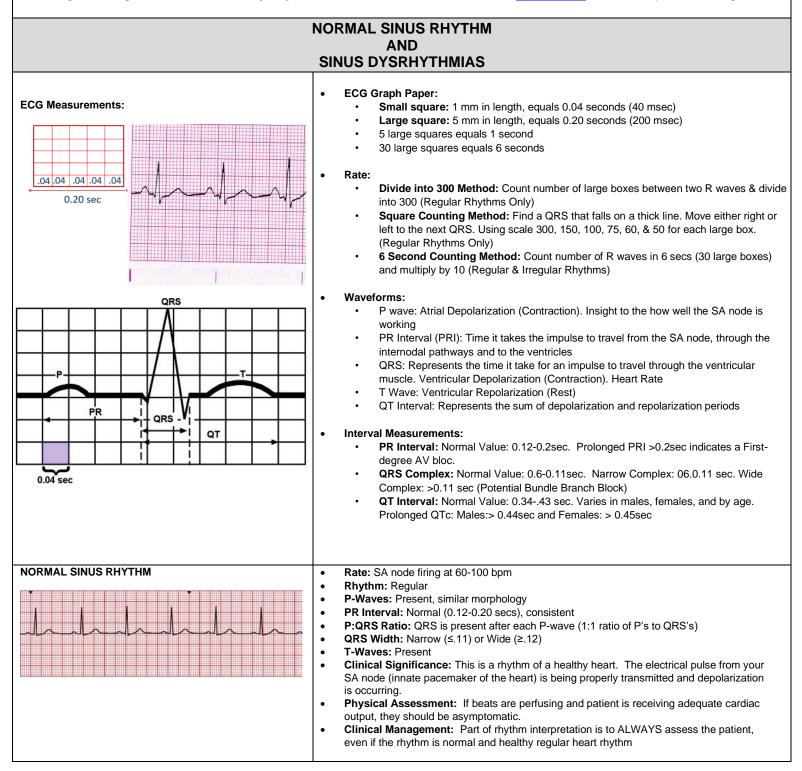
ECG Interpretation Assessment Review Packet

ASSESSMENT FORMAT

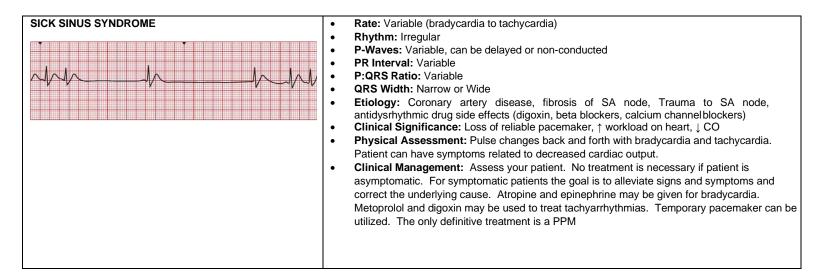
Nurses caring for adults will be provided no more than 2 hours to complete the 60-question assessment. The format of the ECG Interpretation Assessment consists of fill in the blank, multiple choice and select all that apply questions. Calipers will provide calipers for the examination. The exam will cover a variety of concepts, including but not limited to:

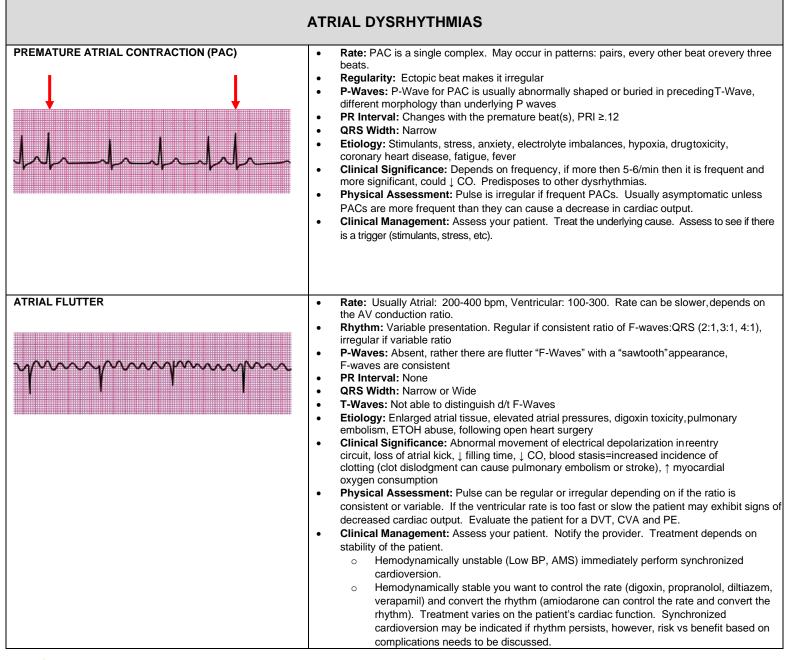
- Interval measurements: e.g. PRI, QRS, QTI
- Atrial and ventricular rhythm regularity and rate
- Nursing considerations: e.g. physical assessment, interventions, treatment
- Rhythm interpretation

