

VENTRICULAR DYSRHYTHMIAS

Pacemakers

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Reading assignments:

Bledsoe Vol 3; pp. 96-109

SOPs: VT with pulse; Ventricular fibrillation/PVT; Asystole/PEA

Drugs: Amiodarone, magnesium, epinephrine 1mg/10mL

Procedure manual: Defibrillation; Mechanical Circulatory Support (MCS) using a Ventricular Assist Device

KNOWLEDGE OBJECTIVES:

Upon completion of the reading assignments, class and homework questions, reviewing the SOPs, and working with their small group, each participant will independently do the following with at least an 80% degree of accuracy and no critical errors:

1. Identify the intrinsic rates, morphology, conduction pathways, and common ECG features of ventricular beats/rhythms.
2. Identify on a 6-second strip the following:
 - a) Idioventricular rhythm
 - b) Accelerated idioventricular rhythm
 - c) Ventricular tachycardia: monomorphic & polymorphic
 - d) Ventricular escape beats
 - e) Premature Ventricular Contractions (PVCs)
 - f) Ventricular fibrillation
 - g) Asystole
 - h) Paced rhythms
 - i) Intraventricular conduction defects (Bundle branch blocks)
3. Systematically evaluate each complex/rhythm for the following:
 - a) Rate (atrial and ventricular)
 - b) Rhythm: Regular/irregular - R-R Interval, P-P Interval
 - c) Presence/absence/morphology of P waves
 - d) Presence/absence/morphology of QRS complexes
 - d) P-QRS relationships
 - f) QRS duration
4. Correlate the cardiac rhythm with patient assessment findings to determine the emergency treatment for each rhythm according to NWC EMSS SOPs.
5. Discuss the action, prehospital indications, side effects, dose and contraindications of the following during VT
 - a) Amiodarone
 - b) Magnesium
6. Describe the indications, equipment needed, critical steps, and patient monitoring parameters for cardioversion and defibrillation.
7. Identify the management of a patient with an implanted defibrillator and/or pacemaker.

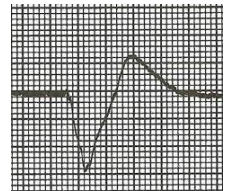
NCH Paramedic Program
VENTRICULAR DYSRHYTHMIAS
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I. Dysrhythmias originating in the ventricles

- A. Ventricular dysrhythmias are frequently unstable and unpredictable. These dysrhythmias are also potentially lethal, because the heart does not provide a synchronous or coordinated contraction so SV and coronary flow are compromised.
- B. Ventricular complexes and rhythms – Idioventricular rhythm (IVR)
- C. Accelerated idioventricular rhythm (AIVR)
- D. Premature ventricular contractions (PVCs)
- E. Ventricular tachycardia: monomorphic and polymorphic
- F. Ventricular fibrillation
- G. Asystole
- H. Artificial pacemaker rhythm

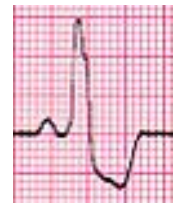
II. Characteristics of a ventricular complex (beat)

- A. There is no regular activity in the atria prior to ventricular depolarization, so there are generally no regular P waves.
- B. **Wide QRS complexes:** Impulse originates in an escape pacemaker site in the ventricles that takes longer than the normal time to conduct - so QRS will be wide (0.12 seconds or greater), distorted and bizarre.



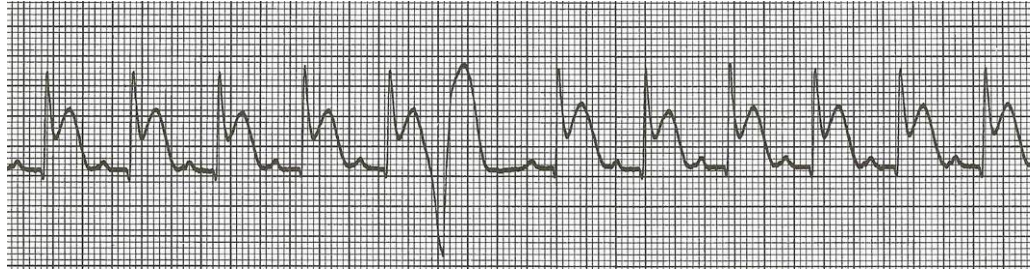
Other causes of wide QRS complexes

- 1. Bundle branch block (BBB): It is impossible to differentiate an intraventricular conduction deficit from a bundle branch block without a 12 L ECG, so we tend to assume that all wide complex QRSs on a rhythm strip when a P wave is present reflect an IVCD until proven otherwise.
 - 2. Electrolyte abnormality (hyperkalemia) (tall, pinched at the top)
 - 3. Paced rhythm
 - 4. Drug effect (QT prolongation)
 - 5. Prior cardiac surgery
 - 6. Normal variable
- C. ST segment and T wave of a ventricular beat often deflect in the **opposite direction** of the QRS complex. Depolarization is abnormal and so is repolarization.
 - D. A low QRS voltage is different from a wide QRS and is a relatively non-specific finding on a screening ECG; the differential diagnosis is broad:
 - 1. Pericardial effusion
 - 2. Myocardial infarction
 - 3. Cardiomyopathy
 - 4. Hypothyroidism
 - 5. Obesity
 - 6. Sarcoidosis
 - 7. Amyloidosis
 - 8. Chronic obstructive pulmonary disease (COPD)
 - 9. Anasarca



III. Premature ventricular contractions (PVC)

- A. **Description** - Early ventricular beat: Electrical impulse originates early from an ectopic focus somewhere in one of the ventricles. It is usually caused by enhanced automaticity. The impulse depolarizes the ventricles abnormally creating a premature QRS complex with an abnormal morphology or look, that occurs before the next expected sinus or junctional beat.
- B. **Characteristics of PVCs**
 - 1. Rhythm: Portion of the strip with the PVC is irregular



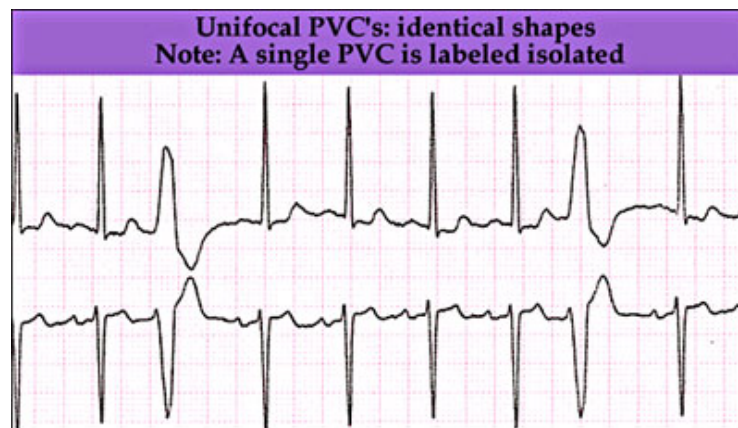
2. Rate: Depends on underlying rhythm (sinus, atrial or junctional)
3. P waves
 - a. None associated with the PVC (beat is not initiated by a P wave)
 - b. If P waves are present, they are usually associated with the underlying rhythm. P wave from the underlying rhythm may be seen preceding the QRS of a PVC or may be seen after the PVC in the ST segment or T wave. May have retrograde depolarization of the atria, so P wave may be inverted.
4. P-R interval: None with the PVC as beat is initiated in the ventricle
5. QRS complex
 - a. Premature complex is always wide (0.12 sec or greater) and abnormal in appearance because depolarization originates in the ventricle and follows an abnormal pathway.
 - b. It may be notched and looks different from the normal QRS complexes.
6. The ST segment and T wave are deflected in the opposite direction from the QRS complex of the PVC.
7. The PVC fails to conduct retrograde through the AV node in half of patients, so it does not depolarize the SA node, thus the discharge timing of the SA node remains the same and the basic rhythm will resume on time after the PVC.

This usually results in a **full compensatory pause** after the PVC. That is, the next P wave occurs at the same time as would be expected had the PVC not occurred. When it does conduct through the AV node, the following P wave may occur either sooner or later than would be expected.

Full compensatory pause: The measurement between the R wave preceding the PVC and the R wave after the PVC is equal to two R-R intervals of the underlying regular rhythm.

