Course Overview

This Study Guide is an extensive outline of content that will be taught in the American Heart Association Accredited Advanced Cardiovascular Life Support (ACLS) 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 required that the student will have the 2015 Updated AHA ACLS Textbook. There is a password on page ii of the textbook that will allow the student to access an AHA student website with extensive additional materials for study and reference. The 2015 ECC Handbook is also helpful to have readily available for review and as a reference.

Agenda

Welcome, Introduction
ACLS Course Overview/ EKG and Algorithms Review
BLS and ACLS Surveys
Management of Respiratory Arrest/Skills Testing
CPR and AED Practice and Testing
ACLS Algorithms Review/Post Resuscitation Care
Megacode and Resuscitation Team Concept
Acute Coronary Syndromes (Certification only)
Stroke (Certification only)
Putting It All Together
Megacode Skills Test
Written Evaluation

Evidence Based Updates

As new research findings suggest change, the AHA updates the guidelines for CPR and Emergency Cardiovascular Care. These updates are necessary to ensure that all AHA courses contain the best information and recommendations that can be supported by current scientific evidence. Evidence based guidelines were developed, documented, debated and then evaluated by scientific experts from outside the United States and outside the AHA. The guidelines were then classified as to the strength of evidence that supports the recommendation.

Objectives

Upon the completion of this ACLS course the participant will be able to:

♥ Identify lethal rhythms
♥ Verbalize the primary survey approach to Emergency Cardiovascular Care (ECC)
♥ Verbalize the secondary survey approach to ECC
♥ Verbalize treatment algorithms for each of the following lethal rhythms:
  Pulseless arrest
  Bradycardia
  Tachycardia
  Acute coronary syndrome
♥ Verbalize steps to determine ischemic stroke and subsequent treatment
♥ Describe the appropriate energy dosing for biphasic defibrillation
♥ Describe the difference between defibrillation and cardioversion
♥ Act as a team leader in a code simulation
Normal Anatomy Review

In order to understand Advanced Cardiovascular Life Support, it is essential to understand normal cardiac function. By understanding the normal electrical pathways in the heart, it will be easier to understand abnormal function. When blood enters the atria of the heart, an electrical impulse is sent out from the SA node that conducts through the atria causing them to contract. The atrial contraction registers on an EKG strip as the P wave. This impulse then travels to the AV node that in turn sends out an electrical impulse that travels through the Bundle of His, bundle branches, and into the Purkinje fibers of the ventricles causing them to contract. The ventricular contraction registers on the EKG strip as the QRS complex. Following ventricular contraction, the ventricles rest and repolarize. This repolarization is registered on the EKG strip as the T wave. The atria repolarize also, but this coincides with the QRS complex and therefore cannot be observed on the EKG strip. Together a P wave, QRS complex, and T wave indicate a Sinus Rhythm.
In general, narrow QRS complexes originate in the atria, at the junction of the heart or near the AV node. Wide QRS complexes usually indicate that a rhythm is originating below the bundle of His or in the ventricles.

Typically, when looking at an EKG strip, a patient will be hooked up to a monitor or a printout will read the heart rate. However, this is not always the case. It is important to be able to determine a heart rate when the monitor or printout rate is not given. There are two ways that will be discussed to determine the heart rate.

1. The most common way to determine heart rate is to count the QRS complexes on a six second strip and then multiply by 10 to give a rate per minute.

2. The second way works especially well in patients without a 6 second strip and in tachycardia patients. In tachycardia patients, it can be time consuming to count the number of QRS complexes on a six second strip. A better method is to memorize the numbers 300 – 150 – 100 – 75 – 60 – 50, as is shown in the diagram on the previous page. One suggestion is to memorize them in triplets “300-150-100” “75-60-50”. It has a nice rhythm. Starting from a QRS complex that falls on a heavy line, count 300 on the next heavy line, then 150 on the next heavy line, and so on until the next QRS complex is reached. This will give a range as to the heart rate with accuracy enough to determine tachycardia or normal rhythm. For bradycardia, counting the QRS complexes on a six second strip will be faster.

3. A third method is to count the number of large boxes between QRS complexes and divide 300 by that number. For example, in the picture above, there are just over 4 boxes between QRSs, so 300 divided by 4 is 75. The rate is approximately 75bpm.
Cardiac Arrhythmias

Pulseless Rhythms

Ventricular Fibrillation
Ventricular Fibrillation (V-Fib or VF) is the most common rhythm that occurs immediately after cardiac arrest. In this rhythm, the ventricles quiver 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.

V-fib = Defib.

Defibrillation stops the heart, like rebooting a computer, and allows it to restart with a corrected rhythm (hopefully).

There are two types of VF, fine and coarse VF. Coarse 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. Caution: Sometimes artifact can look like VF, but we know to always check our patient.