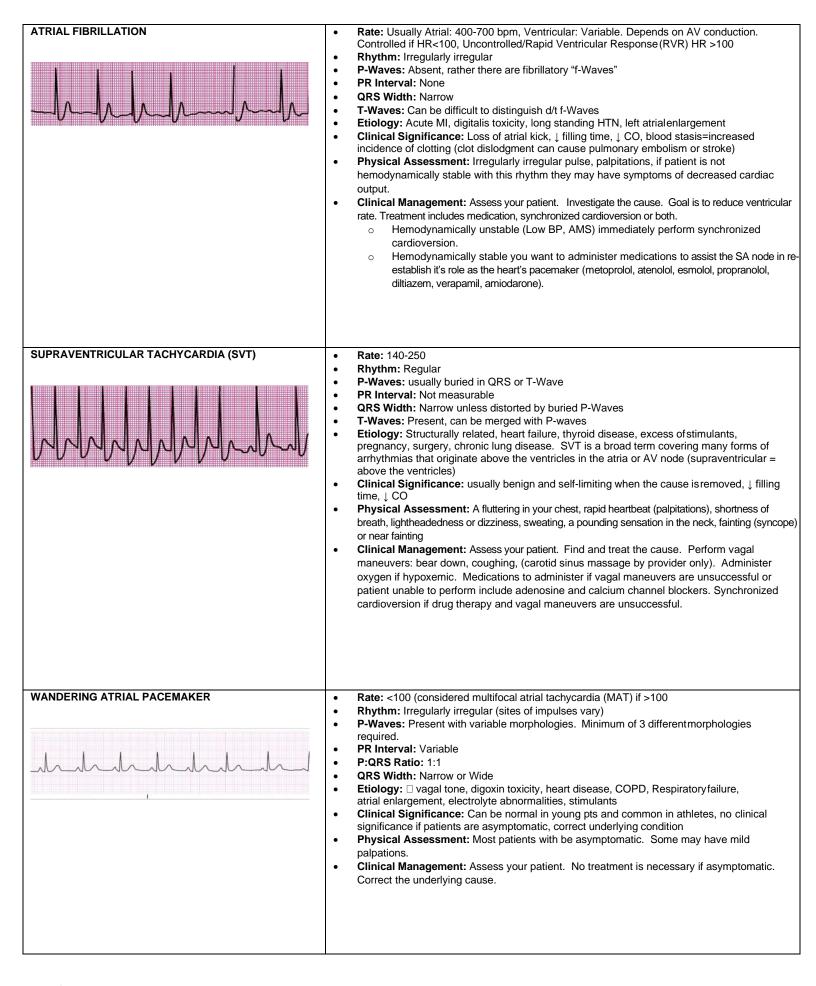
Scoring 83% or higher satisfies the "Basic Dysrthythmia Detection and Treatment" item within skills packets used in multiple work settings.