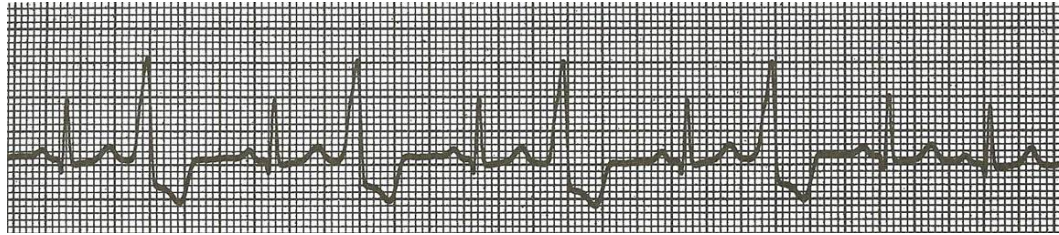
C. Possible presentations

1. **Isolated:** Minimal significance

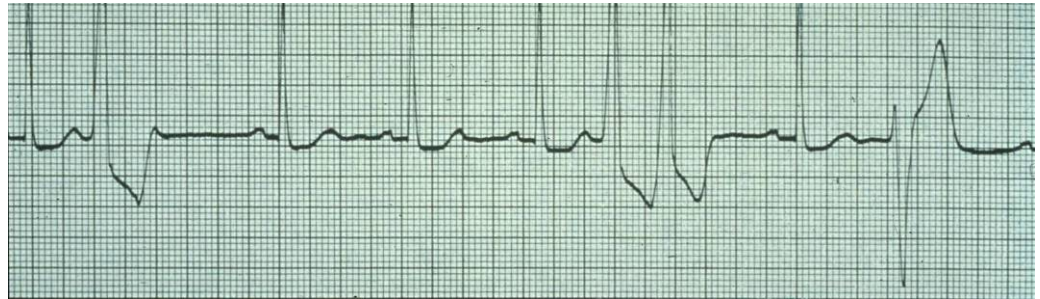


2. **Frequent** (more than 6 per minute)
 - a. Indicates increased ventricular irritability
 - b. Could lead to more lethal dysrhythmia

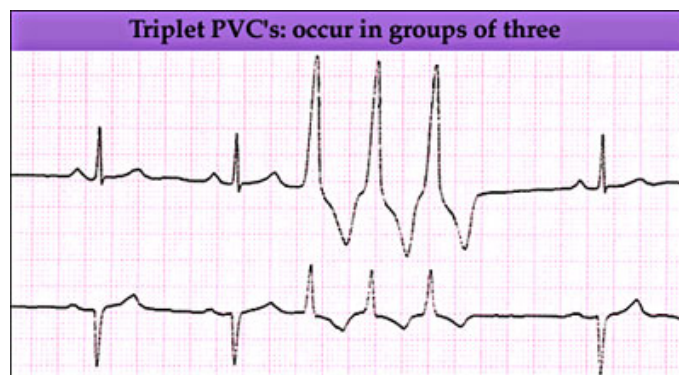
3. **Patterns: If the PVC occurs..**
- a. Every other beat (**ventricular bigeminy**)
 - b. Every 3rd beat (**ventricular trigeminy**)
 - c. Every 4th beat (**ventricular quadrigeminy**)



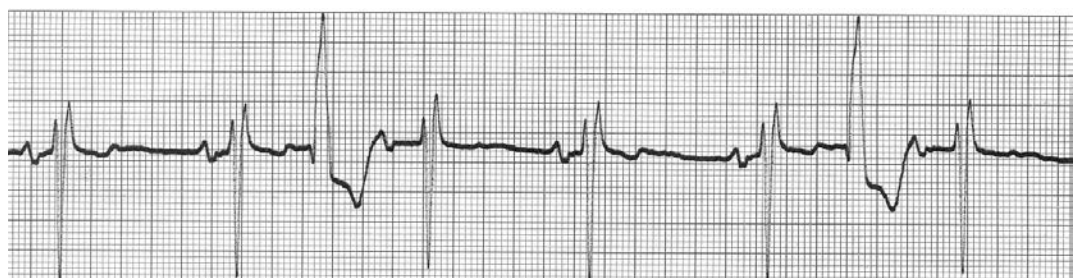
- d. **Couplets or pairs:** 2 PVCs in a row



- e. **Triplets or salvo:** 3 or more PVCs in a row (short run of V-tach)



- f. **Interpolated:** A PVC sandwiched between two normally conducted beats without disturbing the regularity of the underlying rhythm. No compensatory pause.



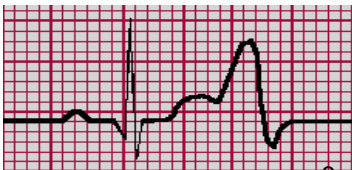
4. **Uniform or multiformed**

- a. Uniform PVCs usually are identical in size, shape, and direction to each other.
- b. If 2 PVCs within one 6 second strip look different they are called **multiformed**. The text also refers to them as multifocal. This may indicate that two or more sites are originating the ectopic beats, but respected cardiologists disagree that a different look always means another ectopic focus.



5. **"R on T" phenomenon**

- a. PVC (R wave) appears on or near the peak of a T wave during the vulnerable period of repolarization.
- b. Stimulation of the ventricle at this point may result in repetitive ventricular contractions and may precipitate VT or VF & death.

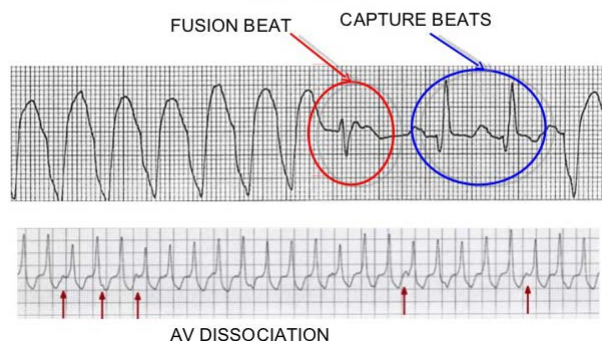


6. **Fusion beats**

- a. Fusion beats are usually a combination of an SA impulse and PVC, or a SA impulse and an artificial pacemaker impulse occurring at the same time.
- b. Fusion or capture complexes confirm the coexistence of supraventricular and ventricular originated beats. The same chamber of the heart is depolarized by 2 simultaneous impulses leading to blending or fusion of the two. The two impulses are both trying to control the heart at same time and the complex has characteristics of the supraventricular and the ventricular beat.
- c. The first part of the complex looks normal because of the sinus influence, but the last half is abnormal. The fusion complex is usually more narrow than a PVC.
- d. Look at both the normal and the PVC beats and then look at the fusion beat to differentiate this dysrhythmia.
- e. Pacemakers produce ventricle foci so a fusion beat is expected in patients with pacemakers. Fusion beats are also seen with ventricular ectopy so their presence can be used to help differentiate SVT with aberrancy from VT.

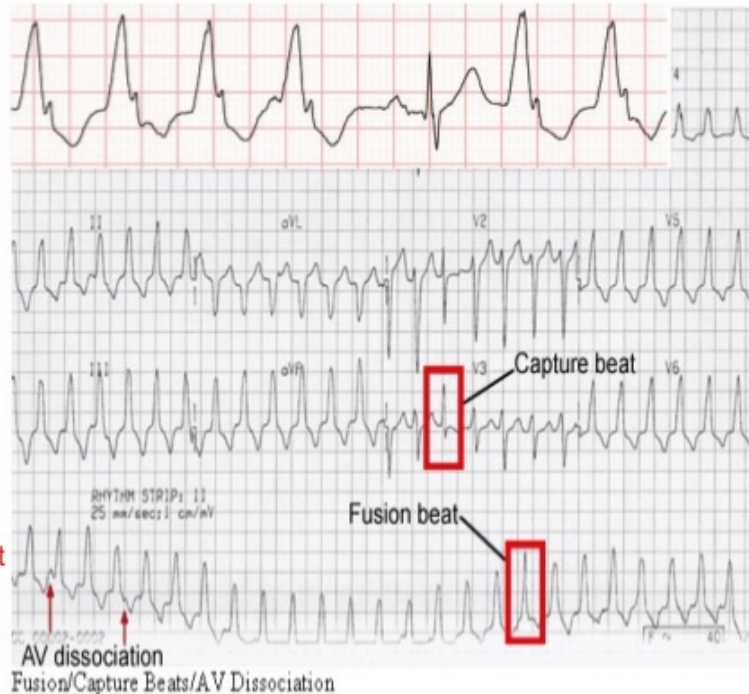
Wide Complex Tachycardias

ECG Findings Suggestive of VT



Capture beat

- When there is interference dissociation between sinus rhythm and faster subsidiary rhythm, the interference occurs in the AV node.
- Both the impulses cannot be conducted due to the refractoriness of the AV node as a result of the wave from each focus.
- At a particular time the slow sinus waves pass through the AV node when it is **no longer refractory** and hence **conducts down the ventricles..ventricular capture beat..momentary activation of the ventricles by sinus impulse in AV dissociation.**



D. Etiology of PVCs

1. Seen in 50-63% of healthy individuals and in 72-93% of post MI patients.
2. Anxiety, stress, excessive caffeine, tobacco or alcohol consumption
3. Hypoxia; acidosis
4. Drugs: Digitalis, epinephrine, isoproterenol, aminophylline; sympathomimetic drugs such as some over-the-counter cold remedies
5. Electrolyte imbalances: \downarrow K; \downarrow Mg
6. Ischemia/MI, HF, ACS, valvular disease (mitral valve prolapse), cardiomyopathy
7. After heart surgery or contact of the endocardium with catheters (pacing leads, PA catheters)
8. More frequent with age
9. Carboxy-Hb of 4-6% that is seen in cigarette smokers is associated with numbers of PVCs in CV patients during exercise. Atmospheric pollution produces similar dysrhythmias.

