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ACLS Study Guide

New Updated 2015 AHA Guidelines

Training You Can Trust

ACLS - BLS - CPR - PALS - First Aid

And More!

Course Overview

This study guide is an outline of content that will be taught in the American Heart Association ACLS (Advanced Cardiac Life Support) Course. It is intended to summarize important content, but since all ACLS content cannot possibly be absorbed in a class given every two years, it is expected that the student study ahead of time.

This guide does not replace the Advanced Cardiac Life Support Provider Manual and is only intended as a guide to help you study for your class, **but even more so**, as a refresher in-between certification classes.

We hope you find value in this study guide.

Good luck!

Heart Savers Training, LLC

* Required ACLS Pre-Test *

- The American Heart Association requires each student to complete an ACLS Pre-Test *prior to* coming to both the 2-Day Initial Class as well as The 1-Day Recertification Class
- The Pre-test can be found at: <http://heart.org/eccstudent>
- The necessary code needed to take the Pre-Test can be found in the *AHA ACLS Provider Manual* in the front of the book
- Per AHA guidelines, you must score a 70% or better on the Pre-Test which will be taken up by the Instructor at the beginning of the class

The BLS Survey

C - A - B

Assess		Assessment Techniques & Actions
1	Check Responsiveness	<ul style="list-style-type: none"> • Tap and shout, "Are you alright?"
2	Activate the Emergency Response System Get the AED	<ul style="list-style-type: none"> • Activate or send someone to activate the emergency response system and get an AED, if one is available.
3	CPR	<ul style="list-style-type: none"> • Check for a carotid pulse and breathing simultaneously for 5-10 seconds (gaspings / agonal respirations is not normal) • If no pulse <u>or you are not sure if there is a pulse</u>, start CPR (30:2) beginning with chest compressions • Compress the center of the chest (lower half of the sternum) hard and fast with compressions at 100-120 per minute at a depth of <i>at least</i> 2 inches [2 in. (5 cm) to 2.4 in. (6 cm)] • Allow complete chest recoil after each compression • Minimize interruptions in chest compressions (10 seconds or less) • Switch providers every 2 minutes to avoid fatigue • Avoid excessive ventilation. Each breath lasts one second or until you see adequate chest rise • If there is a pulse, start rescue breathing at 1 breath every 5-6 seconds. Check for a pulse every 2 minutes
4	Defibrillation	<ul style="list-style-type: none"> • If no pulse, check for a shockable rhythm with an AED/defibrillator as soon as it arrives • Provide shocks and immediately begin CPR starting with chest compressions

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Effective Resuscitation Team Dynamics

Role of the Team Leader – is multifaceted. The team leader

- Organizes the group
- Clearly delegates tasks
- Monitors individual performance of team members—If the Team Leader sees someone about to make a mistake he/she addresses it immediately
- Back up team members
- Models excellent team behavior
- Trains and coaches
- Facilitates understanding
- Focuses on comprehensive patient care

Role of Team Member – must be proficient in performing the skills authorized by their scope of practice.

- Clear about role assignment
- Prepared to fulfill their role responsibilities
- If asked to perform a task that is outside of their scope of practice, asks for a new task
- Well practiced in resuscitation skills
- A thorough working knowledge about the algorithms
- Committed to success

Closed Loop Communications – When communicating with resuscitation team members, everyone should use closed loop communication by taking these steps:

- The team leader gives a message, order, or assignment to a team member
- By receiving a clear response and eye contact, the team leader confirms that the team member heard and understood the message, confirming the order, and advising when completed

Medical Emergency Teams (METs) and Rapid Response Teams (RRTs)

Many hospitals have implemented the use of METs or RRTs. The purpose of these teams is to improve patient outcomes by identifying and treating early clinical deterioration. In-hospital cardiac arrest is commonly preceded by physiologic changes. In one study nearly 80% of hospitalized patients with cardiorespiratory arrest had abnormal vital signs documented for up to 8 hours before the actual arrest. Many of these changes can be recognized by monitoring routine vital signs. Intervention before clinical deterioration or cardiac arrest may be possible.

The ACLS Survey

Airway Management in Respiratory Arrest – Advanced airway equipment includes the ET tube, laryngeal mask airway or Air-Q, and the esophageal tracheal tube. If it is within your scope of practice, you may use advanced airway equipment in the course as treatment when appropriate and available.

If bag-mask ventilation is adequate, providers may defer insertion of an advanced airway. Establishing an IV or IO takes priority over ET Tube placement.

Quantitative Waveform Capnography

- Used to continuously monitor ETT placement
- if PETCO₂ is <10mmHg, attempt to improve CPR quality
- In ROSC, the normal PETCO₂ is 35-45

Basic Airway Adjuncts: Oropharyngeal Airway

The OPA is used in patients who are at risk for developing airway obstruction from the tongue or from relaxed upper airway muscle. This J-shaped device lifts the tongue away from the posterior wall of the pharynx.

The OPA is used in unconscious patients without a gag reflex if procedures to open the airway fail to provide and maintain a clear, unobstructed airway. An OPA **should not** be used in a conscious or semiconscious patient because it may stimulate gagging and vomiting. The key assessment is to check whether the patient has an intact cough and gag reflex. If so, do not use an OPA.

Selecting The Proper Size OPA

Place the OPA against the side of the face. When the tip of the OPA is at the corner of the mouth, the flange is at the angle of the mandible. A properly sized and inserted OPA results in proper alignment with the glottic opening.

Basic Airway Adjuncts: Nasopharyngeal Airway

The NPA is used as an alternative to an OPA in patients who need a basic airway management adjunct. The NPA is a soft rubber or plastic uncuffed tube that provides a conduit for airflow between the nares and the pharynx.

Unlike oral airway, NPAs may be used in conscious or semiconscious patients (patients with an intact cough and/or gag reflex). The NPA is indicated when insertion of an OPA is technically difficult or dangerous. Also, do not use a NPA in a patient with a possible head injury [basal skull fracture.]

Suctioning

Suctioning is an essential component of maintaining a patient's airway. Providers should suction the airway immediately if there are copious secretions, blood, or vomit.

Monitor patient's heart rate, pulse oxygen saturation, and clinical appearance during suctioning. If bradycardia develops, oxygen saturation drops, or clinical appearance deteriorates, interrupt suctioning at once. Administer high flow oxygen until the heart rate returns to normal and the clinical condition improves. Assist ventilation as needed.

Suctioning attempts should not exceed 10 seconds. To avoid hypoxemia, precede and follow suctioning attempts with a short period of administration of 100% oxygen.

Ventilation Rates

Airway Device	Ventilations During Cardiac Arrest	Ventilations During Respiratory Arrest
Bag-Mask	(30:2) 2 ventilations after every 30 compressions	1 ventilation every 5 to 6 seconds
Any Advanced Airway	1 ventilation every 6 seconds (10 breaths per minute)	(10-12 breaths per minute)

Cricoid Pressure:

Cricoid Pressure is *not* recommended during ventilations [when ventilating the patient]. To prevent gastric distention, ventilate only until you see chest rise (approx. 1 second for each breath)

However, cricoid pressure *can* still be used to help visualize the vocal cords but *only* when initially intubating the patient.

Purpose of Defibrillation

Defibrillation does not restart the heart. Defibrillation stuns the heart and briefly terminates all electrical activity, including VF and VT. If the heart is still viable, its normal pacemaker may eventually resume electrical activity (return of spontaneous rhythm) that ultimately results in a perfusing rhythm (ROSC).

Principle of Early Defibrillation

The earlier defibrillation occurs, the higher the survival rate. When VF (Ventricular fibrillation) is present, high quality CPR can provide a small amount of blood flow to the heart and brain but cannot directly restore an organized rhythm. The likelihood of restoring a perfusing rhythm is optimized with immediate CPR and defibrillation within a few minutes of the initial arrest. Restoration of a perfusing rhythm requires immediate CPR and defibrillation within a few minutes of the initial arrest.