	Pate: SA pada firing at <60 hpm
	 Rate: SA node firing at <60 bpm Rhythm: Regular P-Waves: Present, similar morphology PR Interval: Normal, consistent P:QRS Ratio: 1:1 QRS Width: Narrow or Wide
	 T-Waves: Present Etiology: Ischemia/infarction of SA node, hypothermia, cardiac drugs, hypothyroidism, ↑ vagal tone, ↓ sympathetic tone Clinical Significance: Decreased Cardiac output (CO), predisposes individual to ectopic beats Physical Assessment: Slow pulse, S&S of decreased cardiac output (decreased BP, dizziness, altered mental state, syncope, weakness, fatigue, chest pain). Clinical Management: Assess your patient. Are they symptomatic? Treat if symptomatic. Treatment includes oxygen, establish IV access, find and treat the underlying problem, atropine, continuous IV inotropes if atropine not effective (dopamine
SINUS TACHYCARDIA	or epinephrine), transcutaneous pacing, permanent pacemaker (PPM) if cause cannot be reversed.
	 Rate: SA node firing at >100 bpm Rhythm: Regular P-Waves: Present, normal unless merged with T-Wave, similar morphology PR Interval: Normal, consistent
mphaman	 P:QRS Ratio: 1:1 QRS Width: Narrow or Wide T-Waves: Present, may be merged with P-Wave Etiology: Compensatory response to physiologic stressor (pain, emotions, anxiety, blood loss, hypovolemia, early sepsis, heart failure, allergic reaction, fever, exercise),
	 substances (smoking, alcohol, caffeine, drugs) Clinical Significance: ↑ oxygen consumption, ↑ workload on heart, ↓ CO if rate too fast for cardiac filling Physical Assessment: Rapid heart rate/pulse, patients usually will not have many symptoms unless they have decreased cardiac output (decreased BP, dizziness, altered mental state,
	 syncope, weakness, fatigue, chest pain) Clinical Management: Assess your patient. Are they symptomatic? Treat if symptomatic. Treatment would depend on the underlying cause. Pain (give Tylenol or Norco), hypovolemia (give fluids), blood loss (administer blood product), etc. If it is not a compensatory mechanism administer beta blockers or calcium channel blockers. Avoid stimulants.
SINUS ARRHYTHMIA/DYSRHYTHMIA	 Rate: Usually around 60-100 but can be slow or fast Rhythm: Slightly irregular, usually varies with respiration (inspiration/expiration) P-Waves: Present, similar morphology PR Interval: Normal, consistent P:QRS Ratio: 1:1 QRS Width: Narrow or Wide
	 T-Waves: Present Etiology: Common and normal finding in children and young adults, underlyingheart disease, digitalis, exercise, mental stress, circadian rhythms Clinical Significance: Usually none, can make heart more susceptible toventricular dysrhythmias Physical Assessment: Cardiac output is normal, patients usually do not experience any
	 Symptoms. Clinical Management: Assess your patient. If the bradycardia is significant administer atropine. If the patient is on digoxin, notify the MD as it may be dig toxicity.
	 Rate: Single or multiple pauses can occur Rhythm: Irregular (due to break in rhythm) P-Waves: Delayed or non-conducted (after a pause there may or may not be a P-wave) PR Interval: Normal except during the pause event P:QRS Ratio: Usually 1:1 except during the pause event QRS Width: Narrow or Wide T-Waves: Present Etiology: Hypoxemia, ischemia, diseased SA node, drugs (digoxin, betablockers), cardiac disorders, vagal stimulation Clinical Significance: Depends on patient's symptoms, if short and infrequent the patient may be symptomatic. If patient is symptomatic can have symptomsof ↓ CO. Pause may be followed by a Junctional Escape Beat (JEB) to prevent cardiac standstill. Physical Assessment: A prolonged pause causes an irregular pulse. Recurrent pauses or long pauses may cause signs of decreased cardiac output.
	 Clinical Management: Assess your patient. Find the underlying cause. Treat if symptomatic. Goal of treatment is to maintain sufficient cardiac output. Medications such as atropine or epinephrine can be administered. Emergent treatment includes temporary pacing.