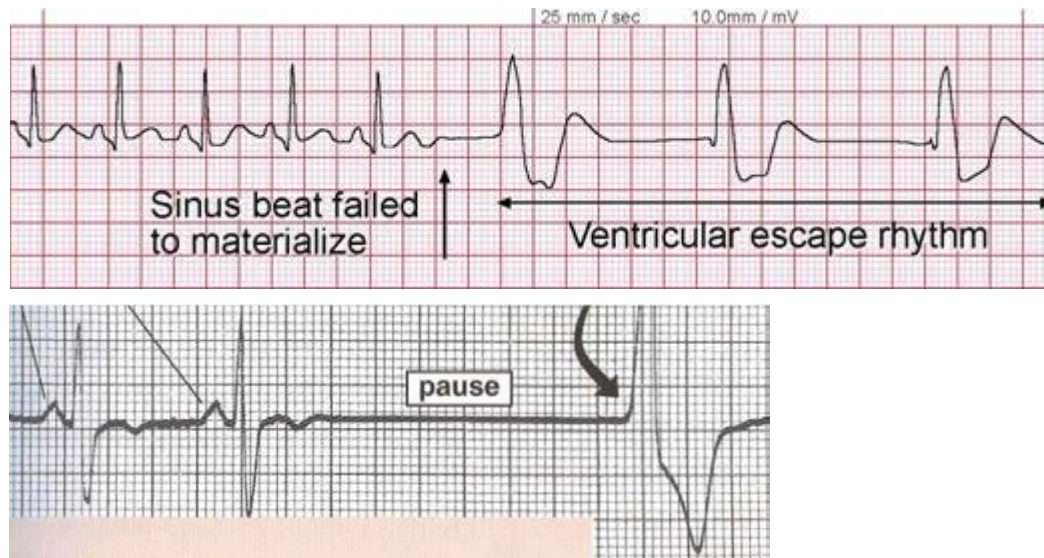
E. Clinical significance: In ACS

1. Patients may be unaware that they are having PVCs, but they may have palpitations, skipped beats, dizziness, SOB, chest discomfort, angina, or hypotension.
2. Indicates increased ventricular irritability
3. In patient who have had an MI, risk of malignant ventricular arrhythmias and sudden death is related to complexity and frequency of PVCs with patients in Lown Classes 3-5 at greatest risk
 - a. Grade 0 No premature beats
 - b. Grade 1 Occasional (<30 /hour)
 - c. Grade 2 Frequent (>30 h)
 - d. Grade 3 Multifocal
 - e. Grade 4 Repetitive (A=Couplets, B=Salvos of ≥ 3)
 - f. Grade 5 R on T pattern (www.afghanheart.wordpress.com)

F. **Treatment - ACS SOP**

1. IMC
2. Look for underlying reversible causes
3. If new, Rx ischemia with ASA and NTG unless contraindicated
4. All antidysrhythmic drugs have a certain proarrhythmic effect and may worsen the rhythm and induce torsades de pointes from QT prolongation. Therefore, drugs are no longer given to suppress PVCs.

IV. **Ventricular escape beats**



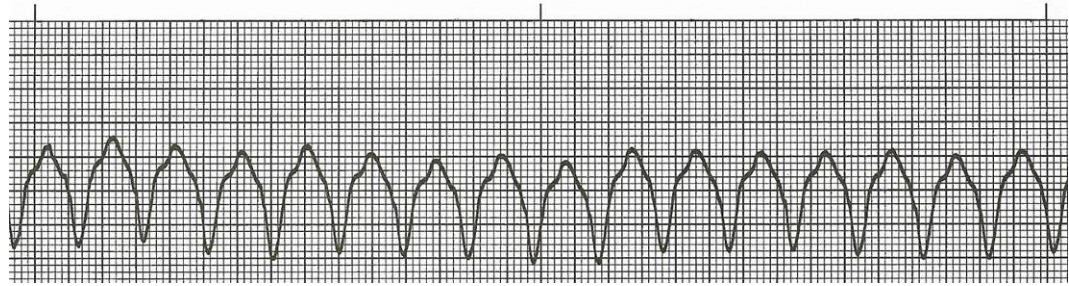
- A. Just like the AV node, the ventricle can serve as an escape pacing site if higher pacemakers fail to fire. This will result in a ventricular escape beat that comes *late* in the cardiac cycle.
- B. Etiology: Increased vagal tone on the SA node

V. **Ventricular tachycardia**

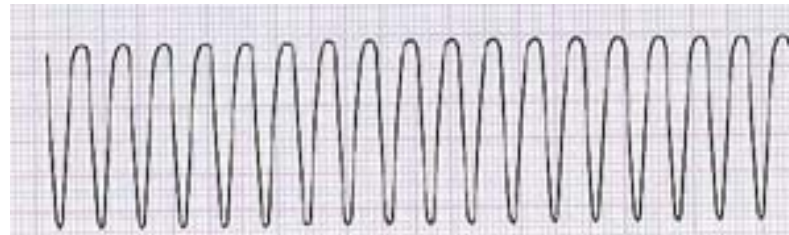
A. **Description**

1. Rhythm originates from a single irritable focus within the ventricles. May develop without warning, but often follows frequent or dangerous PVCs.
2. Ventricular beats (wide QRS with no associated P waves) discharge at a rapid rate over 100 times per minute; usually between 140-250.
3. The rhythm is usually associated with enhanced automaticity or reentry.
4. The ventricular beats do not produce a P wave. However, the sinus node continues to beat independently, and sinus P waves may occasionally be seen between the wide QRS complexes. They are usually hidden in the QRS complexes.
5. QRS complexes should look alike (monomorphic). When they look different, the VT is called **polymorphic**.
6. There is a literature supporting that 80-90% of wide tachycardias when you don't see P waves are VT (*regardless of whether the patient is asymptomatic with good BP or not*). This goes up to over 90% if the patient has a history of heart disease or prior VT (Ken Grauer, 2011).
7. VT may be a **sustained** rhythm (lasting longer than 30 seconds) or occur in short bursts or paroxysms. Three PVCs in a row is a run of nonsustained VT. Nonsustained VT should be tolerated well but can progress into sustained VT.

B. **Interpretation MONOMORPHIC Regular VT**



1. Rate: 101 (140)-250. (If VT occurs at rates > 250 / minute, the QRS complexes appear sawtoothed and the rhythm is called ventricular flutter). This is usually a forerunner to VF. When ventricular tachycardia becomes very fast (200-300 bpm), and you can no longer tell if it is QRS complex, a T wave, or an ST segment, then you have ventricular flutter. V-Flutter can quickly deteriorate into V-Fib



2. Rhythm: Regular to slightly irregular
3. P waves: Often not visible, but may be present (dissociated from QRS). Atrioventricular dissociation during a wide QRS tachycardia is a hallmark of ventricular tachycardia
4. P-R interval: None
5. QRS complex
 - a. Wide (0.12 seconds or greater)
 - b. T waves: If seen, they are the opposite polarity from QRS
 - c.

C. **Etiology**

1. Hypoxia; acidosis; **CAD, myocardial ischemia or infarction**
2. Underlying heart disease: Cardiomyopathy, mitral valve prolapse, HF
3. Digitalis toxicity
4. Drugs that prolong QT interval cause the ventricles to be sensitive to VT: Quinidine, procainamide, amiodarone, tricyclic antidepressants
5. Electrolyte disturbances (hyper or hypokalemia, hyper or hypocalcemia; ↓ Mg)
6. Mechanical stimulation of the heart when placing a catheter
7. Reperfusion following thrombolytic or fibrinolytic therapy or angioplasty

D. **Clinical significance**

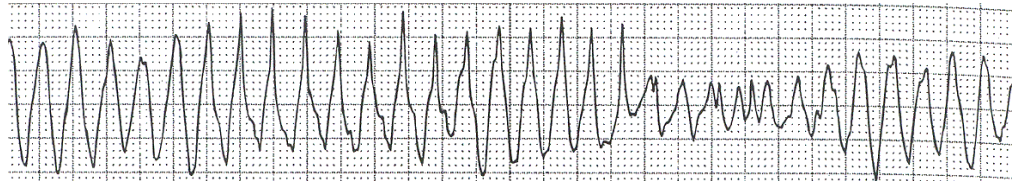
1. Depends on its duration: Nonsustained VT may be tolerated without compromise.
2. **Sustained ventricular tachycardia** is defined as having a duration of 30 seconds or more and may be **life-threatening**
 - a. Rapid ventricular rate + loss of atrial kick reduces CO = ↓ BP and ↓ perfusion to vital organs. Pulse may be present or absent and helps determine treatment.
 - b. Can degenerate into V-fib

E. **Treatment** - See VT SOP

1. Depends on whether or not they have a pulse, the degree of cardiorespiratory compromise, and whether the configuration is monomorphic or polymorphic.

2. All pulseless VT is treated like VF.
3. **Stable VT with none to moderate CR compromise** is treated with drugs based on whether the VT is monomorphic or polymorphic.