Foundational Fact: Paddles vs. Pads

No existing data suggest that one is better than the other. Self adhesive pads, however, reduce the risk of arcing, allow monitoring of the patient's underlying rhythm, and permit a more rapid delivery of a shock.

Delivering Shock

The appropriate energy dose is determined by the identity of the defibrillator – monophasic or biphasic. If you are using a monophasic defibrillator, give a single 360-J shock. Use the same energy dose of subsequent shocks. Biphasic defibrillators use a variety of waveforms, each of which is effective for terminating VF over a specific dose range.

When using biphasic defibrillators, providers should use the manufacturer's recommended energy dose (e.g., initial dose of 120 to 200 J). Hospitals usually use the maximum dose of 200 J. The AHA Guidelines still endorse a 'One Shock Protocol.' If defibrillator will go higher, subsequent shocks, every two minutes should be equal or higher joules.

To minimize interruptions in chest compressions during CPR, continue CPR while the defibrillator is charging.

IMMEDIATELY after the shock, resume CPR, beginning with chest compressions.

As always, follow the Protocols set forth by your Facility

Give 2 minutes (about 5 cycles) of CPR. A cycle consists of 30 compressions followed by 2 ventilations in the patient without an advanced airway.

If the AED does not promptly analyze the rhythm, do the following:

- Resume high-quality chest compressions and ventilations
- Check all connections between the AED and the patient to make sure that they are intact.

NEVER delay chest compressions (more than 10 sec.) to troubleshoot the AED

Synchronized vs. Unsynchronized Shocks

Synchronized (Cardioversion)

- Cardioversion uses a sensor to deliver a shock that is synchronized with a peak of the QRS complex
- Synchronized cardioversion uses a lower energy level than attempted defibrillation.

When to use synchronized shock (Cardioversion)

- o Unstable SVT (50 to 100 Joules -biphasic)
- o Unstable Atrial Flutter (50 to 100 Joules -biphasic)
- o Unstable Atrial Fibrillation (120 to 200 Joules -biphasic)
- o Unstable regular monomorphic tachycardia with pulse
(100 Joules -biphasic)

Foundational Facts:

The first thing you do with an AED is **Turn it on.**

Be sure oxygen is not flowing across the patients' chest when delivering a shock

The pause in chest compressions to check the rhythm **should not exceed 10 seconds**

Unsynchronized (Defibrillation)

- Means that the electrical shock will be delivered as soon as the operator pushes the SHOCK button to discharge the device.
- May fall randomly anywhere within the cardiac cycle.

When to use unsynchronized shock (Defibrillation)

For a patient whose ECG shows:

- **Ventricular fibrillation (V-fib)** or **Pulseless ventricular tachycardia (VT)**
(120 to 200 Joules- biphasic—Use maximum dose available)

Routes of Access for Drugs

Historically in ACLS, providers have administered drugs via either the IV or endotracheal route. Endotracheal absorption of drugs is poor and optimal drug dosing is not known.

For this reason, peripheral IV access is preferred and if not available then use the Intraosseous (IO) route.

Priorities for vascular access are:

- **IV Route** – A peripheral IV is preferred for drug and fluid administration unless central line access is already available. Central line access is **not** necessary during most resuscitation attempts.
- **IO Route** – Drugs and fluids during resuscitation can be delivered safely and effectively via the IO route if IV access is not available.

ACLS Algorithm Review

Acute Coronary Syndrome

The ACLS provider Course presents only basic knowledge focusing on early treatment and the priority of rapid reperfusion, relief of ischemic pain, and treatment of early life-threatening complications. Reperfusion may involve the use of fibrinolytic therapy or coronary angiography with PCI (i.e., balloon angioplasty / stenting).

If the patient has signs/symptoms suggestive of Ischemia or Infarction

Assessment, care and hospital preparation:

- Monitor, support ABCs. Be prepared to provide CPR and defibrillation
- Administer aspirin and consider oxygen, nitroglycerin, and morphine if needed and if **not** contraindicated.
- Obtain a **12 & 15 Lead ECG**; for any and all chest pain - as well as any discomfort from neck to waist, both front and back
 - Notify hospital and transport to a hospital with PCI capability and has the resources to provide care for a STEMI

NOTE:

Relief of pain with nitroglycerin is neither specific nor a useful diagnostic tool to determine the etiology of symptoms in ED patients with chest pain or discomfort.

GI etiologies as well as other causes of chest discomfort can “respond” to nitroglycerin administration. Therefore, the response to nitrate therapy is not diagnostic of ACS.

When Appropriate

- If O2 SAT < 94%, start oxygen at 2L/min, and titrate as needed (Too much oxygen can cause oxygen toxicity)
- Aspirin (chewable) 160 To 325 mg
- Nitroglycerin tablet or spray, sublingual
- Morphine IV, if discomfort and not relieved by Nitro

The 12-lead ECG is at the center of the decision pathway in the management of ischemic chest discomfort and is essential in identifying a STEMI

General Group	Description
STEMI	ST elevation in one or more contiguous leads or new LBBB. Threshold values for ST-segment elevation consistent with STEMI are J-point elevation greater than 2mm (0.2mV) in leads V2 and V3 and 1mm or more in all other leads or by new or presumed new LBBB. 2.5mm in men >40 years; 1.5 mm in all women
High-risk Unstable Angina/NSTEMI	ST depression or dynamic T-wave inversion is characterized by ischemic ST-segment depression (0.05mV) or dynamic T-wave inversion with pain or discomfort.
Intermediate/low-risk Unstable Angina	Normal or non-diagnostic ECG – Serial cardiac studies and functional testing are appropriate. Note that additional information (troponin) may place the patient into a higher risk classification after initial classification.

Remember: It is possible to have a 'normal' ECG but still have abnormal cardiac enzymes and Troponin levels [a Non-STEMI]

Use of Fibrinolytic Therapy

A fibrinolytic agent or “clot buster” is administered to patients with J-point ST-segment elevation greater than 2mm (0.2mV) in leads V₂ and V₃ and 1mm or more in all other leads or by new or presumed new LBBB without contraindication.

Use of PCI

The most commonly used form of PCI is coronary intervention is with stent placement. Primary PCI is used as an alternative to fibrinolytic therapy. Rescue PCI is used early after fibrinolytic therapy in patients who may have persistent occlusion of the infarct artery (failure to reperfusion with a fibrinolytic).

Immediate Coronary Reperfusion with PCI

Following ROSC, rescuers should transport the patient to a facility capable of reliably providing coronary reperfusion and other goal directed post arrest care therapies. The decision to perform PCI can be made irrespective of the presence of coma or the decision to induce hypothermia, because concurrent PCI and hypothermia are reported to be feasible and safe and have good outcomes.

Cardiac Rhythms - 12 & 15 Leads

“It has been stated: “every second that passes that a cardiac cath lab is not activated, 500 heart cells die.” [Bob Page, Multi-Lead Medics, pg. 3].

Therefore, as a healthcare provider, in order to give excellent patient care, it is essential that we understand and are able to recognize cardiac rhythms. To know the differences between non-threatening and lethal arrhythmias, and most of all, how to treat them.