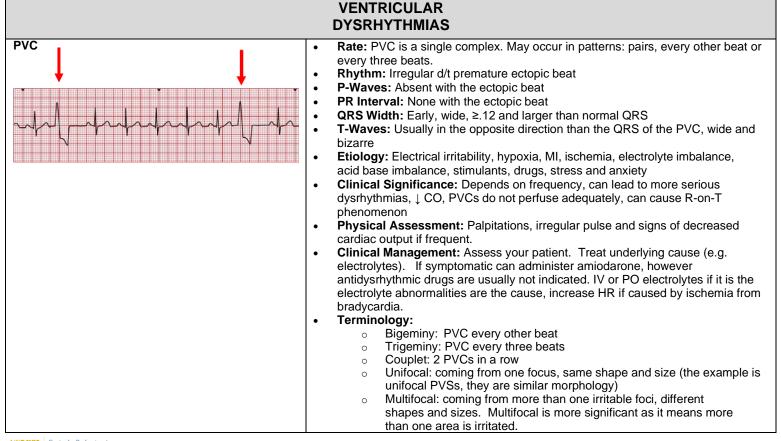




JUNCTIONAL DYSRHYTHMIAS		
PJC	 Rate: PJC is a single complex. May occur in patterns: pairs, every other beat or every three beats. Rhythm: The ectopic beat makes it irregular P-Waves: Variable for the ectopic beat. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted). PR Interval: Variable for ectopic beat, <0.12 if P-wave present before QRS P:QRS Ratio: Variable for ectopic beat, 1:1 if P-wave before QRS QRS Width: Narrow unless distorted by P-wave T-Waves: Present Etiology: Digoxin toxicity, excessive caffeine intake, inferior MI, rheumaticheart disease, valvular heart disease, hypoxia, swelling of AV junction Clinical Significance: Depends on frequency and if patient is symptomatic, if more then 5-6/min then it is frequent and more significant, could ↓ CO. Predisposes to other dysrhythmias. May have a pause after a PJC Physical Assessment: Usually asymptomatic but may have palpations. Irregular pulse and my experience decreased cardiac output if frequent. Clinical Management: Assess your patient. Does not require treatment unless symptomatic. Find and treat the underlying cause. 	
JUNCTIONAL RHYTHM/JUNCTIONAL ESCAPE RHYTHM	 Rate: 40-60 bpm Rhythm: Regular P-Waves: Variable. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted). PR Interval: Variable, short if P-wave present before QRS P:QRS Ratio: Variable, 1:1 if P-wave before QRS QRS Width: Narrow unless distorted by P-wave T-Waves: Present Etiology: Sick sinus syndromes, vagal stimulation, electrolyte imbalances, digoxin toxicity, inferior wall MI, rheumatic heart disease Clinical Significance: ↓ HR and ↓ CO Physical Assessment: Slow regular pulse, usually asymptomatic unless there is decreased cardiac output Clinical Management: Assess your patient. Find the cause. Treat if symptomatic. Administer atropine to increase the heart rate. Temporary pacemaker if patient remains symptomatic. 	
ACCELERATED JUNCTIONAL RHYTHM	 Rate: 60-100 bpm Rhythm: Regular P-Waves: Variable. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted). PR Interval: Variable, short if P-wave present before QRS P:QRS Ratio: Variable, 1:1 if P-wave before QRS QRS Width: Narrow unless distorted by P-wave T-Waves: Present Etiology: Digoxin toxicity, hypokalemia, hypercalcemia, inferior and posteriorMI, rheumatic heart disease, valvular heart disease Clinical Significance: loss of atrial kick and ↓ filling time if retrograde, ↓ CO Physical Assessment: May be asymptomatic. May experience signs of decreased cardiac output. Pulse is regular. Clinical Management: Assess your patient. Correct the underlying cause. If symptomatic and not improving, consider temporary pacing. 	
	 Rate: 100-130 bpm P-Waves: Variable. 3 different P-wave presentations are possible (before, during or after QRS). Can be positive or negative deflection (inverted). PR Interval: Variable, short if P-wave present before QRS P:QRS Ratio: Variable, 1:1 if P-wave before QRS QRS Width: Narrow unless distorted by P-wave T-Waves: Present Etiology: inferior or posterior MI, SA node disease, hypoxia, ischemia, digoxintoxicity, □ vagal tone Clinical Significance: loss of atrial kick and ↓ filling, ↓ CO, predisposes to other dysrhythmias, less dependable pacemaker, predisposes to heart failure Physical Assessment: May be asymptomatic. May experience signs of decreased cardiac output, fluttering sensation, pulse is regular and fast. Clinical Management: Assess your patient. Find and treat underlying cause. Treat decreased cardiac output by slowing heart rate with beta blockers, calcium channel blockers or digoxin. Consider vagal maneuvers and cardioversion. PPM or catheter ablation if rhythm continues. 	