- a. **Monomorphic & polymorphic w/ normal QT interval: Amiodarone** 150 mg mixed w/ 7 mL NS IVP over 8-10 minutes OR 150mg in 100 mL NS IVPB over 10 min)
- b. **Polymorphic with prolonged QT segment: Torsades de pointes (twisting of the points):** The direction of the QRS complexes seems to rotate up and down in the same lead. The ventricular rate is very rapid (much faster than monomorphic VT), and the patient usually becomes hemodynamically compromised very quickly. **See TdP handout.**



(1) **Causes**

- (a) Delayed ventricular repolarization (prolonged QT interval) or the presence of prominent U waves
- (b) Drugs like procainamide, quinidine, amiodarone, sotalol that prolong the QT segment and are used to treat monomorphic VT
- (c) Hypocalcemia, hypokalemia, **hypomagnesemia**
- (d) Bradycardias
- (e) Psychotropic drugs: tricyclic antidepressants, phenothiazines (thorazine)
- (f) Liquid protein diets
- (g) Congenital disorders causing a prolonged QT interval

(2) **Treatment:** Must be recognized as treatment differs.

- (a) Stable: **Magnesium sulfate** 2 Gm mixed with 16 mL NS given IVP over 5 minutes OR 2 Gm in 50 mL NS IVPB on mcgtt tubing over 10 min).
- (b) If patient develops AMS or drops their BP: **Defibrillate per V-Fib SOP**

4. **Monomorphic VT with CR compromise** is Rx with **synchronized cardioversion** at manufacturer-specific J (see SOP). See lab manual for critical steps.

a. **Choosing the proper lead for cardioversion**

- (1) For cardioversion to be carried out safely, the electrical energy discharge is synchronized with the early part of the QRS complex (typically the R wave) in order to avoid energy delivery during the vulnerable period of repolarization (around the peak of the T wave) ("R-on-T" phenomenon), which can result in VF.
- (2) To avoid this error, EMS personnel must recognize the importance of examining additional leads before synchronized cardioversion when bizarre QRS-T complexes are present to avoid high-amplitude T waves being misinterpreted as R waves.
- (3) Defibrillators are frequently automatically programmed to lead II. However, induction of V-Fib with cardioversion has been reported when the T-waves are more prominent than the R waves in this lead.

- (4) In an emergency situation, the exact characteristic of the ECG tracing on the monitor screen of the defibrillator is commonly overlooked. If possible, a lead with a prominent R wave should always be selected.
- b. Once EMS gives the first antidysrhythmic drug, the hospital is committed to using the same drug. Sequential use of more than one antiarrhythmic agent causes a proarrhythmic effect. Preferred drug is amiodarone.
- c. Effective treatment with drugs and/or an **implantable cardioverter defibrillator** reduces the sudden death mortality over the next 12 months to 0-2%.

VI. Ventricular fibrillation (VF)

A. Description

1. Disorganized, chaotic foci take over control of the heart.
2. Ventricles fibrillate. They do not beat in a coordinated fashion, so there is no cardiac output. May occur spontaneously or follow dangerous PVCs or VT.
3. ECG shows an irregular, chaotic baseline with waves of varying sizes, shapes, and height with no discernable QRS complexes present. This represents chaotic, incomplete, and haphazard depolarization of small groups of muscle fibers in the ventricles.
4. If the waves are large, it is considered "coarse" VF.
5. If the waves are small, it is considered "fine" VF.
6. The distinction is important. Coarse VF is usually of more recent onset than fine and is more likely to respond to defibrillation attempts. Fine VF will need CPR, oxygenation and medications before it will respond to defibrillation. Fine VF may be difficult to distinguish from asystole.

B. Interpretation



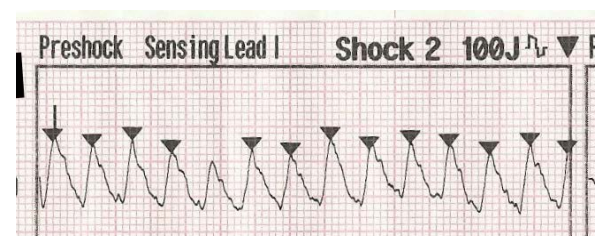
1. Rate: No P waves or QRS complexes are present. Cannot practically count.
2. Rhythm: Irregular and chaotic
3. P waves: None present
4. P-R interval: None
5. QRS complex: None present. Irregular, unorganized baseline

C. Etiology

1. Most common rhythm in sudden cardiac death
2. Significant heart disease: CAD, ACS, AMI
3. Same causes as VT
4. May be preceded by significant PVCs or VT but can occur spontaneously
5. Cardiomyopathy, mitral valve prolapse, cardiac trauma, hypoxia
6. Cocaine toxicity, electrolyte imbalances, acidosis, proarrhythmic drugs
7. During anesthesia, cardiac catheterization, pacemaker implantation, placement of a pulmonary artery catheter, or after accidental electrical shock

D. Clinical significance

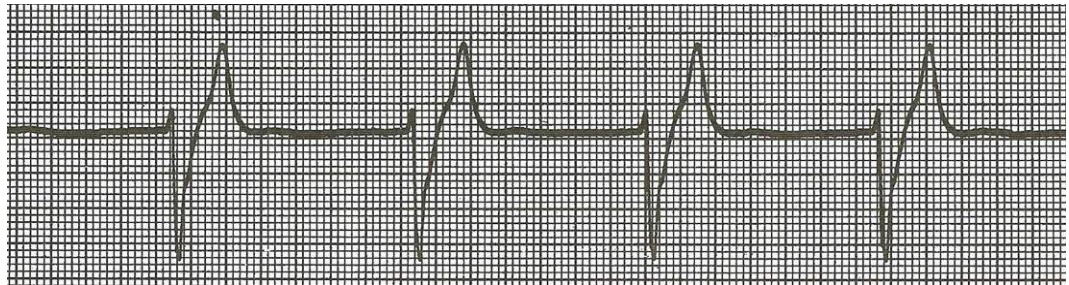
1. Patient will rapidly lose consciousness, may look like he or she is having a seizure, and then become pulseless and non-breathing.



2. DEATH if CPR is inadequate and rhythm is not converted almost immediately.
3. Patients lose 10% survivability for each 1 minute that defibrillation is delayed.
4. **Treatment** - See NWC EMSS SOPs – will cover in depth in Cardiac Arrest class

VII. **Idioventricular rhythm (IVR)** (Ventricular escape rhythm)

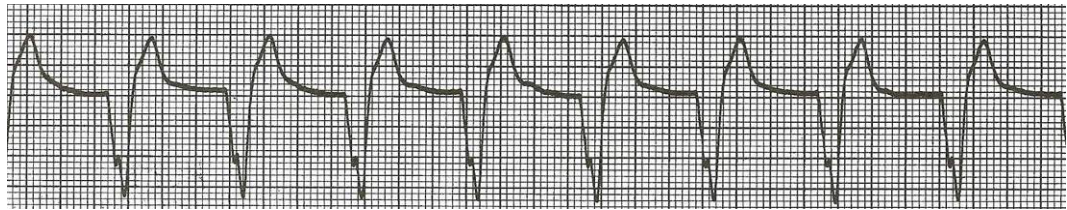
- A. **Description:** Escape rhythm originating in the HIS-Purkinje system (ventricles) that is the slowest & least reliable cardiac pacemaker. All complexes look like ventricular beats.
- B. **Etiology**
1. Rate of impulse formation in higher pacemakers becomes less than the escape pacemaker site in the ventricles.
 2. Impulses from above are blocked and fail to reach the ventricles, so a ventricular escape site kicks in.
 3. May be transient or continuous. Transient IVR lasts from a few seconds to a few minutes and is related to increased vagal tone on higher pacing centers. Is generally not significant.
 4. Continuous IVR is seen in advanced heart disease, HF, and is usually a terminal event.
- C. **Interpretation**



1. Rate: 20-40 beats per minute (intrinsic ventricular rate)
 2. Rhythm: Usually regular
 3. P waves: None; if present, they bear no relation to the QRS
 4. P-R interval: None
 5. QRS complex: 0.12 seconds or greater (wide and bizarre) with ventricular complex configuration because it originated in the ventricle and was not conducted normally
- D. **Clinical significance**
1. Cardiac output falls due to slow rate, poor stroke volume, and decreased cardiac output.
 2. Considered a life-threatening rhythm. Pulses are generally absent (PEA HR < 60)
 3. Search for and treat contributing causes – Hs & Ts
- E. **Treatment – Bradycardia with a pulse or Asystole/PEA SOP**
- DO NOT give any antidysrhythmic medication to this rhythm. You could cause asystole and death by inhibiting the only spontaneous rhythm the patient's heart is able to generate.

VIII. **Accelerated Idioventricular rhythm (AIVR)**

- A. Idioventricular rhythm with a rate 41 - 100
- B. It is inherently a ventricular rhythm exceeding the intrinsic pacing rate of the ventricles, but not fast enough to be called V-tach.