Ventricular Tachycardia
1. Stable vs. Unstable
2. Pulse vs. No pulse

Since this section is about pulseless rhythms, we are looking at Ventricular Tachycardia (VT) without a pulse. Ventricular Tachycardia will be discussed in more detail later. When a VT is present and the victim has no pulse, the treatment is the same as with VF. High dose shocks for defibrillation will give the best chance for converting the patient out of pulseless VT.
Pulseless Electrical Activity

Pulseless Electrical Activity (PEA) occurs when the heart is beating and has a rhythm, it can be any rhythm, but the patient does not have a pulse. **Always treat the patient, not the rhythm strip.** The number one question in this situation is, “Why?”

1. Problem or Possible correctable cause (H’s & T’s)
2. Epinephrine 1 mg 1:10,000--give to anyone WITHOUT a pulse

![ECG Image](image)

The possible causes are referred to as “H’s & T’s” and are the following:

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

In order to treat a pulseless rhythm, bradycardia, or tachycardia, identification of the possible underlying causes is essential. If a cause is not identified, all of the drugs in the world will not cure the problem. For example, if a patient is hypovolemic, unless he or she gets more fluids, it will be impossible to correct the problem.

Asystole

Asystole is when there is no detectable cardiac activity on EKG. It may occur immediately after cardiac arrest or may follow VF or PEA. Asystole may also follow a third degree heart block. **Treatment of asystole is the same as PEA.** The American Heart Association recommends that if a patient is in sustained Asystole for 15 minutes, it is reasonable to call the code, but involve the family in the decision if they are available.

![ECG Image](image)
Bradycardia

Bradycardia occurs when the heart is beating too slowly—less that 50 beats per minute. If symptomatic, provide oxygen, give Atropine 0.5mg, and call for the transcutaneous pacemaker.

Sinus Bradycardia

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 50 due to their physical conditioning. Obviously, they would not need treatment. Some patients may have heart rates less than 50 and be asymptomatic. However, if a patient with a heart rate less than 50 has signs of poor perfusion; begin treatment with oxygen and Atropine 0.5 mg.

First-Degree AV Block—All P waves conducted through the AV node, but delayed

First-degree AV block = prolonged PR interval (> 0.20 seconds or 5 small boxes on the EKG strip)

In first-degree AV block, all of the components of the EKG strip are normal except the PR interval. What happens in this situation is that the impulse from the SA node is delayed at the AV node. All impulses are, however, conducted through the AV node following the delay.

Second-Degree AV Block Type I, (Mobitz I, Wenckebach)—Some P waves conducted through the AV node, others blocked*

Second-degree AV block type I (Mobitz I, or Wenckebach) = **progressive lengthening of the PR interval** with dropped QRS complexes.

The delay in Second-degree AV block type I also occurs at the AV node. The delay, results in progressively lengthening PR intervals and then there will be a P wave that is not followed by a QRS complex. Following this event, the cycle starts over again with progressively lengthening PR intervals followed by a dropped QRS.
Each repeating Wenckebach series has a consistent P:QRS ratio with one less QRS than Ps in the series.

**Second-Degree AV Block Type II (Mobitz II)—Some P waves conducted through the AV node, others are blocked***
Second-degree AV block type II = PR interval stays the same, but there are dropped QRS complexes.

The delay in second-degree AV block type II occurs below the AV node at the Bundle of His or bundle branches. They usually produce a series of cycles consisting of one normal P-QRS-T cycle preceded by a series of paced P waves that fail to conduct through the AV node resulting in no QRS. This is a much more serious rhythm than Wenckebach and transcutaneous pacing is usually recommended.

![ECG Image](image1)

**Third Degree or Complete AV Block—No P waves conducted through the AV node***
Third Degree or Complete AV Block = no communication in the heart between SA and AV nodes

In third-degree or complete AV block, the impulse originating in the SA node is completely blocked. This block may occur at the AV node, bundle of His or bundle branches. In response to this situation, the heart may develop a secondary pacemaker (either junctional or ventricular) in order to stimulate the ventricles to contract. The location of this “escape pacemaker” will determine if the QRS complexes are wide or narrow. A junctional (narrow QRS complex) escape pacemaker rhythm may possibly be stable with a ventricular rate of more than 40 bpm. However, a ventricular (wide QRS complex) escape pacemaker rhythm is usually unstable with a heart rate of less than 40 bpm.

Often heart blocks occur due to cardiac damage following a MI. In high degree blocks (type II second and third-degree AV blocks), characterized by poor perfusion, Atropine 0.5 mg may be considered with a junctional escape rhythm, but for all complete AV blocks, prepare for transcutaneous pacing immediately.

![ECG Image](image2)
Tachycardia

There are 3 basic groups of tachycardias: sinus tachycardia, supraventricular tachycardia (including atrial tachycardia), and ventricular tachycardia. Fortunately, there is only one algorithm to treat all of them. The key factors are: STABLE vs. UNSTABLE and PULSE vs. NO PULSE. Additional factors are NARROW QRS vs. WIDE QRS and REGULAR vs. IRREGULAR.