FIRST DEGREE AV BLOCK	 BLOCKS Rate: Variable, atrial rate = ventricular rate Rhythm: Regular P-Waves: Present, similar morphology PR Interval: Prolonged >.20, consistent P:QRS Ratio: 1:1 QRS Width: Narrow or Wide T-Waves: Present Dropped Beats: No Etiology: Ischemia, myocardial infarction, rheumatic heart disease, CAD, cardiac medications (digoxin, beta blockers, calcium channel blockers) Clinical Significance: Not dangerous by itself, typically no symptoms, predisposes to progression to more serious block Physical Assessment: Usually asymptomatic. There is no impact on cardiac output as there is still atrial kick. Decreased cardiac output would be related to the rate of the rhythm. Clinical Management: Assess your patient. Treat the underlying cause. Watch for progression to second degree AV block.
SECOND DEGREE AV BLOCK TYPE I (MOBITZ I, WENCKEBACH)	 Rate: Ventricular rate is less then atrial rate d/t dropped QRS complexes Rhythm: Variableatrial is regular, ventricular is irregular d/t non-conducted beat P-Waves: Present, similar morphology PR Interval: Variable, progressively gets longer before dropping a QRS complex. The shortest PRI is the one immediately following the dropped beat. The longest PRI is the one immediately before the dropped beat. P:QRS Ratio: Variable, P>QRS QRS Width: Narrow or Wide Dropped Beats: Yes Etiology: Ischemia, MI, CAD, drug toxicity such as digoxin, rheumatic fever, myocarditis, transient after heart surgery, cardiac medications (beta blockers, calcium channel blockers) Clinical Significance: Depends on the frequency of dropped QRS complexes, ↓ CO, predisposes to progression to more serious block Physical Assessment: Pulse is irregular d/t dropped beads. If frequently dropped beats can experience signs of decreased cardiac output. Clinical Management: Assess your patient. Treat if symptomatic with atropine to improve AV node conduction. Temporary pacemaker for long term relief if symptoms persist. Find and treat underlying cause.
	 Rate: Ventricular rate is less then atrial rate d/t dropped QRS complexes Rhythm: Variableatrial is regular, ventricular is regular or irregular depending on number of blocked complexes P-Waves: Present, similar morphology PR Interval: Normal and consistent P:QRS Ratio: Variable, P>QRS. There can be a pattern of conducted beats to dropped beats (example: 2:1—2 P waves for every QRS) QRS Width: Narrow or Wide Dropped Beats: Yes Etiology: Septal wall necrosis, anterior septal MI, myocarditis, CAD, drug toxicity, cardiomyopathy Clinical Significance: Depends on the frequency of dropped QRS complexes, ↓ CO, predisposes to progression to more serious block, unpredictable rhythm Physical Assessment: Pulse is slow due to dropped beats. Symptoms of decreased cardiac output if present. Clinical Management: Assess your patient. Notify provider immediately as it can deteriorate fast. If asymptomatic usually will observe. If symptomatic temporary or permanent pacemaker. Medications include atropine, dopamine, and epinephrine for bradycardia.

THIRD DEGREE AV BLOCK (COMPLETE HEART BLOCK)	 Rate: Variable. Atrial rate usually 60-100 and ventricular rate 40-60 if AV node, 20-40 if Purkinje fibers are pacemaker cells Rhythm: Regular, P-waves will march out, QRS complexes will march out. The P-waves and QRS complexes have no correlation. P-Waves: Present, similar morphology PR Interval: Variable P:QRS Ratio: P> QRS, there is no correlation between P-wave and QRS QRS Width: Narrow or Wide Etiology: MI, digoxin toxicity, acute myocarditis, degenerative changes in the heart, calcium channel blockers, beta-adrenergic blockers, cardiac surgery Clinical Significance: Slow ventricular rate, ↓ CO, unreliable pacemaker, predisposes to lethal dysrhythmias, severity of symptoms depends on ventricular rate, may progress to asystole Physical Assessment: Pulse is regular and slow, most all patients are symptomatic with signs of decreased cardiac output. Severity of symptoms will depend on ventricular rate. Clinical Management: Assess your patient. Correct underlying cause. Make sure patient has IV access, administer oxygen, support cardiac output. Maintain bedrest to decrease oxygen demand. Administer atropine or isuprel to restore synchrony (use with caution), administer dopamine and epinephrine if indicated. Transcutaneously pace until temporary or permanent pacer can be placed.
BUNDLE BRANCH BLOCK	 Rate: Abnormal conduction in ventricle, can be right or left side of the pathway. QRS Width: Wide ≥.12 Right BBB: V1-Rabbit ear appearance with RSR' and inverted T-Wave; V6-widened S-Wave and upright T-wave Left BBB: V1- Wide S-Wave and positive T-Wave; V6- notch at top of QRS, inverted T-Wave Etiology: Right—congenital, anterior MI, CAD, PE. Left—hypertension, aortic stenosis, heart disease, CAD Clinical Significance: LBBB more serious than RBBB, examine V1 to determine if right or left, need 12 lead to diagnose Physical Assessment: Typically, there are no physical findings Clinical Management: Assess your patient. Usually, no treatment is necessary. Determine the underlying cause, watch for other heart blocks.



