Northwest Community EMS System
Paramedic Training Program

VENTRICULAR DYSRHYTHMIAS
Bundle Branch Blocks; Pacemakers

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Reading assignment:
Aehlert Vol. 1 pp 788 – 789
SOPs: VT with pulse; Ventricular fibrillation/PVT; Asystole/PEA
Drugs: Amiodarone, magnesium, epinephrine 1:10,000; vasopressin
Skill: Defibrillation

KNOWLEDGE OBJECTIVES:

Upon completion of the class and study questions, each participant will independently do the following with a degree of accuracy that meets or exceeds the standards established for their scope of practice:

1. Identify the inherent 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 using the following discriminators:
   a) Rate (atrial and ventricular),
   b) Rhythm: Regular/irregular,
   c) Presence/absence/morphology of P waves,
   d) Presence/absence/morphology of QRS complexes,
   d) R-R Interval, P-P Interval,
   e) P-QRS relationships, and
   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.

NWC EMSS Paramedic Training Program

VENTRICULAR DYSRHYTHMIAS; Bundle Branch Blocks; Pacemakers

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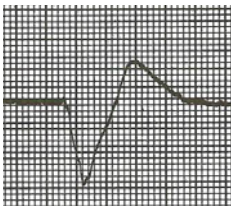
I. Dysrhythmias originating in the ventricles

A. Ventricular escape complexes and rhythms (IVR)
B. Accelerated idioventricular rhythm (AIVR)
C. Premature ventricular contractions (PVCs)
D. Ventricular tachycardia
E. Ventricular fibrillation
F. Asystole
G. Artificial pacemaker rhythm

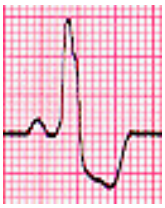
II. Characteristics of a ventricular complex (beat)



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 lead 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:

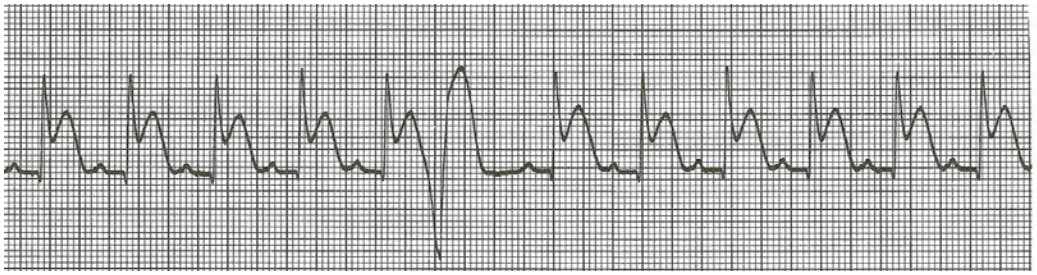
1. Pericardial effusion
2. Myocardial infarction
3. Cardiomyopathy
4. Hypothyroidism
5. Obesity
6. Sarcoidosis
7. Amyloidosis
8. Chronic obstructive pulmonary disease (COPD)
9. Anasarca

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 occurring before the next expected sinus or junctional beat.

B. **Characteristics of PVCs**

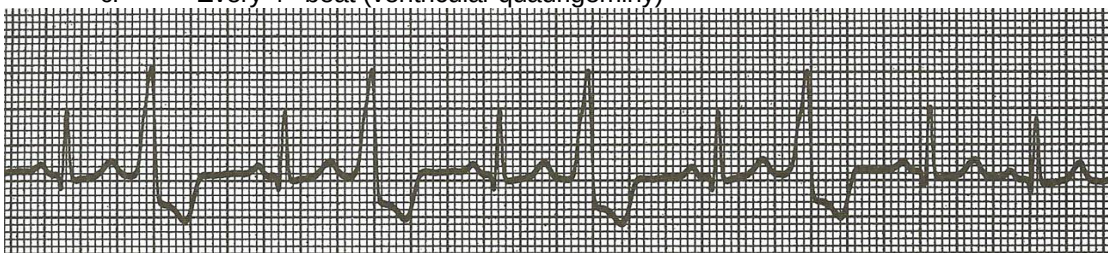
1. Rhythm
   a. Depends on underlying rhythm - usually regular
   b. Portion of the strip with the PVC is irregular