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 100 to 150 bpm. The key to sinus tachycardia is that ALL components of a normal EKG are present, P waves, QRS complexes, and T waves. Sinus tachycardia generally starts and stops gradually. There is often a cause such as pain, fever, or agitation that can be identified and treated.

Supraventricular Tachycardia

Supraventricular Tachycardia (SVT) includes any rhythms that begin above the bundle branches. This includes rhythms that begin in the SA node, atrial tissue, or the AV junction. Since the rhythms arise from above the bundle branches, they are characterized by narrow QRS complexes. A supraventricular tachycardia is not the name of a specific arrhythmia. It is a term used to describe a category of regular arrhythmias that cannot be identified more accurately because they have indistinguishable P waves due to their very fast rate—usually greater than 150 bpm. The P waves are often indistinguishable because they run into the preceding T waves. The most common SVT rhythms are reentry tachycardia, atrial tachycardia, and junctional tachycardia.

Treatment Question #1 = Stable vs. Unstable
If Unstable, Cardiovert
If Stable, answer question #2
Treatment Question #2 = Regular vs. Irregular Rhythm
Regular (SVT or Junctional) = Vagal maneuvers & Adenosine
Irregular (A-Fib, A-Flutter, Multi-focal A-Tach) = Calcium Channel Blockers or Beta Blockers
A = A-fib, A-Flutter  
B = Beta Blockers  
C = Calcium Channel Blockers (usually used 1st to slow the rate)

**Atrial Tachycardia**  
The SA node and AV nodes are the primary pacemakers of the heart. However, there are other “automaticity foci” (sometimes called “ectopic” foci) that are potential pacemakers capable of taking over the pacemaker function in emergency situations. In atrial tachycardia, a very irritable automaticity focus may begin firing leading to a very rapid heart rate. This often begins suddenly. A rhythm that starts suddenly is termed “Paroxysmal”. Therefore, when an atrial tachycardia arises suddenly from a very irritable automaticity focus, it is termed paroxysmal supraventricular tachycardia (PSVT). The atrial tachycardia may be termed “ectopic” or “multifocal”, arising from one or more automaticity foci. Multifocal atrial tachycardia is a chaotic and irregular rhythm due to multiple foci, each with their own rates, stimulating the atria.

![Atrial Tachycardia ECG](image)

**Junctional Tachycardia**  
In junctional tachycardia, the AV junction becomes irritable and begins firing rapidly leading to a very rapid heart rate. If P waves are present (which they would not be in SVT), they would be inverted. The reason that P waves are inverted in junctional rhythms is because the impulse is being conducted backwards through the atria. This is more properly described as the atria being depolarized via retrograde conduction.

![Junctional Tachycardia ECG](image)

**Atrial Fibrillation**  
In atrial fibrillation (A-fib or AF), the atria quiver. This causes blood to pool in the heart where a blood clot can form. This is very critical in treatment of AF. The AF rhythm can be very fast when a significant number of the 400-600 impulses per minute trigger the AV node. Since the impulses being fired in AF are not at a regular rate, the rate of AF is usually irregular and can vary significantly in the ventricular rate generated. Calcium channel blockers are used to decrease the automaticity and slow the heart.

![Atrial Fibrillation ECG](image)
Atrial Flutter
In atrial flutter, the impulse circles around a large area of atrial tissue creating multiple P waves. If the AV node blocks the rapid impulses coming to it, and fires at a regular rate, the resulting rhythm will be regular. If the AV node blocks the rapid impulses coming to it at an irregular rate, the rhythm will be irregular.

Ventricular Tachycardia
Ventricular tachycardia (VT) occurs when an irritable automaticity focus in either ventricle begins firing. This ventricular focus fires at a tachycardia rate and overrides the higher pacemaker sites and takes over control of the heart. It is basically a run of premature ventricular complexes (PVCs). In PVCs or in VT, the ventricles fire prematurely and in an abnormal manner. Because the rhythm is originating in the ventricles, the QRS complex is wide.

Treatment Question #1 = Pulse vs. NO Pulse
If NO Pulse: Defibrillate

If there IS a Pulse, answer question #2
Treatment Question #2 = Stable vs. Unstable
If Unstable, Cardiovert
If Stable start with: Adenosine IF it is a regular pattern NOT irregular. May also use Amiodarone infusion of 150mg over 10 minutes.

Monomorphic Ventricular Tachycardia*
In monomorphic VT, the QRS complexes are of the same shape and amplitude. In ACLS, we ask the questions and follow the step above (in bold).
Polymorphic Ventricular Tachycardia
In polymorphic VT, the QRS complexes are of different shape and amplitude. In ACLS, we treat polymorphic VT the same as VF = defib.