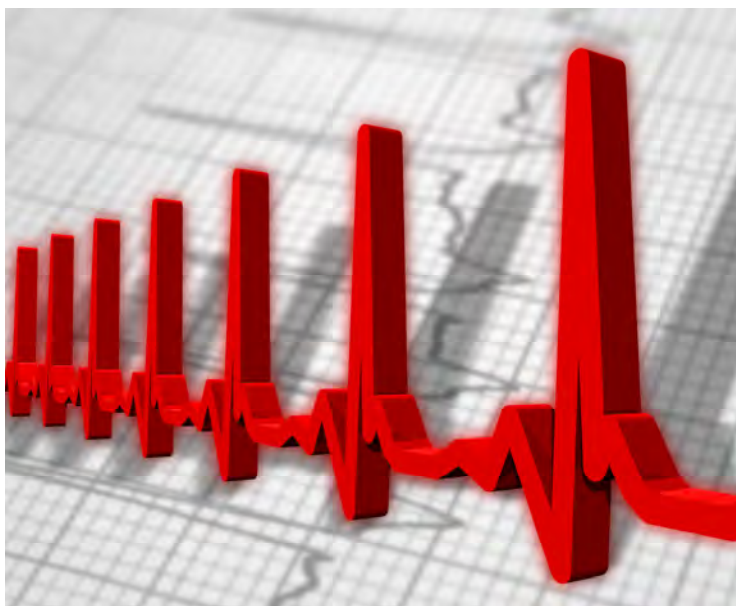


(Picture purchased from Shutterstock)

Everyone with chest pain or any cardiac dysrhythmia needs to have a **12-Lead ECG** done, as well as a **15-Lead ECG**. The **12-lead ECG** uses 10 electrodes: one on each limb and six on the chest. Limb leads should be placed on the arms and legs, and *never* on the chest. The Precordial leads are placed in specified positions on the chest. Three of those same leads are moved to check the posterior portion of the heart in performing the **15-Lead ECG**. **Proper lead placement is absolutely essential and crucial for both! Failure to place the leads in the correct locations alter the angle and amplitude and therefore give an inaccurate ECG reading.** You will learn how to correctly perform both the **12-Lead ECG** and **15-Lead ECG** in class.

This section is in no way a thorough treatment of cardiac rhythms, but simply an overview and review. We encourage you to constantly study and increase your knowledge and understanding of cardiac rhythms. Some books and websites are better than others. You may visit us at www.4cpr.org for additional study materials and/or email us at heartsaverstraining@gmail.com for additional resources that we recommend. You may also choose to further your practical understanding by registering for one of our **EKG Rhythms / 12 & 15-Lead Classes**.

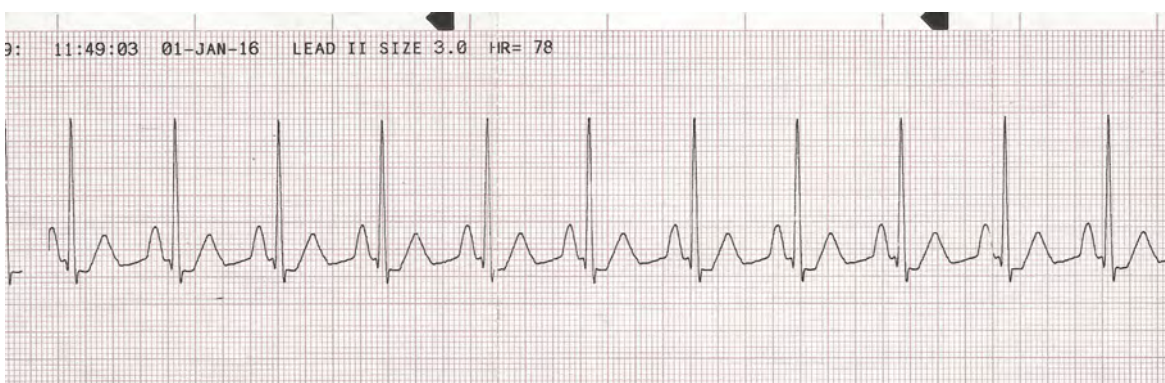
Let's begin...



Sinoatrial [SA] Node Rhythms

- In the SA Node Rhythms, they all have the following characteristics in common:, with only *one* difference:
 - **Rhythm:** Regular
 - **P Waves:** Normal [uniform and upright - they all look similar]
 - **PR Interval:** Within Normal limits [0.12 to .0.20 sec]
 - **QRS:** Normal, narrow [0.06 to 0.12 sec]
 - **Rate:** This is what varies

(Normal) Sinus Rhythm



Normal Sinus Rhythm occurs when the SA node is firing at a rate that is considered 'normal' for a person's age. The rate is generally **60 to 100 bpm**. The key to normal sinus rhythm is that all components of a normal ECG are present: P waves, [normal PR intervals], narrow QRS complex, and T waves. There is usually no treatment required for this rhythm. The term is sometimes considered a misnomer and its use is sometimes discouraged. The 'corrected' term is **Sinus Rhythm**.

All the rhythms found in this ACLS Study Guide have been recorded in Lead II

Sinoatrial [SA] Node Rhythms

Sinus Bradycardia



Bradycardia occurs when the heart is beating too slow (< 60 beats per minute). In Sinus Bradycardia, the SA node fires at a rate slower than normal for a person’s age. Athletes may have heart rates less than 60 due to their physical conditioning. Sinus Bradycardia can also occur while sleeping, from certain medications such as Beta Blockers, vagal maneuvers, ICP, suctioning, and ischemic heart disease. It is possible for a patient to have a heart rate of 50 and be asymptomatic, obviously, they would not need treatment. However, if a patient with a heart rate of less than 60 has signs of poor perfusion (symptomatic / unstable) - systolic B/P <90), begin treatment promptly.

Treatment:

Asymptomatic / Stable:

Seek expert medical consultation and **obtain a 12 lead ECG.**

Symptomatic / Unstable:

Give atropine as <u>first-line</u> treatment	Atropine 0.5 mg IV - may repeat to a total dose of 3 mg	
<u>If atropine is ineffective</u>		
Transcutaneous pacing	Or	Epinephrine 2 to 10 mcg/min
		Dopamine 2 to 20 mcg/kg/min

Sinoatrial [SA] Node Rhythms

Sinus Tachycardia



Sinus tachycardia occurs when the SA node is firing at a rate that is faster than normal for a person's age. The rate is generally **101 to 150 bpm**. The key to sinus tachycardia is that all components of a normal ECG are present: P wave, QRS complex, and T wave. Sinus tachycardia generally starts and stops gradually. Causes can be numerous, such as: pain, fever, fluid and/or blood loss [hypovolemia and/or dehydration], beta blocker withdrawal, CHF, hypoxemia, caffeine, alcohol withdrawal, a recent MI, or anxiety/agitation are some of the causes that can be identified and treated.

Atrioventricular [AV] Blocks

These blocks fall into three categories: First Degree, Second Degree Types I & II [two types], and Third Degree



First Degree Block



First-degree atrioventricular block (AV block), or PR prolongation, is a disease of the electrical conduction system of the heart in which the PR interval is lengthened beyond 0.20 seconds [or five (5) small squares on the ECG]. In first-degree AV block, the impulse conducting from the atria to the ventricles, through the atrioventricular node (AV node), is delayed and travels slower than normal. First Degree Block can be caused by medications such as: Digoxin, Calcium Channel Blockers, and Beta Blockers.

If the R is far from P, then you have a First Degree

Second Degree Block - Mobitz / Type I [Wenckebach]



Second Degree AV Block Type 1 is a disease of the AV node. This heart block is characterized by **progressive prolongation of the PR interval** on the electrocardiogram (ECG) on consecutive beats followed by a blocked P wave (i.e., a 'dropped' QRS complex). After the dropped QRS complex, the PR interval resets and the cycle repeats itself. This particular rhythm can be caused by medications such as: Digoxin, Calcium Channel Blockers, and Beta Blockers. Cardiac ischemia found in the Right Coronary Artery can also cause this rhythm.

Normal, longer, longer, 'drop'... then you have Wenckebach

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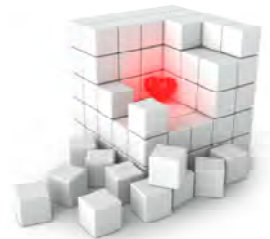
Second Degree Block - Mobitz / Type II



Second-degree AV block Type 2, also known as "Mobitz II," is almost always a disease of the distal conduction system (His-Purkinje System). This block can often cause compromised cardiac output which can lead to a complete AV Block.