- C. Usually related to the automaticity of ventricular cells
- D. Common following AMI and is a frequent reperfusion rhythm
- E. Is often transient and may be tolerated well
- F. Brief episodes of AIVR may alternate with periods of NSR
- G. Treat per SOPs depending on pulse and degree of CR compromise
 - 1. No pulse: PEA
 - 2. Pulse present; HR above 60 with low BP: Cardiogenic shock; HR < 60 treat per Bradycardia with a pulse SOP

IX. Ventricular asystole

A. Description

- 1. Absence of all ventricular electrical activity
- 2. ECG shows only a straight line or just P waves without any QRS complexes
- 3. If P waves only are present, the rhythm probably started as a 2° or 3° AVB

B. Interpretation



- 1. Rate: No QRS complexes present
- 2. Rhythm: No QRS complexes present
- 3. P waves: Usually none present
- 4. P-R interval: None
- 5. QRS complex: Absent

C. Etiology

- 1. Often the ultimate outcome of VT, VF, IVR
- 2. Witnessed asystole may be the result of profound vagal tone to the heart

D. Clinical significance

- 1. No cardiac output--->DEATH
- 2. Prognosis is grim despite treatment

E. Treatment: See SOP – will cover in depth in cardiac arrest class

X. Pulseless Electrical Activity (PEA)

A. Description

- 1. Not a cardiac rhythm but a condition of pulselessness with a rhythm present.
- 2. Can be any rhythm on the monitor, but patient is **pulseless**.

B. Etiologies: Search for and treat contributing causes - Hs & Ts

Search for and treat possible contributing factors (Hs & Ts):

Hypovolemia (Most common treatable cause): IVF challenges

Hypoxia: ✓ airway; O₂ delivery; gas exchange

Hypothermia: Begin core rewarming w/ resuscitation

Hypo/hyperkalemia (? renal failure/dialysis) – bicarb & albuterol

Hydrogen ion (acidosis): (? DKA, renal failure/dialysis, ASA OD) – consider need for bicarb

Thrombosis (coronary): Perform 12 L ECG ASAP after ROSC

Thrombosis (pulmonary): IVF challenges

Tamponade: IVF challenges

Toxins: glucagon (beta blocker); bicarb (tricyclic antidepressants)

Tension pneumo: check lung sounds; pleural decompress prn

C. Interpretation

1. Identify ECG rhythm
2. If pulseless, declare the patient to be in a state of PEA

D. Clinical significance: No pulse--->No cardiac output--->DEATH

E. Treatment: See SOP

XI. Implantable Cardioverter-Defibrillator (ICD)

A. What is an Implantable Cardioverter-Defibrillator (ICD)?

1. A lead system and pulse generator are implanted. This system is capable of sensing either ventricular tachycardia or fibrillation and cardioverting or defibrillating the patient automatically.
2. The first human implantation was in 1980 and only recognized VF. Cardioversion was added in 1982. It received FDA premarket approval in 1985. There have now been thousands implanted.
3. Units can pace, cardiovert, or defibrillate.

B. How does the ICD work?

1. It works like a pacemaker, but instead of sensing a bradycardia and pacing, it senses a tachyarrhythmia and defibrillates.
2. The unit consists of a pulse generator that is implanted. It has a life span of about 3 years and is capable of delivering 100-200 shocks during that time.
3. Two patches or leads are sewn on to the RV and LV **or** there is a superior vena cava lead (also called the spring lead) positioned in the VC mid-atrial junction and a one patch lead sewn on the LV.
4. Rate sensing leads:
 - a. Bipolar transvenous lead is placed in the RV, or
 - b. Two epicardial leads are placed on the outside of the heart
 - c. These leads sense R waves and provide a synchronized shock.
5. The combination of these leads calculates the time span that waveforms are away from the baseline, they sense the morphology of the rhythm and calculate the probability density function.
6. The patches and leads are connected to the pulse generator.
7. Pacers sense and count "R" waves of the QRS complex. It is generally preprogrammed to a rate cutoff of between 120-200 beats per minute. If it senses a faster rate that began abruptly and the rhythm meets morphology criteria, the unit will begin charging (5-15 seconds) and deliver a shock at 25 J within 10-35 seconds after the problem is sensed. The device can recharge in 10-30 sec (x 4).
8. If the defibrillation was successful and the device does not sense the dysrhythmia for 35 seconds, it will re-set. If the dysrhythmia continues, the device will continue sensing the rhythm and fire again up to 3-4 more shocks at 30 J. The ACD will not fire further if the dysrhythmia continues. It requires 35 seconds of non-VT/VF (including asystole) to reset itself and full sequence.

C. Why would someone have this device?

1. An ICD is indicated for sustained VT or VF, survivors of sudden cardiac death (AVID trial [Antiarrhythmics Versus Implantable Defibrillators], secondary prevention), or inducible, sustained, monomorphic VT (MADIT I [Multicenter Automatic Defibrillator Implantation Trial], primary prevention).
2. Based on the results of the MADIT II study, ICDs will routinely be implanted in patients with LV dysfunction, ejection fraction (EF) of <35%, and a previous MI.
3. Emerging indications for implantation of ICDs include patients with syncope who have dilated cardiomyopathy and patients who have hypertrophic obstructive cardiomyopathy (HOCM) and are believed to be at high risk for sudden cardiac death (nonsustained VTs, syncope, and family members who have experienced sudden cardiac death).
4. Patient with wide QRS tachycardia of unknown origin, regardless of hemodynamic status should be evaluated for possible eligibility.
5. *Dual-chamber ICDs should be reserved for patients with an abnormal SA or AV node or conduction system or those with frequent supraventricular arrhythmias, such as atrial fibrillation, to avoid spurious shocks secondary to rapid ventricular responses. When dual-chamber pacing is required, and the LV is severely impaired, consideration should be given to a biventricular ICD.* (nice to know only)
6. CD battery life is currently 5 to 7 years and continues to improve. Follow-ups of PPMs and ICDs are usually every 6 to 12 months, with comprehensive testing of pacing and sensing thresholds.
<http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/cardiology/cardiac-arrhythmias/>)

D. Performance of ALS on patients with the device

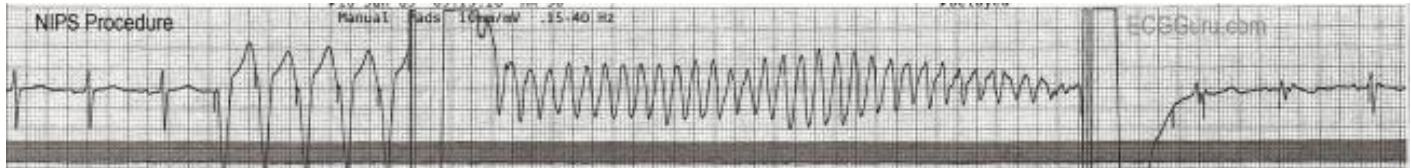
1. Treat the patient with an ACD like any other patient
2. Proceed with Initial Medical Care as usual.
3. DO NOT wait for the device to fire in the presence of VT or VF
4. Begin CPR as necessary
5. Defibrillate per usual procedure if necessary. It will not harm the device unless you discharge current directly over the pulse generator. If defibrillation is unsuccessful, attempt repositioning of the pads to an anterior/posterior position.
6. After initiating care, check the abdomen for pulse generator scars. Check for a Medic-Alert bracelet or ID card.
7. Ask family members about implant history.

E. Risks to the care-giver: None: Anyone touching a patient or performing CPR when the ACD fires may feel a slight buzzing sensation, usually < 2 J.

F. Risks to the patient

1. Most of the equipment or appliances they come into contact with will not affect the ICD system. The device is sensitive to strong electrical or magnetic fields that have the potential to deactivate some devices. Newer units can be programmed not to be affected by magnets and can store electrocardiograms. In some cases, the older ICDs may emit a sound if too close to a magnet. If this happens, move the patient away from the magnet immediately.
2. Potential sources of strong electrical and magnetic fields listed below should be kept at least 12 inches from an ICD pulse generator.
 - a. Stereo speakers from large stereo systems, transistor radios, etc.
 - b. Strong magnets
 - c. Magnetic wands used by airport security and in bingo and other games
 - d. Industrial equipment such as power generators and arc welders
 - e. Battery-powered cordless power tools, such as screwdrivers, drills, etc.

- f. Patients should not lean over any engine that is running as engine alternators frequently emit magnetic fields.
- g. Patients should also consult their physician about the radio frequency remote-controlled transmitters use for toy cars, airplanes, and boats. They can affect some pulse generators.
- h. Some may be sensitive to anti-theft systems also called electronic surveillance (EAS) systems found in stores and public libraries. A patient who lingers between the columns may deactivate their device.
- i. Household appliances should not interfere as long as they are grounded.



This is a rhythm strip from a NIPS procedure (non-invasive programmed stimulation), which is a programming test for an implantable cardioverter defibrillator (ICD). The test is done under light anesthesia, similar to that used for colonoscopy. In this example, the patient is in normal sinus rhythm at the beginning of the procedure. The pacemaker technician overdrives the patient's rate to observe the pacing function, then a stimulus is delivered to cause ventricular fibrillation (V Fib). Initially, the ventricular rhythm is somewhat organized and coarse (V flutter), but it will rapidly deteriorate if not corrected. Before it deteriorates, the ICD delivers a shock, and the patient's rhythm is restored. In this example, bi-ventricular pacing was conducted for a few minutes before the patient resumed NSR. The patient is then recovered from the anesthesia and discharged home.