Polymorphic Tachycardia = irregular size QRS

Torsades de Pointes
In Torsades de Pointes VT, the QRS complexes are of different shape and amplitude. The name means “twisting of points”, and in fact, what differentiates this rhythm from others is that it looks like a twisting party streamer with upward-pointing and then downward-pointing QRS complexes in an alternating pattern. This rhythm can be caused by low potassium or quinidine toxicity. ACLS recommendations include treatment with Magnesium.
ST-Elevation MI (STEMI)
A person who is having a MI will typically exhibit certain characteristics on their ECG. If necrosis or damage has occurred in the heart, a Q wave will be present. Once the damage has occurred, the Q wave will always be present. Therefore, based upon the presence of a Q wave, it is impossible to determine if the MI is acute or occurred in the past. However, the ST segment may become elevated during an acute MI and is usually one of the earliest signs of an MI. Therefore, evaluating the ST segment is a key part of the assessment of a person exhibiting signs of an MI. In order to determine if there is a ST-elevation, the isoelectric line must be evaluated. The isoelectric line is an imaginary line that goes from the beginning to the end of the QRS complex. If this line is level, the isoelectric line is normal. If, however, the ST-segment exits the complex prior to returning to the isoelectric line, the ST-segment is elevated. This indicates acute injury occurring in the heart. Sometimes an ST-segment depression may occur as well. Following the injury, the ST-segment will return to normal. However, if the event caused significant ischemia, a Q wave will develop. If the injury resulting from an MI is mild, the ST-segment may not be elevated, even though an MI has occurred. This is called a Non-ST-Elevation MI (NSTEMI). One other factor that can indicate ischemia during an acute MI is the inversion of the T wave.

Q = Infarction
ST elevation (or depression) = acute injury
T inversion = ischemia
**BLS Review**  
*(Primary Survey Approach to ECC)*

Performing high quality CPR is the most critical component for successful resuscitation of a patient in cardiac arrest. The goal of intervention for a patient in respiratory or cardiac arrest is to restore effective oxygenation, ventilation, and circulation. BLS priorities are a focus on circulation first in a pulseless patient. Any interruptions in chest compressions should be limited to 10 seconds.*

**Steps of Basic Life Support**
1. Make sure the scene is safe
2. Check for responsiveness (if not responsive, shout for help), normal breathing and pulse*
3. If no pulse, activate EMS, get the AED or call a code in the hospital
4. Start chest compressions
5. Defibrillate as soon as equipment is available

**Rescue Techniques – CAB and D**

**Circulation:** Check for a Pulse and normal respirations for 5 to 10 seconds.* If no pulse or normal breathing, activate EMS or a code and begin chest compressions.  

- The best location for performing a pulse check for an adult is the carotid artery of the neck.
- The compression to ventilation ratio is 30:2
- Proper compression technique requires the right rate (100 – 120/min)* and depth of compressions (at least two inches), as well as full chest recoil. Take your weight off your hands and allow the chest to come back to its normal position. Full chest recoil maximizes the return of blood to the heart after each compression.
- Compressions on the adult, two hands are placed in the center of the chest between the nipples on the lower half of the sternum.
- Rotation of 2-man CPR is every 2 minutes (5 cycles of 30:2) to prevent fatigue and improve quality of chest compressions.*
- Minimizing interruptions in chest compressions will increase the victim’s chance of survival.

**Airway:** After 30 compressions, or if the victim has a pulse but is not breathing effectively, open the Airway.

- The *head tilt-chin* lift is the best way to open unresponsive victim’s airway when you do NOT suspect cervical spine injury.
- The *jaw-thrust* with cervical spine immobilization is used for opening airway without tilting the head or moving the neck if a neck injury is suspected (this includes drowning victims). This can be a difficult technique, so after two unsuccessful attempts, use the *head tilt-chin lift*.

**Breathing:** After opening the airway, give 2 breaths, preferably using a bag valve mask (2 people) or a face mask (1 person)
♥ Only provide enough air to see the chest rise and fall. If using a bag mask, there is no need to compress the bag completely. With a good seal, you should only need to compress the bag about half way.

♥ Do not over-inflate the lungs. The positive pressure in the chest that is created by rescue breaths will decrease venous return to the heart. This limits the refilling of the heart, so it will reduce cardiac output created by subsequent chest compressions.

♥ Some victims may continue to demonstrate agonal or gasping breaths for several minutes after a cardiac arrest*, but these breaths are ineffective and will not maintain oxygenation. Perform rescue breathing.

♥ Rescue breathing is performed in someone who has a pulse, but is not breathing. Give 1 breath every 5 to 6 seconds (10 – 12 breaths per minute).*

**Defibrillation:** Attach the Automated External Defibrillator (AED).

♥ Immediate CPR and defibrillation within no more than 3 to 5 minutes gives an adult in sudden cardiac arrest the best chance of survival.

♥ The AED is used on an adult, child, or infant victim who has no pulse.