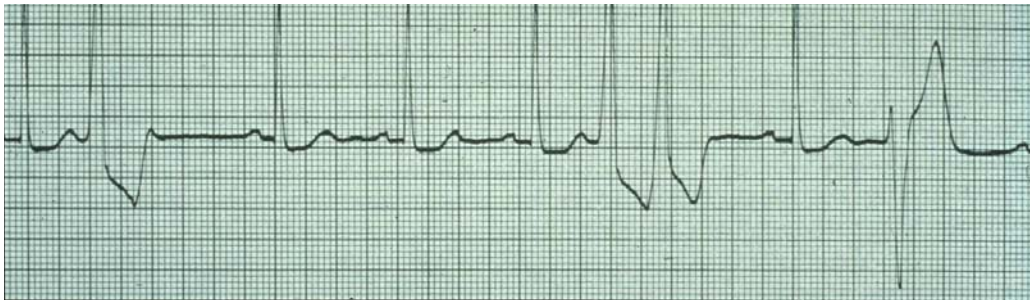
2. Rate: Depends on underlying rhythm (sinus, atrial or junctional)
3. P waves
   a. None associated with the PVC
   b. If P waves are present, they are usually associated with the underlying rhythm. P wave from the underlying rhythm may be seen preceding the QRS of a PVC or may be seen after the PVC 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
5. QRS complex
   a. Premature complex is wide (0.12 sec or greater) and abnormal in appearance
   b. It may be notched and looks different from the normal QRS complexes.
6. The ST segment and T wave are of opposite polarity (deflect in the opposite direction) of the main QRS.
7. Usually has a **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. The SA node is not depolarized by the ectopic beat, so the discharge timing of the SA node remains the same and the basic rhythm will resume on time after the PVC.

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**
   a. Every other beat (**ventricular bigeminy**)
   b. Every third beat (**ventricular trigeminy**)
   c. Every 4th beat (ventricular quadrigeminy)



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



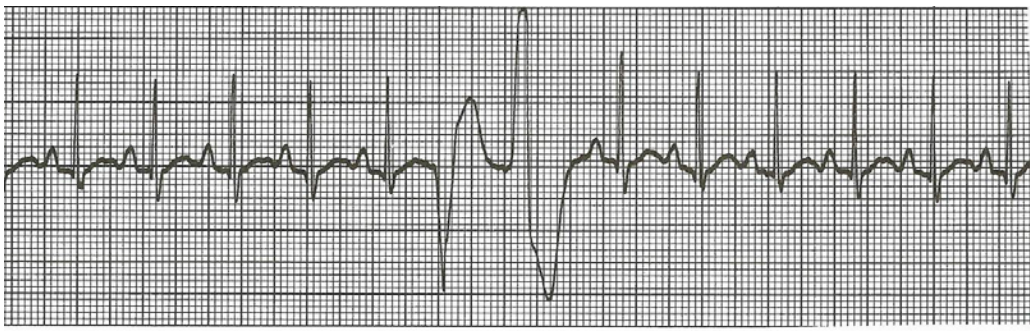
e. **Triplets:** 3 PVCs in a row (run of V-tach)

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.

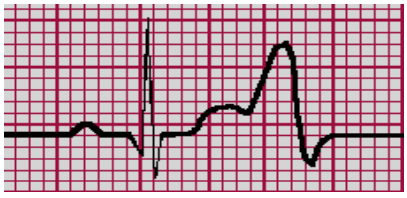
b. If 2 PVCs within one 6 second strip look different they are called **multiformed.** The text also refers to them as multifocal. This may indicate that two or more sites are originating the ectopic beats, but respected cardiologists disagree that a different look always means another ectopic focus.



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

a. PVC 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.



D. **Etiology**

1. Anxiety, excessive caffeine, tobacco or alcohol consumption
2. Hypoxia; acidosis
3. Drugs: Digitalis, epinephrine, isoproterenol, aminophylline
4. Electrolyte imbalances: ↓ K; ↓ Mg
5. HF, ACS, valvular disease, cardiomyopathy
6. After heart surgery or contact of the endocardium with catheters (pacing leads, PA catheters)
7. More frequent with age

E. **Clinical significance**: In ACS

1. Indicates increased ventricular irritability
2. Could lead to V or VF & death

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.