Mobitz II heart block is characterized on a surface ECG by intermittently non-conducted P waves not preceded by PR prolongation and not followed by PR shortening. **When there is a PR interval, it is usually within normal limits**, but could be consistently prolonged. However, there will be dropped beats characterized by P waves without a QRS complex [the beat is blocked at the AV node]. The number of blocked beats can vary in number and will be irregular. This is what makes this such a dangerous block.

If some Ps don't get through, then you have a Mobitz II



Second-degree AV block - Fixed Conduction

There is an additional type of Second Degree AV Block - **Mobitz II with a fixed ratio/conduction of P waves to QRS complexes**. It is still a dangerous block that can lead to Third Degree Block and Death unless treated. This type of Mobitz II is characterized by **a regular number of P waves before every QRS complex**. It will usually present as a regular 2:1 conduction [2 P waves before every QRS], or 3:1 conduction [3 P waves before every QRS], or 4:1 conduction [4 P waves before every QRS], etc.

Example of: Mobitz II with 2:1 conduction



Third Degree Block - Complete Heart Block



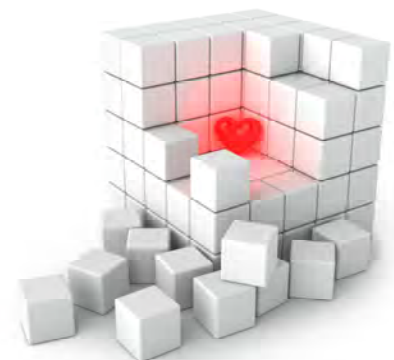
Third-degree atrioventricular block (AV block), also known as **complete heart block**, is a serious medical condition in which the impulse generated in the sinoatrial node (SA node) in the atrium of the heart does not propagate to the ventricles.

Because the impulse is blocked, there is an accessory pacemaker in the lower chambers that will typically activate the ventricles. This is known as an **escape rhythm**. Since this accessory pacemaker also activates independently of the impulse generated at the SA node, this is a very dangerous block because two independent “rhythms” can be noted on the ECG. This rhythm is often associated with cardiac ischemia involving the Left Coronary Arteries

You will find that the P waves and QRS complexes are regular, but not associated with each other. The P waves [usually 60 to 100 bpm] will march out regularly throughout the rhythm. The QRS complexes [usually 30 to 40 bpm] will also be regular and march out. They just don't associate.

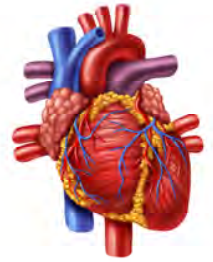
If Ps and Qs don't agree, then you have a Third Degree

Further Notes on Heart Blocks:

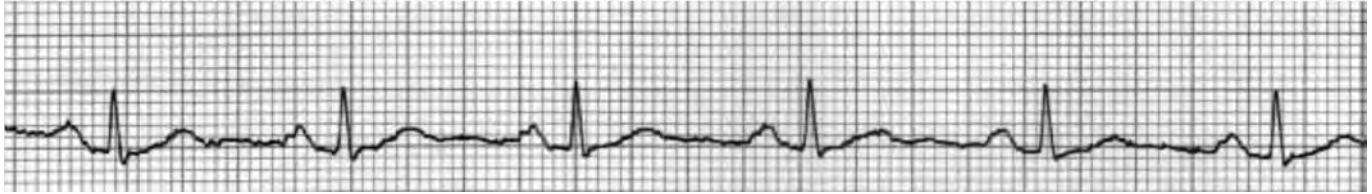


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Cardiac Arrest Rhythms



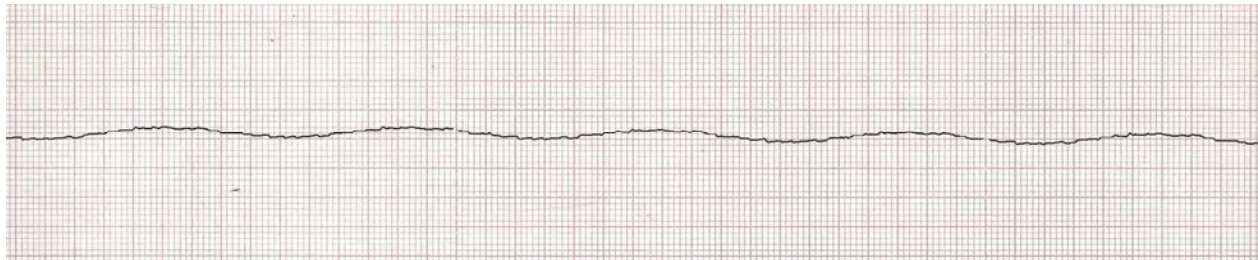
Pulseless Electrical Activity (PEA)



Pulseless Electrical Activity (PEA) occurs when the heart has an electrical beat but without the heart mechanically pumping. It can be any organized rhythm, but the patient does not have a pulse. Start or continue CPR immediately.

Identification of the underlying causes is essential. Use your H's & T's to try and correct the problems.

Asystole



Asystole is a cardiac arrest rhythm in which there is no discernible electrical activity on the ECG monitor and the patient does not have a pulse. Asystole is sometimes referred to as a "flat line." To confirm Asystole you should:

- Check for and confirm that there is no pulse
- Check the leads to make sure they are attached
- Check the rhythm in a second lead to confirm the asystole and to make sure the patient is not in fine V-Fib.
- Treat according to the **Pulseless, Non-shockable Algorithm**

PEA / Asystole

- CPR
- Epi 1mg of a 1:10,000 solution every 3 to 5 minutes [No Limit / Maximum amount]
- Consider the H's & T's

Cardiac Arrest Rhythms



Ventricular Fibrillation

V-Fib or VF is the most common rhythm that occurs immediately after cardiac arrest. In this rhythm, the heart beats with rapid, erratic electrical impulses. This causes the ventricles to quiver uselessly and are unable to uniformly contract to pump blood. It is for this reason that early defibrillation is so imperative. A victim's chance of survival diminishes rapidly over time once the heart goes into V-Fib; therefore, each minute counts when initiating defibrillation.

There are two types of VF, fine and course. Course VF usually occurs immediately after a cardiac arrest and has a better prognosis with defibrillation. Fine VF has waves that are nearly flat and look similar to asystole. Fine VF often develops after more prolonged cardiac arrest and is much more difficult to correct.

Fine V-fib:



Course V-fib:



Treatment:

- **Shock / Defibrillation every 2 minutes in a single one shock, successive, shockable increments**
 - **200 joules - Followed by immediate CPR for 2 minutes / give and circulate a drug(s)**
 - **300 joules - Followed by immediate CPR for 2 minutes / give and circulate a drug(s)**
 - **360 joules - Followed by immediate CPR for 2 minutes / give and circulate a drug(s)**
- **Drugs**
 - **Give Epinephrine 1mg of a 1:10,000 solution every 3 to 5 minutes [No Limit]**
 - **Give either:**
 - **Amiodarone [if not contraindicated, can be given 2x]: 300mg first dose / 150mg second dose at 3 to 5 minutes increments.**
 - **Lidocaine: First dose: 1mg/kg or 1.5 mg/kg. Can repeat it at half the original dose up to a total of 3mg/kg [Second and remaining doses are given at either 0.5mg/kg or 0.75mg/kg depending on your starting dosage.]**
 - **Important: You must choose either/or. You cannot alternate the drugs nor give Lidocaine after giving Amiodarone since it changes the metabolic structure of the drug's affect in the body.**

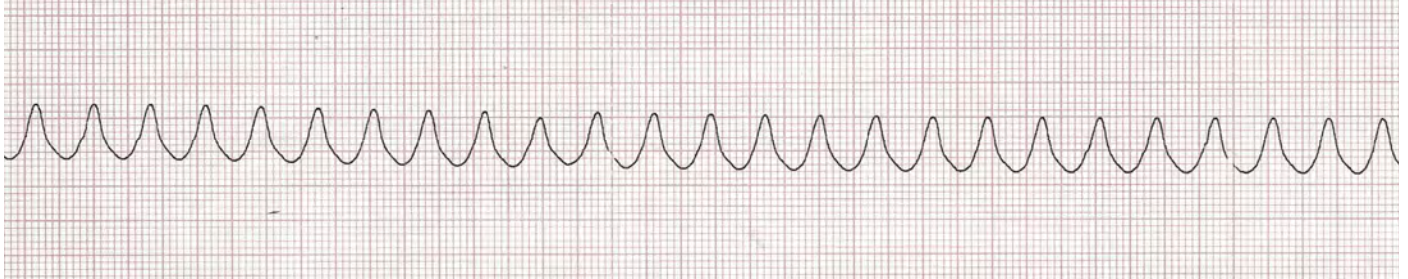
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Cardiac Arrest Rhythms



Pulseless Ventricular Tachycardia

Monomorphic:



The most important question to ask is: “Does this person have a pulse?”