♥ Only use adult AED pads when performing defibrillation on an adult or child over the age of 8.

♥ Child or infant victim: Rescuer should use pediatric pads when available for infants or children up to age 8 yr. If not available use adult pads making sure that they do not touch each other.

♥ Place one pad on the victim’s upper right chest just below the collar bone and right of the sternum and the other pad on the left side and below the nipple, being careful that the pads do not touch. On a small child or infant, pads may be placed in an anterior/posterior position.

♥ Steps for defibrillation are: Power on the AED & attach pads, clear the victim and allow the AED to analyze the rhythm (make sure not to touch the victim during the analyze phase), clear the victim and deliver shock, if advised. If the AED does not promptly analyze the rhythm, resume compressions.

♥ Continue compressions while the AED is charging.*

♥ Make sure to clear the victim before shocking so that you and others helping do not get shocked.

♥ If no shock is advised, leave the AED pads on the victim and continue CPR, beginning with compressions.

♥ CPR alone may not save the life of a sudden cardiac arrest victim. Early defibrillation is needed.

**Foreign Body Airway Obstruction - Choking**

♥ The best way to relieve severe choking in a responsive adult or child - Perform abdominal thrusts.

♥ When a choking victim becomes unresponsive (adult, child, or infant) – Begin CPR. When you open the airway, look for and remove the object (if seen) before giving rescue breaths.
Analyze the Problem and Treat the Rhythm
(Secondary Approach to ECC)

Airway
Look for any signs of airway obstruction. Make sure that the airway is adequate and protected. Secure the airway with:

- **Nasopharyngeal Airway** – use in a semi-conscious person, select the appropriate size by comparing the outer circumference of the NPA with the inner aperture of the nares, the length should be determined by measuring from nose tip to the earlobe.
- **Oral pharyngeal Airway** – use in an unconscious person with no gag reflex, select the appropriate size by placing the tip of the OPA at the corner of the mouth and then the flange should be at the angle of the mandible*
- **Laryngeal Mask Airway (LMA)** – recommended if provider is inexperienced with ETT
- **King Airway or Combitube** – if provider inexperienced or has difficulty placing an ETT
- **Endotracheal Tube (ETT)** – usually the preferred airway
  -- The ETT is placed using a laryngoscope, or fiber optic device, looking for the triangular vocal cords, and placing the ETT through them.
  -- Intubation attempts should be limited to 30 seconds.
  -- Insertion of an advanced airway may be deferred until several minutes into the attempted resuscitation, since airway insertion requires an interruption in chest compressions for many seconds.
  -- ETT placement allows for the most effective PETCO$_2$ monitoring.

Continuous quantitative waveform capnography is recommended for intubated patients as the best way to confirm ET placement*, effectiveness of CPR*, and detecting ROSC (return of spontaneous circulation) based on end-tidal carbon dioxide (PETCO$_2$) values.

Breathing (oxygen)
- **Oxygen** = #1 drug – give oxygen as soon as it is available
  - Provide oxygen: Room air has 21% oxygen.
  - Nasal Cannula (1 to 6 L/min) = increases oxygen by 3% for each liter
  - Face Mask without reservoirs (6 to 10 L/min) = delivers 35 – 60% oxygen
  - Face Mask with reservoir also called a non-rebreathing mask (6 to 15 L/min) = ability to provide 100% oxygen with 10 to 15 L/min
  - Bag Valve Mask (BVM) = 100% with a reservoir. Administer a tidal volume of 500 – 600 ml (approximately ½ bag squeeze)
  - Always monitor a patient with pulse oximetry with the goal of achieving oxygen saturation readings between 94% and 99%.

Endotracheal (E.T.) Tube
- Confirm E.T. tube placement:
  - **Heart** – Chest rise
  - **Mist** in the tube
  - **Heart** – Auscultation of lungs for bilateral breath sounds
  - **Heart** – Auscultation of the gastric area—no gurgling should be heard that would indicate intubation of the esophagus
  - **Heart** – Continuous quantitative waveform capnography

Keep the airway clear with suctioning. Suctioning through an ETT should be done during withdrawal for no longer than 10 seconds.
PETCO₂< 10mm Hg will not achieve ROSC and indicates ineffective compressions.*
Return of ROSC will generally be indicated by a PETCO₂ of 35 to 45 mm Hg)

Once an advanced airway is in place, there is no need to pause chest compressions for ventilations. Provide 100 compressions per minute and 1 breath every 6 seconds which is equal to 10 ventilations per minute. This rate is important because each ventilation increases intrathoracic pressure and thereby decreases cardiac output and cerebral perfusion.* Additionally, it is important to avoid giving excessive volume with each breath because this can cause gastric distension, also increasing intrathoracic pressure, and impeding cardiac output.

Assess for a pneumothorax. If necessary, decompress.