 Rate: 100-250 bpm (Monomorphic) Rhythm: Regular (may be slightly irregular at onset) P-Waves: Absent PR Interval: None P:QRS Ratio: Absent QRS Width: Wide and bizarre Etiology: Myocardial ischemia, MI, CAD, valvular heart disease, heart failure, cardiomyopathy, electrolyte imbalances, drug intoxication, stress, anxiety Clinical Significance: Very unpredictable and dangerous. Unreliable pacemaker. ↓ ventricular filing time, ↓ CO, quickly deteriorate to V Fib and complete cardiac failure. There are 3 presentations Stable VT with pulse Unstable VT with pulse VT without a pulse Physical Assessment: depends on the duration of the rhythm and if the patient is stable or unstable. They may have a flutter in their chest, anxiety and signs of decreased cardiac output. Clinical Management: Assess your patient. Treatment depends on if the patient is stable or unstable and with or without a pulse Pulse is PRESENT: If patient is hemodynamically STABLE, notify provider and expect drug intervention (amiodarone, lidocaine, procainamide) and a cardiology consult If patient is hemodynamically UNSTABLE synchronized cardioversion. Follow AHA Ventricular Tachycardia WITH a pulse algorithm Pulse is ABSENT call a code, start CPR, perform immediate defibrillation. Follow AHA Ventricular Tachycardia WITHOUT a pulse algorithm
 Rate: 150-250, Torsades is a variant of Polymorphic Ventricular Tachycardiac caused by a prolonged QT Rhythm: Irregular P-Waves: Absent PR Interval: None P:QRS Ratio: Absent QRS Width: Wide with changing amplitudes Etiology: Prolonged QT from drugs, myocardial ischemia, electrolyte abnormalities—hypokalemia and hypomagnesemia Clinical Significance: dangerous rhythm that can lead to ventricular standstill Physical Assessment: depends on the duration of the rhythm and if the patient is stable or unstable. They may have a flutter in their chest, anxiety and signs of decreased cardiac output. Clinical Management: Assess your patient. Treatment depends on if the patient is stable or unstable and with or without a pulse. Administer magnesium for prolonged QT, K+ level, overdrive ventricular pacing may be necessary. Pulse is PRESENT: If patient is hemodynamically STABLE, notify provider and expect drug intervention (amiodarone, lidocaine, procainamide) and a cardiology consult If patient is hemodynamically UNSTABLE synchronized cardioux pousult If patient is nemodynamically UNSTABLE synchronized cardioux pousult Pulse is ABSENT call a code, start CPR, perform immediate defibrillation. Follow ACLS Protocol for Ventricular Tachycardia WITHOUT a pulse.