This is a good example of the relative safety of shocking the well-perfused heart. Although it is possible to put the heart into an intractable V Fib with this test, the ICD usually is able to convert the potentially lethal rhythm easily. It is a good reminder that we need to perfuse the heart well before performing defibrillation on a person with unwitnessed cardiac arrest. (Fri, 09/27/2013 - 15:20 -- [Dawn](#))

XII. Bundle Branch Blocks and Intraventricular conduction delays – will cover more after 12L

A. Description

1. There is a delay or obstruction in transmission of electrical current through one of the bundle branches (either R or L) or through the Purkinje system. Impulses usually flow through both simultaneously causing synchronous depolarization of the ventricles. A block in one bundle branch causes that ventricle to get depolarized slightly later than the healthy side. This delay is reflected in a widened QRS complex.
2. If the underlying rhythm is sinus, all QRS complexes are preceded by a P wave, but the QRS complex is abnormally wide and may be bizarre. **Differentiating between a right and left sided block requires a 12 lead ECG.**
3. May be present in rhythms other than a sinus rhythm, so P waves may be absent depending on the native rhythm.

B. Etiology

1. RBBB may be found in healthy persons with apparently normal hearts and may be permanent or transient. Sometimes it appears only if the HR exceeds a critical rate.
2. LBBB almost always indicates a diseased heart. It may also be permanent or transient and be rate-related. New onset LBBB often suggests AMI.
3. Common causes
 - a. AMI
 - b. Coronary and hypertensive heart disease
 - c. Cardiac tumors

- d. Cardiomyopathy
- e. Pericarditis
- f. Myocarditis
- g. HF
- h. Syphilitic, rheumatic, and congenital heart disease
- i. Degenerative disease of the electrical conduction system

C. **Interpretation**



- 1. Rate: Depends on underlying rhythm
- 2. Rhythm: Depends on underlying rhythm; usually regular
- 3. P waves: Usually precede each QRS complex
- 4. P-R interval
 - a. May be normal or delayed
 - b. Constant
- 5. QRS complex: 0.12 seconds or greater
- 6. **Clinical significance**
 - a. May be complicated by an AV block especially in the presence of AMI
 - b. Acute hyperkalemia can cause an IVCD without a RBBB or LBBB. If the ECG pattern by 12 lead does not match either a RBBB or LBBB, consider the possibility of hyperkalemia immediately as the patient may be about to code. ACLS drugs and pacing do not work on a patient who is hyperkalemic. They must be treated with bicarbonate or albuterol ASAP.
 - c. If acute in onset after an MI, may need a pacemaker.
- 7. **Treatment:** IMC; treat patient, not rhythm

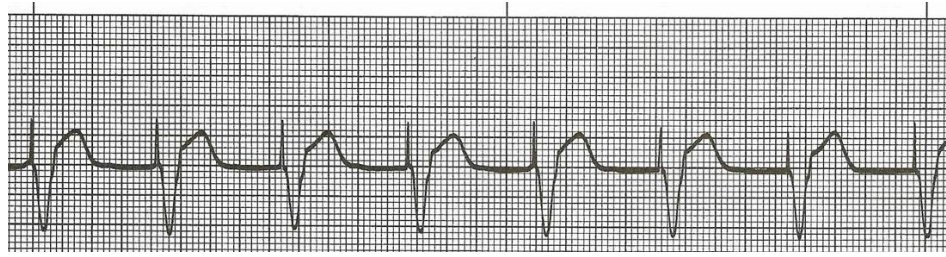
XIII. **Pacemaker rhythms**

A. **Description**

- 1. A pacemaker is a battery-powered device that delivers an electrical stimulus to the heart with the intent of producing a cardiac contraction.
- 2. Implantation of a permanent pacemaker requires specific levels of evidence and indications based on American College of Cardiology–American Heart Association (ACC/AHA guidelines.⁹ Class I and Class II indications are appropriate for the implantation of a permanent pacemaker (PPM).
- 3. Correlation of symptoms with underlying bradyarrhythmias or heart block is required. They are used when the person's own rate is too slow, when there is a potential for asystole to occur as in 2° and 3° AVB; or to overdrive an ectopic focus.
- 4. **Pacers function at a fixed rate or as a demand pacemaker**
 - a. Fixed rate delivers impulses at a set rate regardless of the pt's native rhythm. They may end up competing with the native rhythm and may be potentially dangerous as the pacing stimulus may fall on the vulnerable period and induce a ventricular dysrhythmia.
 - b. Demand pacers are made with a sensing mechanism that only discharges when the patient's rhythm is inadequate within a pre-determined period of time.
- 5. **Many types are available**

- a. **Single chamber:** Sense and pace **either** the atrium or the ventricle.
 - b. **Dual chamber:** Sense and pace **both** the atrium and ventricle.
 - (1) Advantage of dual chamber: Can restore the AV synchronous sequence of the heart, re-establishing the atrial kick which contributes 20-30% of the CO.
 - (2) More advanced models can vary rate based on myocardial needs (exercise) and some have internal defibrillators to stop rapid dysrhythmias.
 - c. Rate responsiveness simulates the chronotropic response of the sinus node and uses minute ventilation or, more commonly, motion to estimate the needed heart rate
 - d. **Common components**
 - (1) **Pulse generator:** Houses the battery; contains the various controls or settings for pacemaker function (mA, sensitivity or mV, HR setting, mode of pacing, specialized settings, etc.)
 - (2) **Pacing catheter:** Lead or electrode serves to connect the pulse generator and the endocardium. Conducts electrical current from pulse generator to myocardium and sends native rhythm information back to the pulse generator.
 - e. **May be temporary or permanent**
 - (1) Temporary include external transcutaneous (EMS application) and transvenous units. Electrode is inserted into the RV and connected by a bridging cable to an external pulse generator. External controls allow operator adjustments.
 - (2) Neither of these units are effective in the absence of contractile activity. For significant pathology, permanent pacing is required.
 - (3) Permanent pacers are implanted after a risk/benefit analysis. It is usually inserted using a transvenous approach through a major vein (subclavian) and advanced into the heart where it is placed in the endocardium of the RA or RV or both. The pulse generator is implanted into the subcutaneous tissues of the chest below the right or left clavicle. If endocardial pacing is ineffective, the pacer is implanted by a Tran thoracic surgical approach using general anesthesia. The catheter is sewn to the epicardial surface of the RV or LV and the generator is implanted into the abdominal wall.
6. **Basic functions of all pacemakers**
- a. **Sense:** Implies that the pulse generator is able to detect the patient's native (intrinsic) rhythm.
 - b. **Fire:** The pulse generator delivers an electrical stimulus to the heart measured in milliamps (mA). The discharge rate is preset in internal pacers and set by the operator with transcutaneous pacers.
 - c. **Capture:** The heart responds to the electrical stimulus.
 - (1) **Electrical capture:** Change in P wave or QRS following a pacing spike (stimulus artifact) confirms electrical depolarization. Ventricular pacing causes sequential depolarization instead of synchronous. This prolonged time results in a wide QRS.
 - (2) **Mechanical capture:** Pulse is present

B. **Interpretation**



1. A pacemaker rhythm occurs when the heart's rhythm is completely pacemaker induced. All QRS complexes are wide immediately following a spike (if one present).
2. Rate
 - a. Usually set about 70 (Big tip). Can't see a spike with a regular, wide, rhythm with no P waves? Assess rate. If right on 70, may be paced rhythm.
 - b. Dependent on patient's own underlying rhythm if a demand pacer
3. Rhythm: Regular or irregular dependent on patient's own underlying rhythm
4. P waves
 - a. None with the demand and external paced beats
 - b. Present with the A-V sequential - preceded by a "pacer spike"
5. P-R interval
 - a. None with the demand and external pacemaker
 - b. Set between .12 and .20 sec in the sequential pacemaker
6. QRS complex
 - a. Preceded by a "pacer spike"
 - b. Usually wider than 0.10 sec
 - c. Look for a tall, broad T wave that is the telltale sign of true electrical capture.
7. Perform, but do not rely solely on a manual pulse check. Consider using an instrument like an SpO2 monitor to verify mechanical capture.
8. **Fusion beat:** Occurs when the pacemaker fires an electrical impulse at the same time a patient's normal impulse has been activated in the ventricles. The two forces simultaneously depolarize the ventricles. Resulting complex is different in configuration and height from that caused by the native rhythm or paced beats.

C. **Recognizing patients with pacemakers**

1. Patient usually carries an ID card.
2. There is a standard system of pacer ID in a 3 letter code
 - a. 1st letter is the chamber paced (A,V,D)
 - (1) A: Atria
 - (2) V: Ventricles
 - (3) D: Dual chamber or single chamber
 - b. 2nd letter: Chamber sensed (A,V,D)
 - c. 3rd letter: Mode (T, I, O)
 - (1) T: Triggered
 - (2) I: Inhibited or inhibits pacing
 - (3) Dual: both I and T
 - (4) O: NA or none
3. The pacemaker spike will tell which chamber is paced depending on whether it is in front of the P wave or QRS complex
4. 4th letter: programmable functions: Programmable, multiprogrammable, communicating, or rate modulating

5. 5th letter: tachyarrhythmia function (antitachycardia pacing)
6. **Meaning of selected individual letters**
 - a. V = ventricle
 - b. A = atrium
 - c. D = double (with mode of response this means atrial triggered and ventricular inhibited)
 - d. O = none
 - e. T = triggered
 - f. I = inhibited
 - g. R = reverse
 - h. P = programmable (rate and/or output)
 - i. M = multi-programmable
7. The more common models include fixed rate (asynchronous — VOO, AOO, DOO), ventricular demand (VVI, VVT), atrial demand (AAI, AAT), atrial synchronized (VAT), and AV sequential (DVI). If the patient expects to do any activity, he needs a rate adjusting pacemaker. This type of pacemaker stores the previous R-R and generates the next beat based on it.

