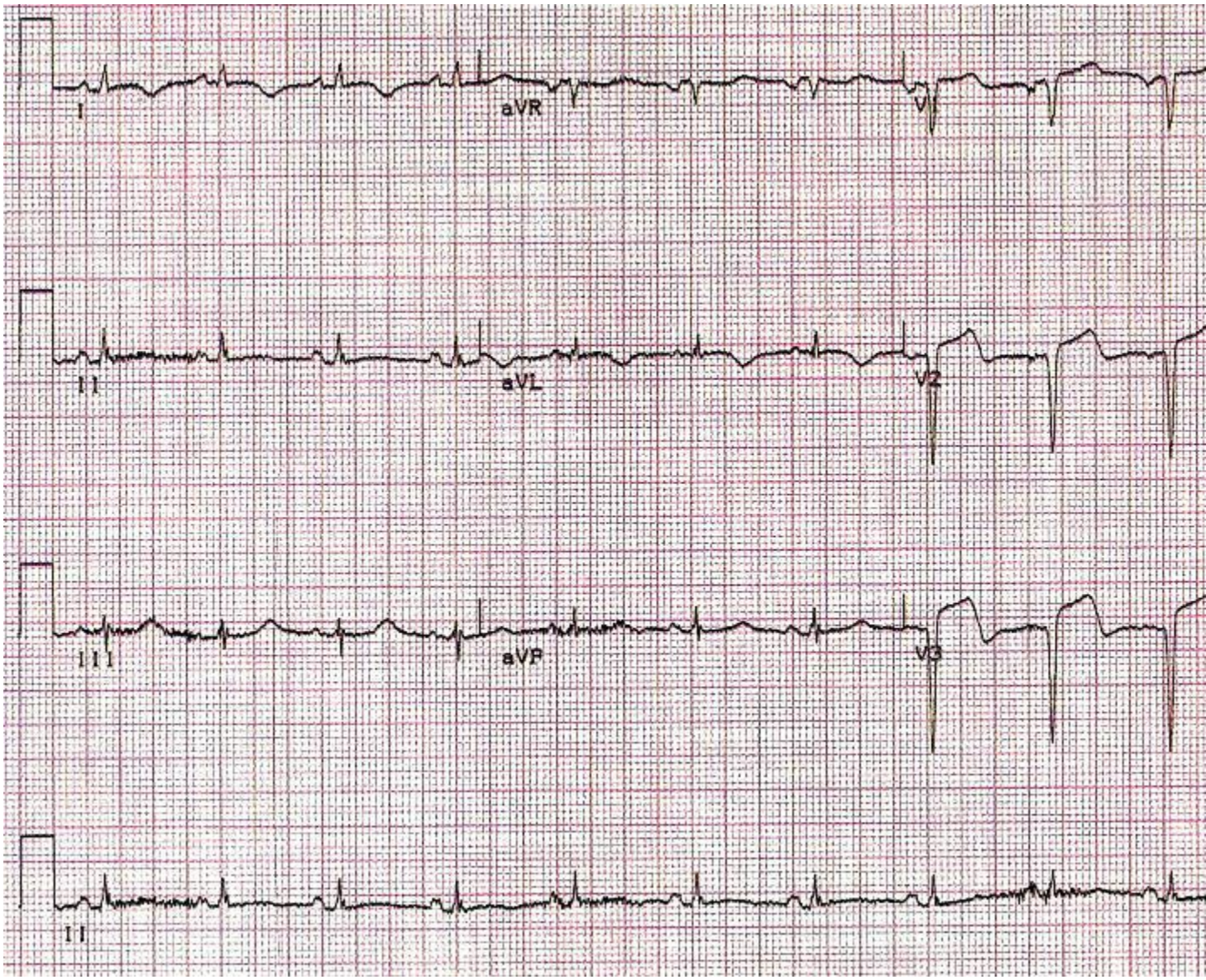
16.