Ventricular Tachycardia (VT) can present itself with or without a pulse. When VT is present and the victim **has no pulse**, the treatment is the same as VF. Pulseless VT can rapidly deteriorate to VF

Electrical defibrillation in high dose shocks for VF/PVT will give the best chance for converting the patient out of pulseless VT. In fact, as with VF, the earlier defibrillation occurs, the higher the survival rate.

Treatment for Pulseless V-Tach is the same as V-Fib

Note: Vasopressin has been removed from the 2015 AHA Guidelines for VF and Pulseless VT. The AHA states that Vasopressin offers no advantage as a substitute for Epinephrine in cardiac arrest

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Bradycardic Rhythms

- Sinus Bradycardia is any rhythm where the heart rate is < 60 bpm. Bradycardia usually involves one of the following rhythms:

- Sinus Bradycardia
- First degree AV block
- Second degree AV block
 - Type I (Mobitz I / Wenckebach)
 - Type II (Mobitz II or Fixed)
- Third degree AV block

The Key Clinical Questions to ask are, Is the Bradycardia causing the patient's symptoms or Is there some illness perhaps causing the Bradycardia

- Sinus Bradycardia in a patient can have multiple causes. Some will require treatment, while at other times it will not, often depending on the cause(s) and the physical condition / health of the patient. Bradycardia can present itself as either **'Stable or Unstable.'**

- **There are three criteria to determine if a patient with Bradycardia is Symptomatic/ Unstable.** They are:

- **The Heart rate is SLOW**
- **The patient has SYMPTOMS**
 - Unstable Signs and Symptoms can include:
 - Chest discomfort
 - Shortness of Breath / Dyspnea
 - Decreased Level of Consciousness
 - Weakness
 - Fatigue
 - Syncope, or Pre-syncope
 - Fatigue
 - Lightheadedness
 - Hypotension
 - Diaphoresis

- **The symptoms are due to the SLOW HEART RATE**

Treatment for Bradycardia:

- Treating Bradycardia will be determined by the severity of the patient's condition
- **THE** primary determining factor in the algorithm decision on how aggressive we need to be in treating the patient is determined by :
 - Asking '**How well is the patient PERFUSING?**' and
 - The **severity of the Patient's Condition**

<p style="text-align: center;">If Stable [Perform on every patient]</p>	<ul style="list-style-type: none">• Do your Primary [Life-threatening] and Secondary [S.A.M.P.L.E] Surveys to determine the new onset and possible cause(s) of the Bradycardia• Establish an IV, Draw Labs, and obtain Vital Signs• Perform a 12-Lead• Oxygen as needed• Consider the H's & T's• Seek expert Medical Consultation
<p style="text-align: center;">If Unstable / Poor Perfusion [In addition to the above treatment]</p>	<ul style="list-style-type: none">• Consider:<ul style="list-style-type: none">• Atropine at 0.5mg IV [maximum 3mg]• Transcutaneous Pacing• Epinephrine 2 - 10 mcg/min<ul style="list-style-type: none">• Titrate to patient's response• Dopamine 2 - 20 mcg/kg/min<ul style="list-style-type: none">• Titrate to patient's response

Tachycardia

Tachyarrhythmias are rhythms when the heart rate is greater than 100 bpm. This includes Rhythms that begins in the SA node, Atrial tissue, or the AV junction. When the rhythms arise from above the bundle branches, they are characterized by narrow QRS complexes. When they don't, the QRS will be wide in it's complex.

Tachycardias can be classified in several ways based upon the appearance of their:

- **QRS complex**
- **Heart Rate**
- **Regular or Irregular**
- Those same rhythms can be either **Stable or Unstable** and can include:
 - **Sinus Tachycardia**
 - **Atrial Fibrillation**
 - **Atrial Flutter**
 - **Reentry Supraventricular Tachycardia (SVT)**
 - **Monomorphic VT**
 - **Polymorphic VT**
 - **Wide-complex tachycardia of uncertain type**

Just as in Bradycardia, the Healthcare Provider needs to determine / ask several things:

- **Are there pulses present?**
- **Is the patient Stable or Unstable?**
 - Hypotension
 - Signs of Shock
 - Dyspnea / Shortness of Breath
 - Chest Pain
 - Heart palpitations
 - Lightheadedness
 - Altered mental status
 - Syncope
 - Acute Heart Failure
- **Are symptoms due to the tachycardia?**
- **Is the QRS complex narrow or wide?**
- **Is the QRS monomorphic or polymorphic?**
- **Is the rhythm Regular or Irregular?**
- **What is causing the tachycardia?**
- **Will treatment improve the patient's Signs and Symptoms?**

The Key Clinical Questions to ask are:

Is the Tachycardia causing the hemodynamically instability with the patient's current serous Signs and Symptoms?

Or

Is the distress and pain the patient is having a direct result from the AMI and therefore causing the tachycardia?

Depending on the answers received will determine the subsequent diagnosis and treatment

The Difference between Synchronized and Unsynchronized Shocks



Unsynchronized Shock Also known as **Defibrillation** simply means that the electrical shock is delivered as soon as the healthcare provider pushes the Shock Button on the device. The shock can fall randomly anywhere within the cardiac cycle. Usually these shocks are at a higher energy dose than the synchronized shock.

Synchronized Cardioversion Uses a sensor in the defibrillator machine itself, to deliver the shock at a specific point in the rhythm - at the peak of the QRS complex which is the highest point [on the R wave]. When the healthcare provider presses the Sync Button and 'capture' is achieved, there will be a *delay* in the delivery of the energy when the shock button is pressed because the device will synchronize the shock to deliver the joules at the highest point of the QRS / R wave. It is avoiding the delivery of the shock during the cardiac repolarization period [T wave] which could precipitate VF. Often , synchronized cardioversion will use a much lower energy dose than when defibrillating / using unsynchronized shocks.

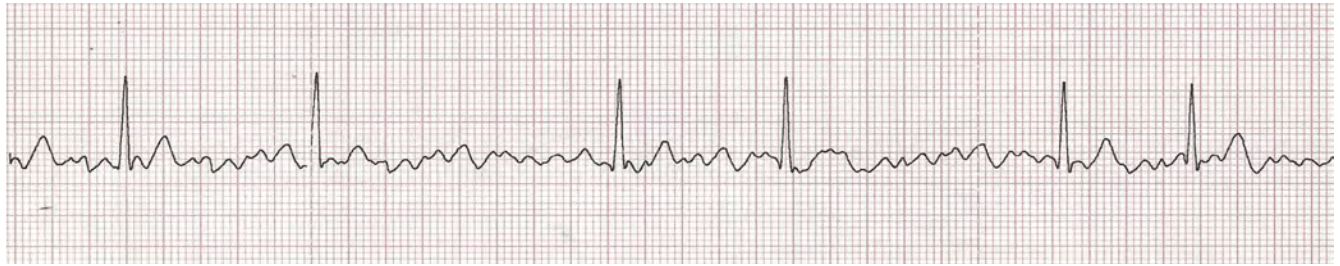
Note: Synchronized Cardioversion is relatively simple, but problems can occur. For further study on this matter, see The 2015 AHA ACLS *Provider Manual*. It is also unlikely that synchronized cardioversion will be effective in the treatment of Junctional Tachycardia, Ectopic Tachycardia, or Multifocal Tachycardia because of cells spontaneously depolarizing at a very rapid rate. Trying to stop these rhythms with synchronized cardioversion can actually *increase* the rate of the tachyarrhythmia.