D. Malfunctions

1. **Failure to capture.** Spike may occur on time but is not followed by a QRS. Common with temporary pacers due to lead dislodgement. May also be caused by insufficient current. On transcutaneous pacers, mA can be increased up to 200.
2. **Undersensing or non-sensing:** Pulse generator does not sense native rhythm accurately resulting in competition. Pacing spike may occur earlier than it should. Ventricular capture may or may not occur.
3. **Loss of pacing artifact:** The pacing stimulus does not produce a spike on the ECG — this is the result of battery failure, pulse generator breakage, or wire disconnections or breakage).
4. EMS personnel are not expected to diagnose the specifics of undersensing, but they are expected to recognize that the patient has a paced rhythm.

Table 1: Vaughn-Williams Classification of Antiarrhythmic Medications

Class	Actions (Examples)
I	Sodium channel blockers
IA	Depress phase 0 of action potential; delay conduction, prolong repolarization—phase III or IV (quinidine, procainamide, disopyramide)
IB	Little effect on phase 0 of action potential in normal tissues; depress phase 0 in abnormal tissues; shorten repolarization or little effect (lidocaine , tocainide, mexiletine, diphenyl-hydantoin)
IC	Depress phase 0 of action potential; markedly slow conduction in normal tissues (flecainide, propafenone, moricizine)
II	β-Adrenergic blocking agents (acebutolol, atenolol, bisoprolol, carvedilol, metoprolol, nadolol, pindolol, propranolol)
III	Prolong action potential duration by increasing repolarization and refractoriness (amiodarone , sotalol, bretylium, dofetilide, azimilide, ibutilide)
IV	Calcium channel blockers (diltiazem, verapamil)
Others	Digoxin, adenosine

From Chaudhry G., Muqtada, M.D., Haffajee, C.I. (2000): Antiarrhythmic agents and proarrhythmia. Crit Care Med; 28:N158-N164.

Homework Questions

1. Ventricular rhythms originate below the branching portion of the _____
2. All organized beats originating in the ventricles are associated with narrow / wide QRS complexes.
3. Which is a classic characteristic of a PVC?
 - A. QRS complex less than 0.12 seconds
 - B. T wave of opposite polarity to the QRS
 - C. P-R interval longer than 0.20 seconds
 - D. short PR syndrome with delta wave into the QRS
4. What type of pause is associated with a PVC? Compensatory / noncompensatory
5. If a PVC appears every other beat, the pattern is known as _____
6. If a PVC appears every third beat, the pattern is known as _____
7. If PVCs appear in pairs, they are called _____
8. If three or more consecutive ventricular beats are present at a rate > 100, it is termed a run of _____
9. PVCs that all look alike are called _____
10. PVCs that differ in size, shape, and direction are called _____
11. A PVC sandwiched between two normally conducted sinus beats, without disturbing the regularity of the underlying rhythm is called an _____ PVC.
12. If a PVC occurs during the vulnerable period of ventricular repolarization, it is called an _____ phenomenon.
13. Stimulation of the ventricle during the vulnerable period can result in: _____
14. List three causes of PVCs:

15. Should PVCs be treated with antidysrhythmic agents? ☐ Yes ☐ No
16. A ventricular beat that occur late, rather than early, in the cycle, is called a ventricular _____ beat.
17. Ventricular tachycardia originates in an _____ focus in the ventricles, discharging impulses at a rate over _____ beats per minute.
18. The R-R in VT is generally regular / irregular.
19. Are P waves usually present preceding R waves in VT? ☐ Yes ☐ No
20. QRS complexes in VT are narrow / wide.

21. One form of polymorphic VT with a prolonged QT segment is called _____ (twisting of the points).
22. If a patient with the above rhythm is stable with a pulse, they should be treated with
- A. cardioversion at lower J settings.
 - B. immediate defibrillation at 360 J.
 - C. magnesium sulfate 2 Gm slow IVP.
 - D. amiodarone 150 mg IVP.
23. If they are unstable, they should be treated with _____
24. List the two major reasons why sustained VT can be life-threatening
- _____
- _____
25. What drug should be given to patients with monomorphic stable VT with a pulse?
- _____
26. What are the actions of this drug?
- _____
- _____
- _____
27. What is the dose and dilution of this drug for the patient in VT?
- _____
28. Over what period of time must be it be administered?
- _____
29. What is the side effect if it is given too quickly?
- _____
30. If a patient with monomorphic VT is unstable, but has a pulse, what is the treatment of choice?
- _____
31. When performing synchronized cardioversion, the defibrillator will sense and fire on the
- A. P wave.
 - B. R wave.
 - C. ST segment.
 - D. T wave.
32. This is done to avoid the
- A. QRS complex.
 - B. vulnerable period.
 - C. relative refractory period.
 - D. absolute refractory period.
33. What step must be taken to make sure the monitor is sensing the native R wave?
- _____

34. What is the desired physiologic effect of cardioversion?
- A. Deliver synchronized energy to the heart muscle
 - B. Cause the ventricles to contract
 - C. Jump start the heart by activating the SA node
 - D. Depolarize all the myocardial cells at once
35. How should a paramedic select the joule setting prior to cardioversion?
-
36. If a patient has VT, but does not have a pulse, they should be treated like _____
37. Where do Idioventricular rhythms originate?
-
38. IVR has a rate ranging between _____ beats/minute.
39. P waves are present / absent in IVR.
40. QRS complexes are narrow / wide in IVR.
41. An Idioventricular rhythm with a rate between 41-100 is called _____
42. What are the Hs and Ts to consider if the patient presents with a pulseless Idioventricular rhythm?
-
-
-
-
43. If a patient presents with a rhythm on the monitor, but is pulseless, their condition is referred to as
- A. AV dissociation.
 - B. electrical mechanical synchronization.
 - C. pulseless electrical activity (PEA).
 - D. agonal.
44. How is a patient with the above condition treated?
-
45. What is an ICD?
-
46. What makes it fire?
-
47. What should a paramedic do if confronted with a patient in cardiac arrest with an ICD firing?
-
48. The impulse in a bundle branch block does / does not originate in ventricular tissue.
49. A bundle branch block is characterized by a QRS complex of _____ seconds or longer.
50. How can an Idioventricular rhythm be distinguished from a rhythm with a bundle branch block?
-

51. What is the clinical significance of a new onset LBBB?

52. How can a paramedic detect an implantable pacemaker rhythm?

53. At what rate is it usually set? _____

54. Think about it...is it possible to have a strip with only pacing spikes? Why or why not?

NWC EMSS Skill Performance Record

DEFIBRILLATION

Name:	1 st attempt: <input type="checkbox"/> Pass <input type="checkbox"/> Repeat
Date:	2 nd attempt: <input type="checkbox"/> Pass <input type="checkbox"/> Repeat

Performance standard	Attempt 1 rating	Attempt 2 rating
0 Step omitted (or leave blank) 1 Not yet competent: Unsuccessful; required critical or excess prompting; marginal or inconsistent technique 2 Successful; competent with correct timing, sequence & technique, no prompting necessary		
Prepare/assess patient * Determine unresponsiveness; open airway (manually); assess for breathing/gasping; suction prn; simultaneously		
Assess pulse: If not definitively felt in <10 sec - Begin quality CPR with compressions per SOP p. 89		
<input type="checkbox"/> Remove all clothing from the patient's chest <input type="checkbox"/> Remove all nitro patches, briskly wipe skin with a dry towel or gauze <input type="checkbox"/> Disconnect Lifevest batteries; remove vest if present; DO NOT disconnect VAD batteries <input type="checkbox"/> If pulseless pt has an LVAD; ✓ SpO ₂ . If perfusing: NO CPR and DO NOT DEFIBRILLATE (even if VF). If questionable: Call VAD Coordinator for instructions.		
As quickly as possible: Prepare equipment <input type="checkbox"/> ✓ electrodes for expiration date <input type="checkbox"/> Connect defib cable to pace/defib electrodes.		
* Peel back the protective liner on the electrodes slowly, beginning with the cable connection end. Make sure gel is moist and in the middle of the electrode.		
* With compressions continuing: Place anterior electrode (black) without gaps or wrinkles on the patient's right upper torso, lateral to the sternum and below the clavicle.		
* Place the lateral (♥) red electrode under and lateral to patient's left nipple in the midaxillary line, with center of the electrode in the midaxillary line if possible.		
* Smooth electrode center and edges onto the patient's chest to eliminate air pockets between the gel surface and the skin. Firmly press all adhesive edges to the skin.		
* Select paddles mode		
* ✓ rhythm: Pause compressions just long enough to determine if rhythm is shockable (< 5 sec) (PVT/VF)		
Perform procedure * Immediately resume compressions. Charge monitor to device-specific joule setting; listen to ramping tone		
* Compressor verbally counts down to the pause in compressions to shock: 5-4-3-2-1; briefly pause CPR (< 5 sec); look around 360°; clear patient		
<input type="checkbox"/> *Depress current discharge button(after last compression -not a ventilation) <input type="checkbox"/> *Without checking ECG or pulse, change compressors and resume chest compressions for 2 mins. Limit time from last compression to shock delivery & resumption of compressions to ≤5 sec. <input type="checkbox"/> NO rhythm/pulse check until after 2 min of CPR unless pt wakes or begins to move extremities		
*If persistent/refractory VF: change pad location to A-P. If 2 monitors available: consider dual sequential defibrillation at device-specific joule settings		
Critical Criteria - Check if occurred during an attempt <input type="checkbox"/> Failure to determine the patient's need for rapid defibrillation <input type="checkbox"/> Performs any improper technique resulting in potential for patient harm <input type="checkbox"/> Exhibits unacceptable affect with patient or other personnel <input type="checkbox"/> Uses or orders a dangerous or inappropriate intervention		