I I
I aVR
I V1
I II
I aVL
I V2
III
I aVF
V3
x1.0 .05-150Hz 25mm/s Comments:

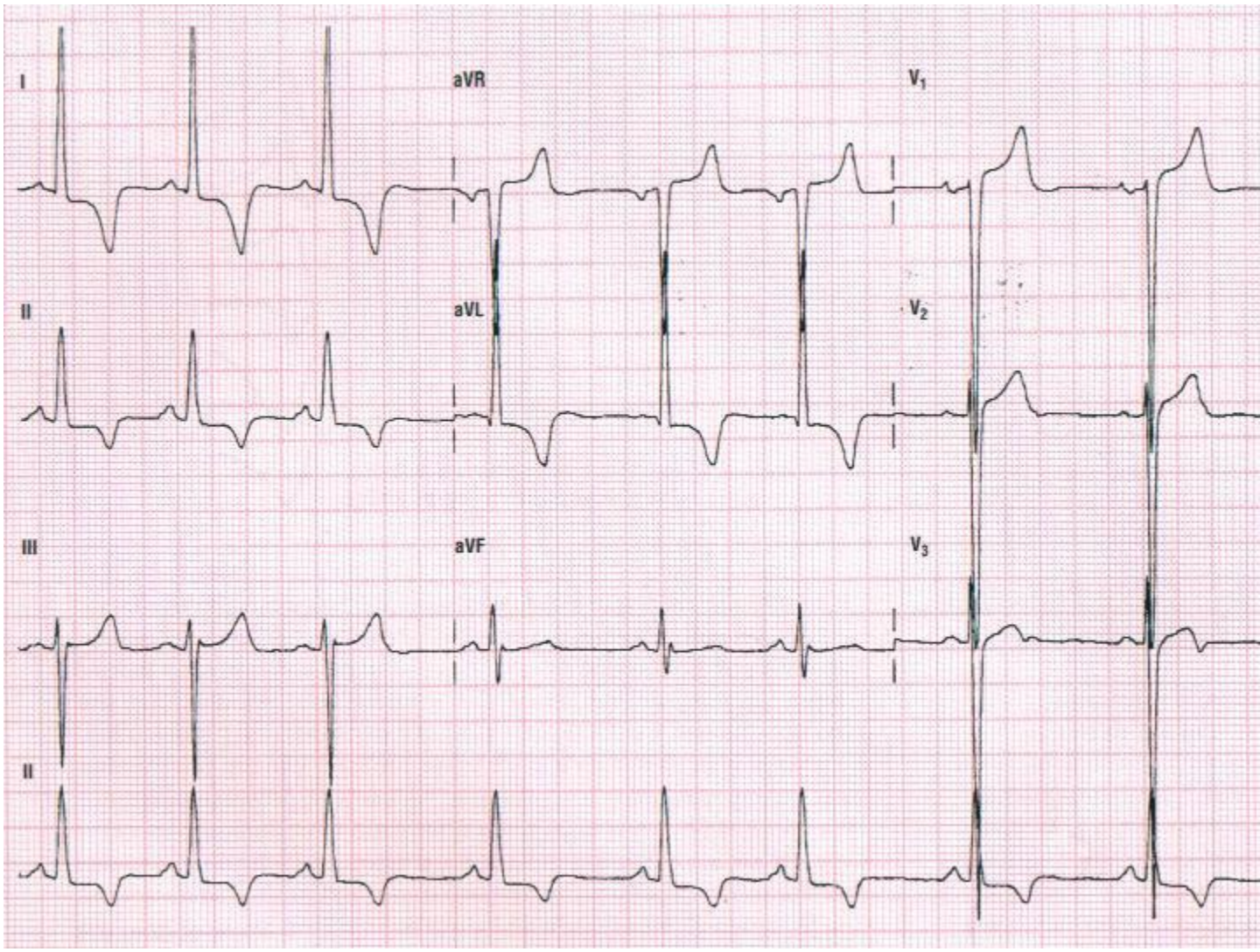
17.


I I
I aVR
I V1
I II
I aVL
I V2
I III
I aVF
I V3
x1.0 .05-150Hz 25mm/s Comments:

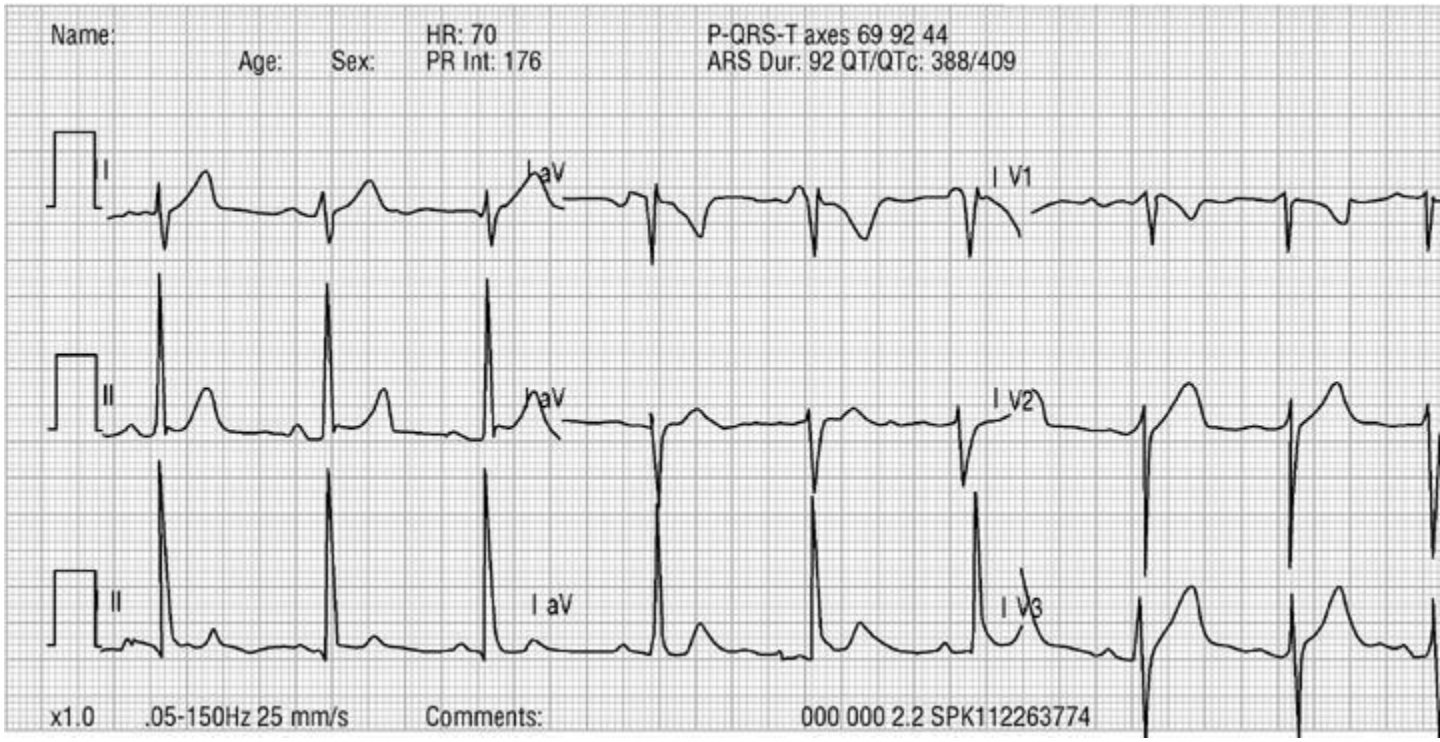
18.



19.



20.



21. Electrical conduction in the heart begins with a change in resting potential. This change stimulates which of the following nodes first?

a. SA node

b. AV node

c. Bundle of His

22. Electrical conduction in the heart follows which of the following paths?

a. SA node,> Bundle of His> Purkinge Fibers> Bundle Branch> AV node

b. SA node> AV node> Bundle of His> Purkinge Fibers> Bundle Branch

c. AV node> SA node> Bundle of His> Purkinge Fibers> Bundle Branch

23. The Sino Atrial node is also called the…

a. The pace maker of the heart

b. The electrical stimulator

c. The beat maker

24. Which of the following nodes cannot sustain life?

a. AV node

b. SA node

c. Bundle of His

25. When your patient's ECG reading is Asystole, what must you do?

a. Check that the leads are on correctly

b. Call a code

c. Turn off the monitor and turn it back on.

26. When your patient is in A Fib what should you do?

a. Call a code

b. Check the status of your patient

c. Check that the electrodes are on correctly

27. When monitoring your patient in CCU you usually pull a ….

a. 6 second strip

b. 60 minute strip

c. 60 second strip

28. Which of the four chambers of the heart has the thickest walls?

a. The right atrium

b. The right ventricle

c. The left ventricle

29. When looking at a rhythm strip you notice that the vertical direction is measured in _____

And the horizontal direction is measured in _____

a. Milli volts, seconds

b. eV, minutes

c. seconds and eV

30. When applying leads it is most preferred to …

a. Clip the hairs of the chest

b. Shave the hairs of the chest

c. Pluck the hairs of the chest

Review Questions
12-Lead ECG

1. The QRS interval should normally be _____ or smaller.
a. 0.20 sec
b. 0.11 sec
c. 0.18 sec
d. 0.36 sec
2. The point at which the QRS complex meets the ST segment is known as the:
a. Delta wave
b. End point
c. J point
d. Vector
3. ST segment depression indicates:
a. Myocardial ischemia
b. Coronary vasospasm
c. Prinzmetal's angina
d. Chronic pericarditis
4. ST segment elevation is a primary indicator of:
a. Ventricular atrophy
b. Ventricular hypertrophy
c. Myocardial injury
d. Atrial aneurysm
5. ECG changes that may be anticipated as a result of myocardial ischemia, injury, and/or necrosis of the myocardial tissues include all of the following EXCEPT:
a. PR interval prolongation
b. ST segment elevation
c. ST segment depression
d. Pathologic Q wave