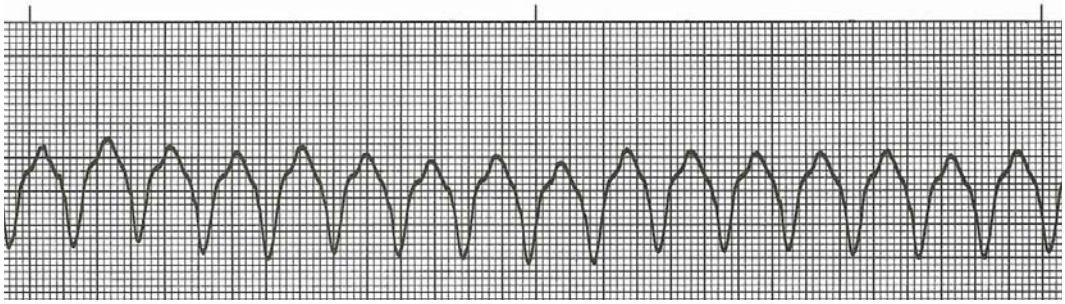
B. Etiology: Increased vagal tone on the SA node

V. Ventricular tachycardia

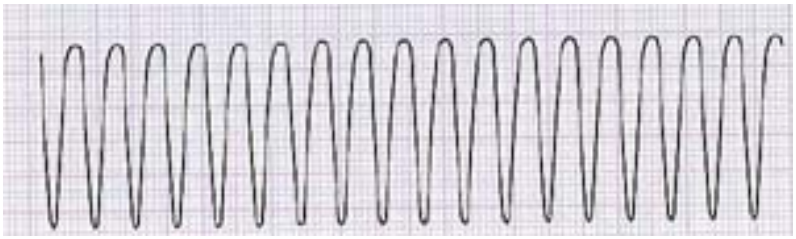
A. Description

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. 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. VFlutter can quickly deteriorate into VFib



2. Rhythm: Regular to slightly irregular
3. P waves: Often not visible, but may be present (dissociated from QRS)
4. P-R interval: Not measurable
5. QRS complex
    a. Wide (0.12 seconds or greater)
    b. T waves: Opposite polarity from QRS

C. **Etiology**

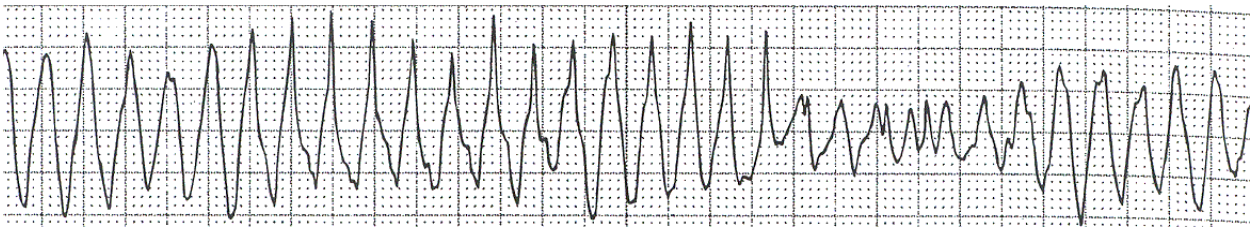
1. Hypoxia; **CAD, myocardial ischemia or infarction**
2. Underlying heart disease: Cardiomyopathy, mitral valve prolapse, CHF
3. Digitalis toxicity
4. Drugs that prolong QT interval cause the ventricles to be sensitive to VT: Quinidine, procainamide, amiodarone, tricyclic antidepressants
5. Electrolyte disturbances (↓ K, ↓ 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
2. Nonsustained VT may be tolerated without compromise.
3. Sustained VT is usually symptomatic and may be **life-threatening**
    a. Rapid ventricular rate + loss of atrial kick reduces CO = ↓ BP and ↓ perfusion to vital organs
    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.
    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.



(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 2 Gm mixed with 16 mL NS given IVP over 5 minutes.

(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 biphasic equivalent (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 large R wave of the QRS) in order to avoid energy delivery during the vulnerable period of repolarization, that is, around the peak of the T wave on the ECG ("R-on-T" phenomenon), which can result in ventricular fibrillation.

(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 (Xavier, 2004) 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.