**Circulation**
- Establish IV/IO access in order to: give fluids as necessary, give medications, flush drugs in with a 20 ml fluid bolus.
- Treat heart rate and rhythm
- Check blood pressure
- Send blood samples to lab
- Assess diastolic intra-arterial pressure—If the arterial relaxation pressure is <20mmHg, it is reasonable to try to improve chest compressions and vasopressor therapy.

**Differential Diagnosis – H’s and T’s**

“Thinking it Through” Unless the cause of an arrhythmia is correctly identified, it will be impossible to treat. A hypovolemic person in PEA will not be helped by all of the epinephrine in the world. H’s and T’s are essential to nearly every algorithm.

- ♥ Hypovolemia – give fluids
- ♥ Hypoxia – give oxygen, check E.T. tube
- ♥ Hydrogen ion (acidosis) – sodium bicarbonate
- ♥ Hypo-/Hyperkalemia – potassium or sodium bicarb
- ♥ Hypothermia – rewarm
- ♥ Toxins – Drug overdose = give Narcan or other antidote
- ♥ Tamponade, cardiac - pericardiocentesis
- ♥ Tension pneumothorax – needle decompression
- ♥ Thrombosis, pulmonary - ? surgeon
- ♥ Thrombosis, coronary - PCI

**Disability**
- Mental status, pupil response
- Glasgow Coma Scale
- Stroke screen as indicated
**Team Work**

FIRST OF ALL, TRY TO PREVENT A CARDIAC ARREST. CALL THE RRT (Rapid Response Team) or MET (Medical Emergency Team). They can identify and treat early clinical deterioration.*

If there is a code:
Each team member in resuscitation must be proficient in skills according to his or her scope of practice. If assigned a task that is beyond the licensed scope of practice, ask for a new task or role.*

There are several elements of team dynamics that will make any code run more smoothly. These elements include:
1. Closed loop communication*
2. Delivery of clear messages
3. Having clear roles and responsibilities
4. Knowing one’s limitations
5. Knowledge sharing
6. Constructive Interventions*
7. Reevaluation and summarizing
8. Mutual respect

Some key team roles are:
1. Team Leader
2. Airway
3. Compressor
4. IV/IO
5. Recorder/Timer
6. Monitor

In an emergency situation, the Team Leader should always clearly delegate roles* and insure that these things are addressed first:
1. High quality CPR, if no pulse
2. Oxygen, as necessary, determined by respiratory status and pulse oximetry monitoring
3. Monitor for cardiac rhythm
4. IV/IO access, at least one IV, preferably two. Peripheral IV is the preferred method of access. The antecubital vein is the recommended first choice site. If unable to obtain IV access after two attempts, try to obtain IO access.
5. Someone to record the times and events and monitor two minute intervals of CPR as needed.
Algorithms to Treat the Rhythm

Pulseless Arrest
Pulseless Arrest includes:
1. Ventricular Fibrillation and Pulseless Ventricular Tachycardia
2. Asystole and Pulseless Electrical Activity

V-Fib and Pulseless VT ARE shockable*
Asystole and PEA ARE NOT shockable

IF SHOCKABLE (V-fib and Pulseless VT):

![Graph of the ECG waveform showing ventricular fibrillation.]

Defibrillation is usually performed using biphasic technology. On rare occasions, the older monophasic technology may be utilized, but it is not as effective. The initial shock should be:

Biphasic = 120 J to 200 J initial shock dose, if unknown, use maximum available.
Second and subsequent doses should be equivalent, and higher doses may be considered, based upon manufacturer’s protocol.
Monophasic = 360 J

This depends upon the manufacturer’s recommendations and hospital protocol.

♥ Steps for defibrillation:
1. When the AED or defibrillator arrives, turn it on
2. Position appropriate pads or electrodes (apply conductive paste if using paddles). Hands-free pads are preferred because they allow for a more rapid defibrillation.
3. Analyze the rhythm (do not touch the victim during this phase) if the rhythm is V-Fib or pulseless VT (or if the AED recommends a shock), prepare to shock
4. Prepare to shock by selecting the appropriate # of Joules and selecting defibrillate mode
5. Press the charge button—announce that you are doing this—continue CPR while charging, thereby providing quality compressions immediately before a defibrillation attempt
6. Clear: I’m clear (you are not touching the patient or bed), You’re clear (includes making sure that the oxygen is away from the patient), Everybody’s clear (no one is touching patient, or bed)
7. Press the shock button and wait for shock discharge

♥ Immediately following the shock, resume CPR starting with chest compressions.*
♥ Perform CPR for 2 minutes
♥ After 2 minutes of CPR, stop compressions just long enough to check the rhythm and check for a pulse
♥ If another shock is needed, prepare to shock and give a medication as close to the shock as possible.
♥ Repeat this sequence until the rhythm is not shockable

Reasons for CPR immediately after the shock:
♥ If the first shock fails, CPR will circulate the blood and bring more oxygen to the heart, making a subsequent shock more likely to be successful.
♥ Even when a shock eliminates VF, it often takes several minutes for a normal heart rhythm to return and more time for the heart to create blood flow. Chest compressions can deliver oxygen and sources of energy to the heart, increasing the likelihood that the heart will be able to effectively pump blood after the shock.