6. ST segment depression may be evident on a 12-Lead ECG strip following both angina and strenuous exercise.
a. True
b. False

7. ECG changes of significance with myocardial ischemia includes ST segment depression, T wave inversion, or:
a. Depressed T wave
b. Peaked T wave
c. Peaked P wave
d. Inverted P wave
8. Inferior wall infarctions are generally associated with blockage of the:
a. Right coronary artery
b. Left coronary artery
c. Bundle of His
d. Coronary sinus
9. Myocardial infarctions may be classified as either transmural or:
a. Supraendocardial
b. Subendocardial
c. Endocardial
d. Precardial
10.If ST segment elevation is noted in the lower limb leads (Leads II, III and aVF), this finding is indicative of:
a. Anterior myocardial infarction
b. Lateral myocardial infarction
c. Superior myocardial infarction
d. Inferior myocardial infarction

11.ECG leads that record the electrical impulse formation in uninvolved myocardium directly opposite the involved myocardium are termed:
a. Facing leads
b. Viewing leads
c. Reciprocal leads
d. Endocardial leads
12.If your patient is hypotensive and exhibiting ECG changes consistent with an inferior wall injury pattern, you should consider the possibility of:
a. Right atrial infarction
b. Left atrial infarction
c. Right ventricular infarction
d. Left ventricular infarction
13.Leads V3 and V4 visualize the _____wall of the heart's left ventricle.
a. Medial
b. Lateral
c. Anterior
d. Posterior
14.Right bundle branch will obscure ECG evidence of myocardial injury on the 12-Lead ECG:
a. True
b. False
15.Which of the following statements regarding LVH is true:

a. LVH will abnormally widen the QRS complex.

b. LVH causes global ST segment depression on the 12-Lead ECG.
c. LVH causes concordant ST-T wave deflection (same polarity as QRS).
d. LVH causes discordant ST-T wave deflection (opposite polarity of QRS).

Course Review

- Normal QRS is < 0.12 seconds
- Q wave- first negative deflection (below the baseline)
- R wave- First positive deflection (above the baseline)
- S wave- First negative deflection after the R wave

T Wave

- First upward deflection after the QRS
- Represents the electrical activity during re-polarization (resting state) of the ventricles.
- Shape can be changed by electrode placement
- Can be normal (upward), inverted or even flat

PR Interval

- Represents the time it takes for the electrical current to travel from the SA node through the AV node to the top of the ventricles
- Measured from the beginning of the P wave to the beginning of the QRS complex.
- Normal PR interval is 0.12-0.20 seconds.

QT Interval

- Time of ventricular depolarization and re-polarization
- Measured from the beginning of the QRS to the end of the T wave.
- Length is inversely related to the heart rate.
- affected by disease states, drugs, electrolyte imbalances.
- prolonged QT linked to ventricular arrhythmia's
- Normal QT< 0.44 seconds

ST Segment

- Located in the space from the end of the QRS to the beginning of the T
- Normally isoelectric but can be elevated or depressed
- early re-polarization of the ventricles
- Can give valuable information about ischemia and injury on a 12 lead ECG

REMEMBER THE 7 STEPS OF INTERPRETATION

1. IS THIS A REGULAR RHYTHM?

2. WHAT IS THE HEART RATE?

3. EXAMINE THE P WAVES

4. MEASURE THE PR INTERVAL

5. EXAMINE THE QRS COMPLEXES

6. MEASURE THE QT INTERVAL

7. INTERPRET THE RHYTHM

Welcome to Tele Tech Class Review

Anatomy and Physiology

The heart lies in the center of the chest. Under the sternum and in between the lungs.

2/3's of the heart lies to the left of the sternum. It is about the size of your fist and weighs about 10.6 oz.

It is divided into 4 chambers. The 2 top chambers are called the atria. And the 2 bottom are called the ventricles

For Reference Videos and other helpful tutorials please see the below links.