When to Use Unsynchronized Shocks / Defibrillation

- For a patient who is in VF or pulseless VT
- When you are unsure whether monomorphic or polymorphic VT is present in the Unstable patient
- For a patient demonstrating clinical deterioration [such as in Prearrest], when those in severe shock or polymorphic VT, when you think a delay in converting the rhythm will result in cardiac arrest.
- **When to Use Synchronized Shocks** [IISF = increasing in stepwise fashion] [J = Joules]
- Unstable SVT **50 - 100 J** [IISF]
- Unstable Atrial Flutter **50 - 100 J** [IISF]
- Unstable Atrial Fibrillation **120 - 200 J [Biphasic]** [IISF] / **200 J [Monophasic]**
- Unstable Tachycardia with pulses
 - Wide Regular **100 J**
 - Wide Irregular **Defibrillation [not synchronized cardioversion]**

Atrial Fibrillation

Atrial Fib: SLOW



Atrial Fib: FAST [Uncontrolled with RVR]



Atrial fibrillation is an irregular and often rapid heart rate [known as: Atrial Fibrillation with Rapid Ventricular Response (Atrial Fib w/ RVR)] that can increase risks of: Stroke, Heart failure and other heart-related complications.

During atrial fibrillation, the heart's two upper chambers (the atria) beat chaotically and irregularly [multiple sites in the atrium are trying to 'assist' the heart by becoming the primary pacemaker site, since the SA Node is no longer predominant] — thus, out of coordination with the two lower chambers (the ventricles) of the heart [since the ventricles no longer have a 'primary voice - SA Node to listen to.] Atrial fibrillation symptoms can often include: Heart palpitations, Shortness of breath and Weakness.

Episodes of atrial fibrillation can come and go, or you can develop atrial fibrillation that doesn't go away and may require treatment. Although atrial fibrillation itself usually isn't life-threatening, it *can be* a serious medical condition that sometimes requires emergency treatment and lead to complications.

Atrial fibrillation can lead to blood clots forming in the heart that may circulate to other organs and lead to blocked blood flow (ischemia). Recent studies have also indicated that individuals who drink large amounts of energy drinks and caffeine have an increased risk of developing uncontrolled Atrial Fib early in life.

Treatments for atrial fibrillation may include: Medications and other interventions [e.g. synchronized cardioversion] to try to alter the heart's electrical system.

Unstable Atrial Fibrillation

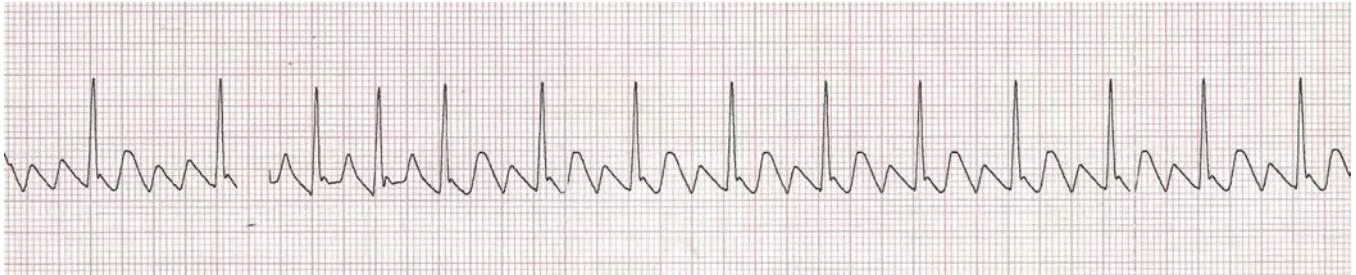
- **Synchronized Cardioversion 50 - 100 J**

Atrial Flutter

1 to 1 Conduction



2 to 1 Conduction



3 to 1 Conduction



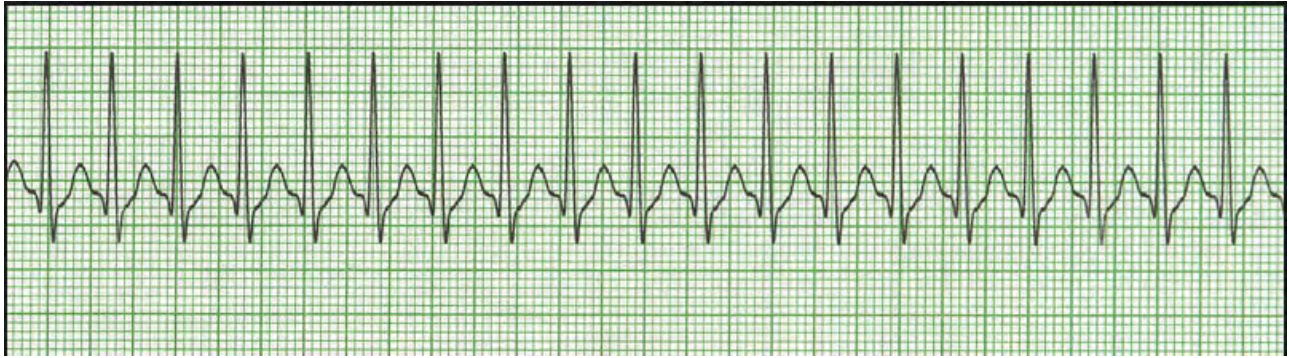
Atrial flutter is caused by problems with the heart's electrical system which causes a type of rhythm where your heart's upper chambers (atria) beat too quickly. This causes the heart to beat in a fast, regular rhythm where the atria fires more rapidly than the ventricles. The P Waves are characterized by what we call Flutter Waves that have a 'shark fin, fence post, and saw-toothed'. They are predominantly uniform in nature and present in a 1:1, 2:1, 3:1, etc. conductive, regular pattern and rhythm. Atrial Flutter will usually produce a heart rate of approximately 150 bpm, but can get as high as 250 - 350 bpm. Patients are usually stable if there is no serious heart disease, *however* Atrial Fib *may* be the first indicator of impending cardiac disease.

Note: Adenosine will *not* have any affect on Atrial Flutter.

Unstable Atrial Flutter

- **Synchronized Cardioversion 50 - 100 J**

Supraventricular Tachycardia (SVT)



With Supraventricular Tachycardia (SVT) the rate can be so fast that you may not even see any P waves since they 'run into' the preceding T waves. Depending on your reference materials, SVT's rates can range from **150 / 180 to 250+ bpm**, and the rhythm is almost always regular [which distinguishes it from Rapid Atrial Fibrillation.]

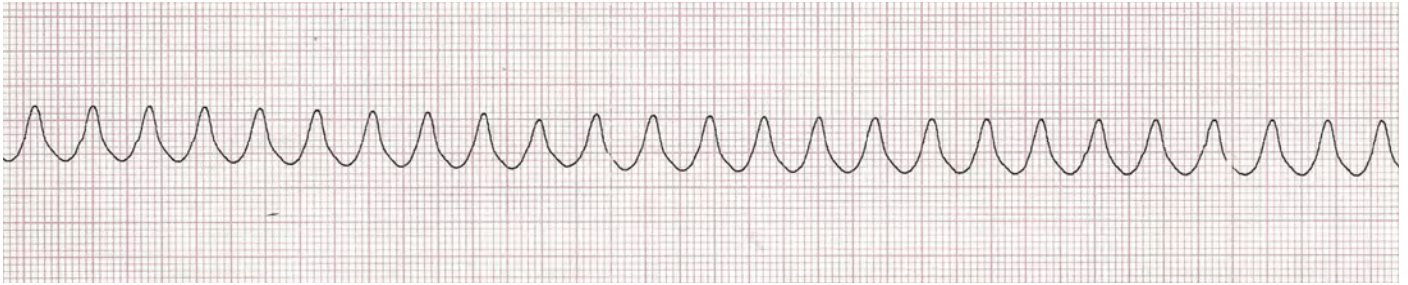
Patients with SVT/PSVT can experience Dyspnea, Palpitations, Hypotension, Angina/Chest Pain, Anxiety, and Lightheadedness. It can also be related to such things as: Anxiety, Caffeine, Stress, Drugs, and Nicotine.