Drug delivery should not interrupt CPR. Rescuers should prepare the next drug dose before it is time for the next rhythm check so that the drug can be administered as soon as possible after the rhythm check. The timing of the drug is less important than minimizing interruptions in chest compressions.

A drug may be administered:
- During the CPR, preferably immediately after a shock
- While the defibrillator is charging

Medication Sequence:
- **Epinephrine 1 mg IV/IO 1:10,000 every 3 to 5 minutes**
- For refractory VF, if Epinephrine is not working:
  - **Amiodarone 300 mg IV/IO** push followed by
  - **Amiodarone 150 mg IV/IO** push in 3 to 5 minutes

If VF cardiac arrest has been present for several minutes prior to CPR, it is reasonable to consider performing 2 minutes of CPR before the shock. A period of CPR before shock delivery will provide some blood flow to the heart, delivering oxygen and sources of energy to the heart muscle. This will make a shock more likely to eliminate the VF and will make the heart more likely to resume an effective rhythm and effective pumping function after shock delivery.

**IF NOT SHOCKABLE (Asystole and PEA):**

While checking the rhythm, always check a pulse. If no pulse:
1. **CPR**
2. **Epinephrine 1 mg of 1:10,000 IV/IO every 3 to 5 minutes**
3. **Possible causes** = H’s & T’s, hypovolemia is most common and often easiest to treat
Note: Asystole should not be called “flat line”. Flat line indicates a lead is off or the gain and sensitivity need to be adjusted.

If a patient is in sustained asystole for 15 minutes, it may be reasonable to consult the family and consider calling the code.

**Bradycardia**

**Assess respiratory effort:** Adequate oxygenation is essential, since hypoxia can be the cause of the bradycardia.

**Atropine 0.5 mg IV** repeated every 3 to 5 minutes up to a maximum of 3mg is the drug of choice. Have the transcutaneous pacemaker ready.

Chronotropic drug infusions are recommended as an alternative to pacing for treatment of adults with symptomatic and unstable bradycardia

- **Dopamine IV Infusion:** 2 – 10 mcg/kg per minute
- **Epinephrine IV Infusion:** 2 – 10 mcg per minute

Though Atropine is now recommended for any symptomatic bradycardia, Atropine will probably not work in high-degree heart blocks (type II second-degree block or third-degree AV block). They will almost certainly require **transcutaneous pacing**.

**Steps for transcutaneous pacing:**

1. Consider sedation
2. Attach pacing electrodes to the patient as shown on package (AP position preferred)
3. Turn pacer on
4. Set the pacing rate—start at 60 bpm and then can adjust higher if needed
5. Look for electrical capture on the strip (turn up mA dial until capture is achieved—widening QRS & broad T waves)
6. Assess mechanical capture by assessing right arm or right femoral pulses
7. Once capture is achieved, set pacing at about 2 mA higher than the threshold of initial capture.
Tachycardia with a Pulse

#1 Question = STABLE vs. UNSTABLE (Unstable signs include altered mental status, ongoing chest pain, hypotension or other signs of shock)*
STABLE = Medication and seek expert consultation

Narrow QRS Regular Rhythm (SVT)

1. Try Vagal maneuvers
2. Adenosine: First dose 6 mg, Second dose 12 mg RAPID IVP*
   Note: A brief period of asystole may follow the injection

Narrow QRS Irregular Rhythm (A-fib, A-flutter, etc.) or does not convert with Adenosine

Control the rate with Calcium Channel Blockers (Diltiazem) or β-Blockers
A = A-fib, A-flutter
B = β-Blockers
C = Calcium Channel Blockers
If a person in A-fib does not convert with Calcium Channel Blockers, cardioversion may be necessary. Since the atria are not pumping effectively, blood clots may form in the atria due to blood pooling. If a person has been in AF for more than 48 hours, anticoagulant therapy is indicated for several days prior to cardioversion. Always seek expert consultation from the cardiologist.

Wide QRS (VT with pulse)

Adenosine: First dose 6 mg, Second dose 12 mg RAPID IVP, ONLY if Regular and monomorphic (as a diagnostic tool)
Amiodarone 150 mg IV over 10 minutes, other antiarrythmic alternatives are Procainamide 20-50 mg/min, and Sotalol 100 mg over 5 min
May need synchronized cardioversion
Polymorphic VT—defibrillate

Wide QRS (torsades de pointes)

Magnesium load with 1-2 g IV over 5-60 minutes, then infusion

**UNSTABLE (WITH PULSE) = SYNCHRONIZED CARDIOVERSION**

Prepare for IMMEDIATE cardioversion. While preparing, you may try Adenosine 6 mg if there is time. Also, sedate the patient if possible.