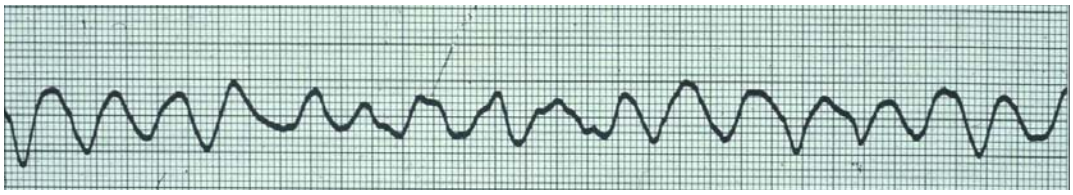
b. Once EMS gives the first antidysrhythmic drug, the hospital is committed to using the same drug. Sequential use of more than one antiarrhythmic agent causes a proarrhythmic effect. Preferred drug is amiodarone.

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

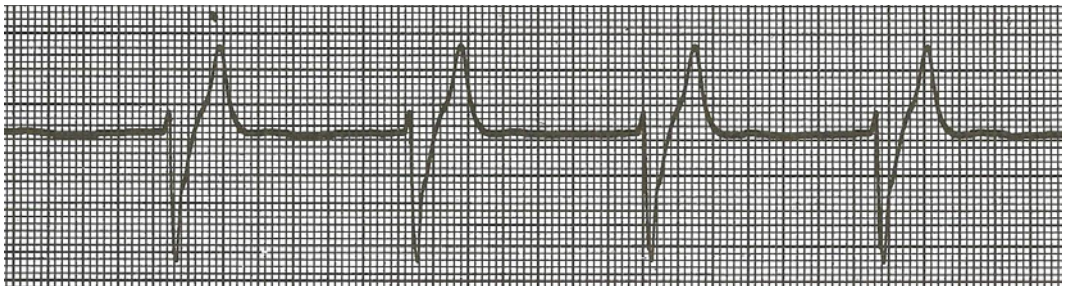
VII. **Idioventricular rhythm** (IVR) (Ventricular escape rhythm)

A. **Description**: Escape rhythm originating in the ventricles that are 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
2. Rhythm: Usually regular
3. P waves: None
4. P-R interval: None
5. QRS complex: 0.12 seconds or greater with ventricular complex configuration

D. **Clinical significance**

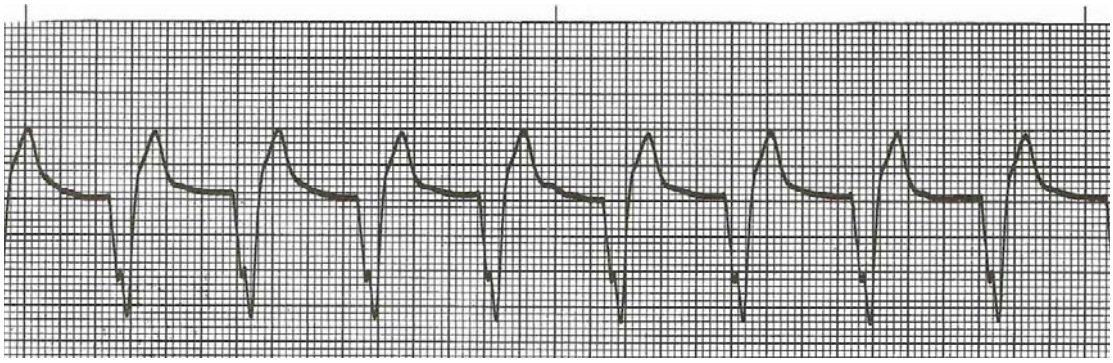
1. Cardiac output falls due to slow rate, poor stroke volume, and decreased cardiac output.
2. Pulses are generally absent (PEA HR < 60)
3. Search for and treat contributing causes – Hs & Ts

E. **Treatment – Bradycardia with a pulse or Asystole/PEA SOP**

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

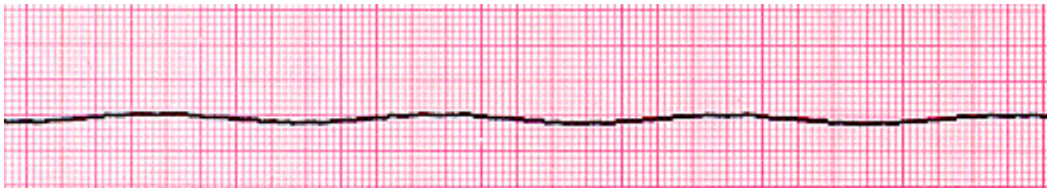
1. No pulse: PEA
2. Pulse present; HR above 60 with low BP: Cardiogenic shock; HR < 60 treat per Bradycardia with a pulse SOP