PSVT [Paroxysmal Supraventricular Tachycardia] is SVT that starts and stops suddenly [and for accurate interpretation, both the beginning and ending of the (P)SVT must be seen. Characteristics are the same. PSVT may sometimes be referred to as Paroxysmal Atrial Tachycardia (PAT),

Stable SVT	<ul style="list-style-type: none">• Attempt Vagal Maneuvers<ul style="list-style-type: none">• Diving Reflex [Ice to the Face]• Valsalva Maneuver [Hold your breath and bear down like your going to have a bowel movement]• Blow through a straw or the big end of an empty syringe• Give Adenosine<ul style="list-style-type: none">• Give 6mg of Adenosine over 1 - 3 seconds, RAPID IV PUSH at the antecubital or other large vein, followed by an immediate 10 - 20cc flush of Normal Saline, and raise the arm for 10 seconds• Repeat Adenosine at 12mg after 1 to 2 minutes if the first dose was ineffective, in the same manner as directed above. Note: The half life of Adenosine in the blood stream is less than 10 seconds.
Unstable SVT	<ul style="list-style-type: none">• Consider Sedation• Synchronize Cardiovert at 50 - 100 J [increasing in a stepwise fashion]

Ventricular Tachycardia (with a pulse)

Monomorphic:

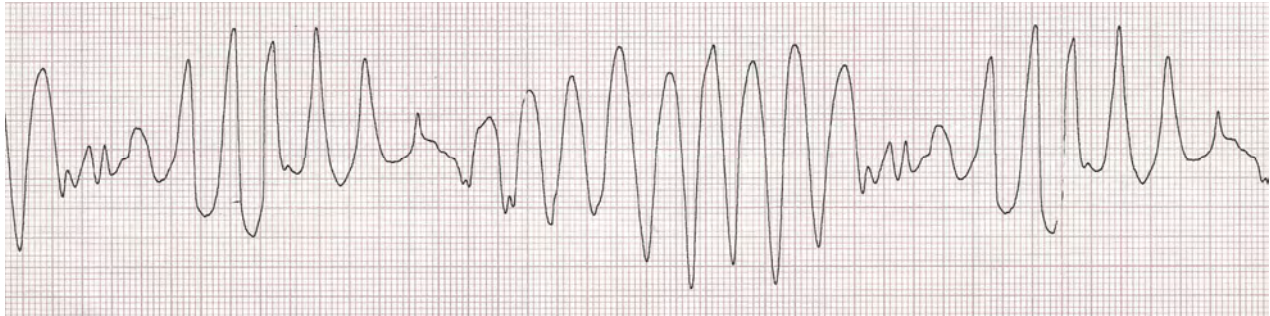


Ventricular Tachycardia (VT) As stated earlier, VT can present itself with either with a pulse or without a pulse. When the VT is a *Stable Wide-QRS Tachycardia WITH a pulse*, there is both Drug Therapy and Synchronized cardioversion treatment to consider depending on the condition of the patient.

Antiarrhythmic Infusions	
Stable Wide-QRS Tachycardia	<ul style="list-style-type: none">• Procainamide IV dose: 20 - 50 mg/min until arrhythmia is suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose of 17mg/kg if given. Maintenance infusion: 1 - 4 mg/min. Avoid if prolonged QT or CHF• Amiodarone IV dose: First dose: 150mg over 10 minutes. Repeat as needed if VT recurs. Follow up with a maintenance infusion of 1mg/min for 6 hours.• Sotalol IV dose 100mg (1.5mg/kg over 5 minutes. Avoid prolonged QT
Unstable Wide-QRS Tachycardia	<ul style="list-style-type: none">• Synchronized Cardioversion [Wide Regular <i>with</i> a pulse]: 100 J

Ventricular Tachycardia

Polymorphic: [Torsades de Pointes]



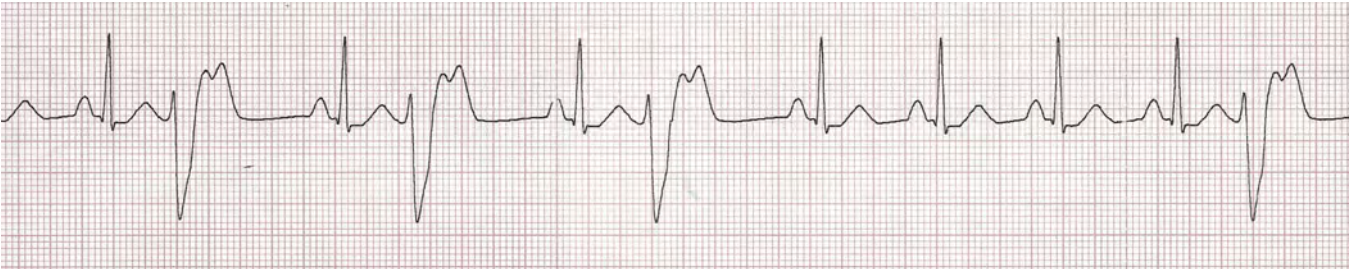
Torsades de pointes (TdP) is a distinctive polymorphic ventricular tachycardia in which the QRS amplitude varies [irregular] and the QRS complexes appear “to twist” around the baseline. Torsades de Pointe is often associated with a prolonged QT interval, which may be congenital or acquired.

Torsades de pointes is usually not sustained and terminates spontaneously. However, it can frequently recur unless the underlying cause is corrected. Torsades may degenerate into sustained pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF) and sudden cardiac death. **Torsades is a life-threatening arrhythmia** and may present as sudden cardiac death in patients with structurally normal hearts.

<p>(Stable) Wide Irregular QRS Tachycardia Torsades de Pointes</p>	<ul style="list-style-type: none">• Magnesium: Intravenous magnesium is the drug of choice for Torsades de Pointes. Magnesium is effective even in patients with normal magnesium levels.<ul style="list-style-type: none">• In Cardiac Arrest (Due to Hypomagnesemia or Torsades de Pointes): 1 to 2 g (2 to 4 ml of a 50% solution diluted in 10ml [eg, D5W, normal saline] given IV/IO)• Torsades de Pointes With a Pulse or AMI with Hypomagnesemia: Loading dose of 1 to 2 g mixed in 50 to 100 ml of diluent (eg, D5W, normal saline) over 5 to 60 minutes IV. Follow with 0.5 to 1 g per hour IV (titrate to control torsades)• Discontinuation of any offending agent (stop all QT-prolonging drugs) and correction of any underlying cause such as hypokalaemia, hypomagnesaemia and bradycardia.• Use with caution: If Renal Failure is present, or there may be an occasional fall in blood pressure with rapid administration that may occur
<p>Unstable Wide-QRS Tachycardia Torsades de Pointes</p>	<ul style="list-style-type: none">• Treat as VF with high energy defibrillation / unsynchronized shocks in increasing doses

Premature Ventricular Contractions - PVCs

Unifocal PVCs



Multi-focal PVCs



Couplets



Premature ventricular contractions (PVCs) are extra, abnormal heartbeats that begin in one of your heart's two lower chambers (ventricles). These extra beats can disrupt your regular heart rhythm, sometimes causing you to feel a skipped beat in your chest. Premature ventricular contractions can be very common and often occur in most people at some point for a variety of reasons.