VENTRICULAR FIBRILLATION	 Rate: Absent Rhythm: Irregular P-Waves: Absent
mmmmmmmm	 PR Interval: None PRS Ratio: Absent QRS Width: None Etiology: Myocardial ischemia, MI, heart block, untreated V tach, acid base imbalance, electrolyte imbalance, drug toxicity, severe hypoxia, terminal event in
	 many disease states Clinical Significance: Ventricles are quivering instead of contracting, cardiac output is nonexistent, pacemaker cells are still working, leads to ventricular standstill and cardiac death
	 Physical Assessment: Unresponsive, no blood pressure, no pulse, apneic Clinical Management: Assess your patient. Call a code, start CPR, perform early defibrillation. Follow ACLS Protocol for Ventricular Fibrillation.
PULSELESS ELECTRICAL ACTIVITY Any organized rhythm will be present on the monitor (wave forms are visible) but the patient will NOT HAVE A PULSE	 Rate, Rhythm, P-Waves, PR Interval, P:QRS Ratio, QRS Width, T-Waves: Variable—All based on the underlying rhythm Etiology: Look at the H's and T's: H—hypovolemia, hypoxia, hydrogen ions, hyperkalemia, hypokalemia, hypothermia. T—Tablets, toxins, tension pneumothorax, thrombosis, thromboembolism, trauma Clinical Significance: Heart muscles loses its ability to contract even though electrical activity is preserved. No heart contraction, no blood flow, no heart rate, no cardiac output Physical Assessment: No pulse, no heart sounds, no BP, unconscious, apneic Clinical Significance: No pulse, no heart sounds, no BP, unconscious, apneic
IDIOVENTRICULAR RHYTHM	Clinical Management: Assess your patient. Call a code and start CPR. Follow ACLS Protocol for PEA including epinephrine and finding and treating the cause.
	 Rate: 20-40 bpm, if 40-100 then it is accelerated idioventricular rhythm, >100 is Ventricular Tachycardia Rhythm: Regular (ventricular rhythm only, no atrial rhythm) P-Waves: Absent PR Interval: None P:QRS Ratio: None
	 QRS Width: Wide ≥.12 T-Waves: Deflected opposite of QRS Etiology: Digoxin toxicity, heart disease, pacemaker failure, metabolic imbalance, common following reperfusion therapy Clinical Significance: Slow ventricular rate and loss of atrial kick reduce CO, progress to more lethal dysrhythmia. If just one idioventricular beat is
	 generated it is called a ventricular escape beat Physical Assessment: palpations, signs of decreased cardiac output. Clinical Management: Assess your patient. Treatment goals are to increase heart rate, improve cardiac output and establish a normal rhythm. Medications administered include atropine to increase the heartrate. Dopamine and epinephrine can also be used to increased heartrate. Transcutaneous pacing may be utilized emergently until a temporary or permanent pacemaker can be inserted.
ASYSTOLE	 Rate: None Rhythm: None
	 P-Waves: None PR Interval: None PRS Ratio: None QRS Width: None T-Waves: None
	 Etiology: MI, severe electrolyte disturbance, massive PE, prolonged hypoxemia, severe uncorrected acid-base imbalance, electric shock, drug overdose, cardiac tamponade, hypothermia Clinical Significance: atrial and ventricular activity is at a standstill, terminal
	 rhythm, critical to determine if rhythm is true asystole or a pause, need to lookin a minimum of 2 leads Physical Assessment: No pulse, no heart sounds, no BP, unconscious, apneic Clinical Management: Assess your patient, confirm in 2 lears. Call a code, Start CPR. Follow ACLS Protocol for Asystole including epinephrine and finding and treating the cause.





VENTRICULAR PACED



DUAL CHAMBER PACED



PACEMAKER RHYTHMS

Type of Pacemakers:

- Atrial: Pacemaker spike is followed by atrial depolarization (P-wave)
- Ventricular: Pacemaker spike is followed by ventricular depolarization (QRS)
- Dual: There will be an atrial pacing and ventricular pacing

Pacemaker Sensing Definitions:

- Undersensing: Pacemaker has failed to sense that the heart has initiated an intrinsic beat.
 - Pacing when it shouldn't
 - Pacemaker <u>did not sense the patient's own intrinsic beat.</u> This causes the pacemaker to send an impulse when it shouldn't. An impulse is sent, and extra cardiac depolarization can occur. This impulse can occur during the intrinsic beat of the patient, predisposing the patient to R-on-T phenomenon.
- Oversensing: Electrical activity is registered as cardiac activity that is not coming intrinsically and is counted as a cardiac beat
 - Not pacing when it should
 - Pacemaker <u>sensed non-cardiac activity and interpreted it as an</u> <u>intrinsic beat</u>. Pacemaker impulse is inhibited as the pacemaker falsely sensed the patient as having an intrinsic beat. An impulse is not sent, and cardiac depolarization does not occur. This leads to an overall decrease in heart rate and cardiac output.

Pacemaker Capture Definitions:

• Capture: When the pacemaker delivers an electrical impulse strong enough to result in depolarization.

 100% capture occurs when each pacemaker impulse produces a corresponding depolarization (atrial pacemaker impulse results in a P-wave, ventricular pacemaker impulse results in a QRS Complex). This means that the pacemaker is appropriately capturing/depolarizing.

• Failure to capture: When the pacemaker delivers an electrical impulse that does not result in depolarization.

 The pacemaker sends an impulse, the pacer spike is visible, however, no corresponding waveform is present following the pacer spike (no p-wave for an atrial pacemaker, no QRS complex for a ventricular).

• Failure to pace: No electrical impulse is delivered, complete failure to sense the need or deliver the impulse to initiate pacing.

• The pacemaker fails to send a pacing impulse when appropriate and no visible pacing spike is seen.