Scoring: All steps must be independently performed in correct sequence with appropriate timing and all starred (*) items must be explained/ performed correctly in order for the person to demonstrate competency. Any errors or omissions of these items will require additional practice and a repeat assessment of skill proficiency.

Rating: (Select 1)

- ☐ **Proficient:** The paramedic can sequence, perform and complete the performance standards independently, with expertise and to high quality without critical error, assistance or instruction.
- ☐ **Competent:** Satisfactory performance without critical error; minimal coaching needed.
- ☐ **Practice evolving/not yet competent:** Did not perform in correct sequence, timing, and/or without prompts, reliance on procedure manual, and/or critical error; recommend additional practice

NWC EMSS Skill Performance Record

Mechanical Circulatory Support (MCS) using a Ventricular Assist Device

Name:	1 st attempt: <input type="checkbox"/> Pass <input type="checkbox"/> Repeat
Date:	2 nd attempt: <input type="checkbox"/> Pass <input type="checkbox"/> Repeat

Notes: Unit runs on electricity provided by a Power Base Unit (PBU) during stationary use or by rechargeable batteries worn during mobile use. Because blood bypasses aortic valve, there may be no pulse, especially with continuous flow pumps.

Performance standard	Attempt 1 rating	Attempt 2 rating
0 Step omitted (or leave blank) 1 Not yet competent: Unsuccessful; required critical or excess prompting; marginal or inconsistent technique 2 Successful; competent with correct timing, sequence & technique , no prompting necessary		
* State purpose of MCS: Assist a failing heart by taking blood out of LV, through the pump, & back into ascending aorta – reduces need for native heart to pump blood through aortic valve, reducing cardiac workload & O ₂ demand.		
Response to a pt with a VAD <input type="checkbox"/> Call VAD Coordinator immediately if known – phone number from pt or caregiver or one of the listed centers below if specific Coordinator unknown <input type="checkbox"/> Get history/instructions, VAD parameters from family/caregiver. Patients will be on anticoagulation medications – get list of all meds Patients will often have pacemakers and/or Internal Cardioverter Devices (ICDs). <input type="checkbox"/> Ask if pt is looking, feeling, or acting differently than their baseline		
Decision tree responsive patient <input type="checkbox"/> Assess ABCs: SpO ₂ waveforms may be flat; without amplitude despite accurate readings <input type="checkbox"/> If breathing labored; O ₂ per SOP <input type="checkbox"/> Assess circulation: May NOT have a pulse (NORMAL); check cap refill, color, temp, mental status <input type="checkbox"/> Listen for VAD sounds LUQ (when working device makes a quiet whiling sound) <input type="checkbox"/> Look and listen for alarms; pt & caregivers can help troubleshoot alarms		
Decision tree unresponsive patients <input type="checkbox"/> Airway, breathing assessment/Rx per SOP <input type="checkbox"/> Quick check for driveline or wire existing abdomen, batteries, cable, system controller <input type="checkbox"/> Caution removing clothes, especially using trauma scissors – DON'T CUT CABLES OR WIRES <input type="checkbox"/> Assess circulation: May NOT have a pulse (NORMAL); check cap refill, color, temp, mental status <input type="checkbox"/> Listen for VAD sounds LUQ (when working device makes a quiet whiling sound) <input type="checkbox"/> Look and listen for alarms; pt & caregivers can help troubleshoot alarms – see below <input type="checkbox"/> Consider other causes of AMS: stroke, cardiogenic shock, respiratory arrest, hyper or hypoglycemia – Rx per SOP		
State common causes of VAD alarms Pt not connected to power properly <input type="checkbox"/> Check all connections; fix loose connections <input type="checkbox"/> ✓ Driveline connection to System Controller <input type="checkbox"/> ✓ System Controller to battery clip <input type="checkbox"/> ✓ Batteries “engaged” in battery clips – NEVER DISCONNECT BOTH BATTERIES AT THE SAME TIME or pump will stop <input type="checkbox"/> ✓ System controller in cable connected to wall unit <input type="checkbox"/> Have pt/caregiver show how to silence alarms, use a hand pump if applicable		
Patient condition exists where low or no flow (cardiac output) is present <input type="checkbox"/> Do they appear to be in cardiogenic shock? Can be from electrical disruption to pump or pump malfunction (rare) <input type="checkbox"/> If yes, start SOPs; contact VAD Coordinator – provide assessments and VAD parameters if able <input type="checkbox"/> Transport to nearest VAD Center if possible; if no airway – transport to nearest hospital <input type="checkbox"/> Avoid external chest compressions if possible: Pose a risk due to location of outflow graft on aorta & inflow conduit in the LV apex. Dislodgement could lead to fatal hemorrhage. Contact VAD Coordinator for instructions re: CPR. Get instructions for hand pumping if applicable. CHEST COMPRESSIONS ARE ALLOWED if patient is unconscious and non-breathing.		
ECG findings: <input type="checkbox"/> VADs fix the plumbing - electrical conduction system should be intact; Do NOT expect asystole; pt may be conscious w/ V-fib <input type="checkbox"/> ECG waveforms may have a lot of artifact due to the device.		

Performance standard		Attempt 1 rating	Attempt 2 rating
0	Step omitted (or leave blank)		
1	Not yet competent: Unsuccessful; required critical or excess prompting; marginal or inconsistent technique		
2	Successful; competent with correct timing, sequence & technique , no prompting necessary		
<input type="checkbox"/>	Can have dysrhythmias but are better tolerated because pump continues to function despite irregular rhythm – Rx dysrhythmias with drugs per SOP		
Caveats on DEFIBRILLATION Majority of VAD pts can be shocked without disconnecting the percutaneous lead from the System Controller or stopping the pump prior to delivering the shock; but older units may need to be disconnected first and hand pumped before defib <input type="checkbox"/> Contact VAD Coordinator BEFORE defibrillating <input type="checkbox"/> Only shock if pt is unresponsive with poor perfusion/decreased circulation per cap refill (remember, no pulse is normal) and if you cannot contact VAD coordinator <input type="checkbox"/> Do not defibrillate over the pump; defibrillate at nipple line or above. Anterior-posterior pad placement preferred. <input type="checkbox"/> Warning: If VAD stops operating & blood is stagnant in pump & conduits for > a few min (depending on pt's anticoagulated state) there is risk of stroke and/or thromboembolism if device is restarted. Retrograde flow may occur during pump stoppage.			
Transport to nearest VAD center if possible			
Bring all VAD equipment if possible: batteries, battery clips, power base, plugs, battery charger (pt cannot be out of power)			
Allow family member/caregiver to ride in ambulance if possible			
Notes: NO MRIs - CT Scans are ok; avoid water submersion; avoid contact with strong magnets or magnetic fields			

Scoring: All steps must be independently performed in correct sequence with appropriate timing and all starred (*) items must be explained/ performed correctly in order for the person to demonstrate competency. Any errors or omissions of these items will require additional practice and a repeat assessment of skill proficiency.

Rating: (Select 1)

- ☐ **Proficient:** The paramedic can sequence, perform and complete the performance standards independently, with expertise and to high quality without critical error, assistance or instruction.
- ☐ **Competent:** Satisfactory performance without critical error; minimal coaching needed.
- ☐ **Practice evolving/not yet competent:** Did not perform in correct sequence, timing, and/or without prompts, reliance on procedure manual, and/or critical error; recommend additional practice

CJM 12/16

Preceptor (PRINT NAME – signature)

Heartmate XVE & Heartmate II

Illinois Mechanical Circulatory Support Implant Centers

Advocate Christ Medical Center - Oak Lawn	1-877-684-4327
Loyola University Medical Center - Maywood	1-708-216-8000
Northwestern Memorial Hospital - Chicago	1-312-695-9611
Rush University Medical Center - Chicago	1-312-656-6813
OSF Saint Francis Medical Center - Peoria	1-309-655-4101
University of Chicago Medical Center - Chicago	1-773-753-1880 id# 4823