Premature ventricular contractions are also called:

- Premature ventricular complexes / PVCs or (sometimes) Ventricular premature beats
- PVCs that look the same are called **Unifocal PVCs**. PVCs that are different shaped are called **Multifocal PVCs**. A PVC every other beat is known as **Bigeminy**. Every third beat - **Trigeminy**. Every 4th beat - **Quadrigeminy**. Two or more PVCs together, are called **Couplets**. Three or more PVCs *used to be called* 'Triplets,' but are now referred to as a **Short run of V-Tach**

Healthy individuals can experience occasional PVCs that can be caused by a number of reasons. However, if you have any underlying heart problems or disease, and experience PVCs, treatment is needed.

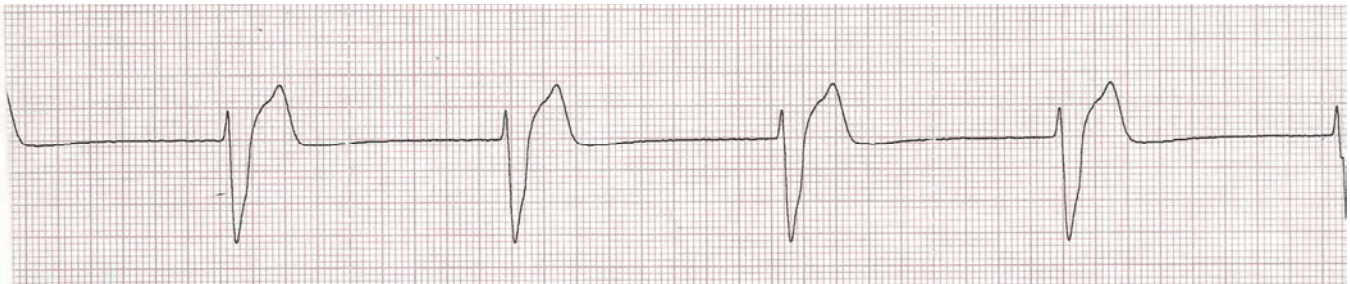
Other Rhythms

Junctional Rhythm



Junctional heart rhythm is an abnormal heart rhythm resulting from impulses coming from a location in the area of the atrioventricular (AV) node, at the "junction" between the atria and ventricles, other than the SA Node. Its rate can be from bradycardic to tachycardic. It usually will present without a P wave or with an inverted P wave

Idioventricular Rhythm



The **Idioventricular Rhythm** rate is usually between 30 - 40 beats per minute and occurs because the ventricles are not receiving any messages from the SA Node or at any other Junctional Point in-between. The ventricles become the primary pacemaker of the heart and this rhythm usually can, and often has a very poor outcome. If the rate is above 40, it is called an **accelerated idioventricular rhythm**.

Pacemaker Rhythm



A **Pacemaker** will often present with what is known as a **Pacing Spike** [pictured above]. The spike indicates that the pacemaker has fired. The pacer spike can occur before the Atrium or Ventricle depending on where capture occurs. If there is atrial capture, the spike will appear before the P wave with small p waves following each pacer spike. If you have ventricular capture, the pacer spike will appear before each QRS, therefore followed by a QRS each time there is capture.

Some further thoughts on ECG Strips:

Again, this is by no means a **comprehensive** study or listing of all the ECG Rhythms. There are others that we have not touched on. ECG features and rhythms such as:

- J points
- Sinus Pause/Arrest
- Sinus Arrhythmia
- Wolffe-Parkinson-White Syndrome [WPW]
- Premature Atrial Contractions [PACs]
- Premature Junctional Contractions [PJs]
- Junctional Tachycardia or Escape Beats
- Atrial Tachycardia
- Different ways to count and determine the rate, and the list goes on.

However, we have tried to give you an accurate initial resource for your study and review. We encourage you to take the initiative to continue your study in ECG Rhythms in order to give exceptional, excellent, and compassionate patient care. Contact us at www.4cpr.org for further classes available.

H's & T's

Problem or Possible correctable causes : When working a code, or with anyone having heart problems of any type, it is essential that we determine any possible underlying problems that may have caused the patient's current condition, and they we can correct. **[We will go over each of these more in-depth in class]**

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo / Hyperkalemia
- Hypothermia
- Toxins
- Tension pneumothorax
- Tamponade, cardiac
- Thrombosis, pulmonary
- Thrombosis, coronary

Things to consider after ROSC

- Monitor vital signs [Blood Pressure, etc.], Obtain a 12 lead ECG, Monitor O2 Sat, Order and Recheck Labs, Maintain the airway- [Intubate if necessary. If intubated, monitor with waveform capnography **Remember:** Waveform capnography is the best way to monitor continuous ETT placement], Take the patient to the Cath Lab if appropriate
- **'Blast from the Past':** Remember when everyone received a 100% via non-rebreather mask? **Not any more!**
Maintain SpO2 >94% on the least amount of O2 as possible. **Important:** Too high of an O2 concentration can cause oxygen toxicity
- **Warning:** Excessive ventilation can cause a(an):
 - Increase in Intrathoracic Pressure
 - Decrease venous return
 - Decrease cardiac output
- Treat hypotension (systolic B/P < 90) with an IV fluid bolus first. If unsuccessful, then you can try drugs (Epinephrine, Dopamine or Norepinephrine)

Targeted Temperature Management (Formerly Therapeutic Hypothermia - and often referred to in hospitals as: **Artic Sun**, **Code Cool**, etc.)

To protect the brain and other organs, the resuscitation team should induce therapeutic hypothermia in adult patients who remain comatose (lack of meaningful response to verbal commands) with ROSC after out-of-hospital VF Cardiac Arrest.

- Healthcare providers should cool patients to a target temperature of **32C to 36 C for at least 24 hours.**
- Induced hypothermia should not affect the decision to perform PCI, because concurrent PCI and hypothermia are reported to be feasible and safe.



- Transfer patient to a PCI capable hospital

ACUTE STROKE

It refers to acute neurologic impairment that follows interruption in blood supply to a specific area of the brain.

Two types of Strokes:

- **Ischemic Stroke** – Accounts for 87% of all strokes and is usually caused by an occlusion [blockage] of an artery to a region of the brain
- **Hemorrhagic Stroke** – accounts for 13% of all strokes and occurs when a blood vessel in the brain suddenly ruptures into the surrounding tissue [bleed].

The goal of stroke care is to minimize brain injury and maximize the patient's recovery.

- Rapid Recognition and reaction to stroke warning signs

• **F.A.S.T.** **Face** **Arm Drift** **Speech** **Time**

- Rapid EMS dispatch
- Rapid EMS system transport and pre-arrival notification by alerting the receiving hospital with a possible Stroke Alert
 - EMS should do a thorough and rapid assessment of the Stroke Patient
 - Determine the time of ONSET
 - **Ask:** *'When was the last time the patient appeared normal?'*
 - If the patient meets the necessary criteria, **rtPA** must be administered as soon as possible, as long as it is within the **3 to 4.5 hour window**
 - Treatment for the patient should be performed enroute and not on scene
 - Transport the patient to a Stroke Center Hospital
 - Rapid diagnosis and treatment in the hospital
 - The goal of the stroke team, emergency physician, or other experts should be to assess the patient with suspected stroke within 10 minutes of arrival in the ED.

- **Patients with a stroke who require hospitalization should be admitted to a hospital with a stroke unit, even if it is further away.**
- **Stable stroke patients should have a non-contrast CT scan performed within 25 mins of arrival in the ED and should be read within 45 minutes from performance to determine the type of stroke! Remember: "TIME IS BRAIN"**

Note: All strips are taken from an actual cardiac monitor and dysrhythmia machines owned by our company.

Note: Treatment and Descriptions are based on the Diagrams, terminology, and treatment taken from the guidelines as set forth in the American Heart Association's ACLS Provider Manual and ECC Handbook.

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