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

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**

• Hypovolemia	• Toxins (drug OD/poisoning)
• Hydrogen ion (acidosis)	• Tamponade, cardiac
• Hypoxia	• Tension pneumothorax
• Hyper/hypokalemia	• Thrombosis, coronary or pulmonary
• Hypothermia	• Trauma
• Hypoglycemia	

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 (ACD)?**

1. 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.
2. 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. If 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 in 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 10-30 seconds.
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. Many have had a previous episode of VF and survived sudden cardiac death.
2. Now those who have had an AMI and have non-sustained VT can be tested for eligibility.
3. Patient with wide QRS tachycardia of unknown origin, regardless of hemodynamic status should be evaluated for possible eligibility.

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.

XII. **Bundle Branch Blocks and Intraventricular conduction delays**

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**

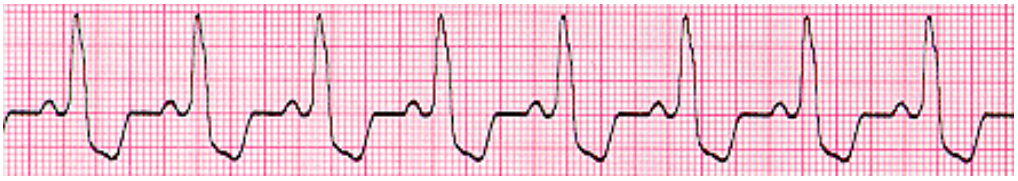
1. RBBB may be found in healthy persons with apparently normal hearts and may be permanent or transient. Sometimes it appears only if the HR exceeds a critical rate.

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. 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.
3. **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.
4. **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. **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.

d. **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.

5. **Basic functions of all pacemakers**

a. **Sense:** Implies that the pulse generator is able to detect the patient's native (intrinsic) rhythm.

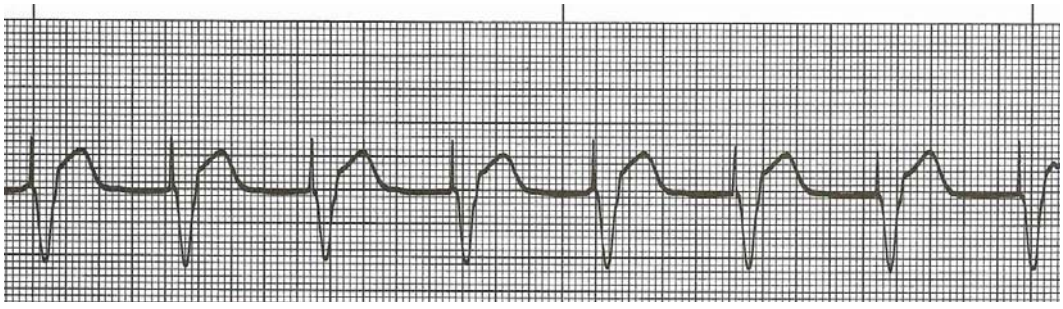
b. **Fire:** The pulse generator delivers an electrical stimulus to the heart measured in milliamps (mA). The discharge rate is preset in internal pacers and set by the operator with transcutaneous pacers.

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.

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 .10 sec
7. **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. **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:** Pulse generator does not sense native rhythm accurately. Pacing spike may occur earlier than it should. Ventricular capture may or may not occur.
3. EMS personnel are not expected to diagnose the specifics of undersensing, but they are expected to recognize that the patient has a paced rhythm.

D. **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: Double
    b. 2nd letter: Chamber sensed (A,V,D)
    c. 3rd letter: Mode (T, I, 0)
        (1) T: Triggered
        (2) I: Inhibited
        (3) 0: N/A
    d. Most common
        (1) VVI: Ventricular inhibited
        (2) DDI: Atrioventricular sequential
    e. Rarely used: VVO: Ventricular fixed rate
3. The pacemaker spike will tell which chamber is paced depending on whether it is in front of the P wave or QRS complex

References

Xavier LC, Memon, A. (2004) Synchronized cardioversion of unstable supraventricular tachycardia resulting in ventricular fibrillation. *Ann Emerg Med,* 44, 178-80.

Study 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 PVCs 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 does 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 of an 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?