**Initial recommended doses of synchronized cardioversion shocks:**
- Narrow regular: 50 – 100 J
- Narrow irregular: 120 – 200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

♥ Steps for cardioversion:
1. Consider sedation
2. Turn on defibrillator
3. Apply electrode pads to the patient
4. Press “SYNC” mode button
5. Look for monitor markers on R waves indicating sync mode
6. Select appropriate energy level
7. Press the charge button—announce that you are doing this
8. Clear: I’m clear, You’re clear—includes making sure that the oxygen is away from the patient, Everybody’s clear
9. Press the shock button and wait for shock discharge (this may take a few seconds while the machine looks for R waves and determines where to sync the shock)
10. Analyze the rhythm again. If still in tachycardia, increase the joules and try again.

♥ **Note:** Reset the sync mode after each synchronized cardioversion because most defibrillators default back to unsynchronized mode. Also, if the shock fails to discharge, check to see if the patient still has a pulse. No shock will be delivered if there is not a pulse, since the pulse is required to indicate where the sync function should deliver the shock.
**Stroke**

Signs and symptoms of a stroke include:
- Sudden weakness or numbness of the face, arm, or leg on one side of the body
- Sudden dimness or loss of vision, particularly in one eye
- Loss of speech, or trouble talking or understanding speech
- Sudden, severe headache with no apparent cause
- Unexplained dizziness, unsteadiness or sudden falls, especially along with any of the previous symptoms

EMS must be notified immediately.
There is a 3 hour window from onset of symptoms during which fibrinolytic therapy will be most effective.* This can be increased to 4.5 hours in some patients.

Within 3 hours, the following must be completed:
1. Support ABCs, give oxygen as needed
2. Evaluate with pre-hospital assessments: Cincinnati Pre-hospital Stroke Scale (facial droop, arm drift, abnormal speech) or Los Angeles Pre-hospital Stroke Screen
3. Check blood sugar (severe hypo- or hyperglycemia can mimic a stroke)
4. Establish time that the stroke occurred
5. Alert the hospital and transport to a stroke unit*
6. History, Physical, Neurological assessment
7. Non-contrast CT scan of the head—must be read by a radiologist*
8. Monitor and treat BP (Systolic BP must be <185, Diastolic <110 to be eligible for fibrinolytic therapy)
9. If all inclusion and exclusion criteria are acceptable, fibrinolytic therapy may be started

**Acute Coronary Syndromes (ACS)**

The primary symptom of an acute MI may be chest pain radiating to the jaw or left arm. However, various other milder symptoms may also signal varying degrees of coronary artery occlusion and ischemia resulting from atherosclerosis. Of particular concern are women, who tend to have more vague signs of a MI. This is further complicated when a woman has a history of diabetes. Diabetes can alter the pain perception due to neuropathy. Therefore, a diabetic woman can have ischemic symptoms that are overlooked because they are vague and not the characteristic chest pain. Any time there is any possibility of ACS, a 12-lead ECG should be obtained quickly to look for ST segment changes.*

Acute coronary syndromes can be divided into three groups:
1. Unstable angina
2. non-ST-segment elevation MI (NSTEMI)
3. ST-segment elevation MI (STEMI)
Any of these may lead to sudden cardiac death.

**Unstable Angina**

In an artery that is partially occluded by a thrombus, there may be intermittent episodes of ischemia. The symptoms may be prolonged and may even occur at rest. These symptoms are better treated with aspirin, not fibrinolytic therapy, because fibrinolytics
may cause the thrombus to be released and create full occlusion and subsequent necrosis.

Non-ST-segment Elevation MI (NSTEMI)
A thrombus, that intermittently occludes an artery, may cause myocardial necrosis producing a NSTEMI. This necrosis is usually in a smaller area and not as significant as necrosis that would cause ST-segment elevation. A Q wave may not develop following a NSTEMI.

ST-segment Elevation MI (STEMI)
When a thrombus occludes the coronary vessel for a prolonged period, the resulting necrosis usually produces a STEMI. A STEMI represents significant myocardial damage and usually results in a Q wave developing. This condition is best treated with heparin, fibrinolytic therapy (e.g., Alteplase, Retreplase, Streptokinase, Tenecteplase) and a trip to the cardiac cath lab.

Treatment
1. The first treatment consideration is to support the ABCs.
2. If a person is unresponsive, prepare to defibrillate since VF is often the rhythm that follows a significant MI.
3. If a person is stable, remember MONA.
   M = Morphine (#4) – Only if Nitro doesn't relieve chest pain. Use with caution in NSTEMI.
   O = Oxygen (#1) – 4 liters per NC
   N = Nitroglycerin (#3) – 3 doses SL if NO Viagra or other phosphodiesterase inhibitor use within 72 hours & BP is O.K.
   A = Aspirin (#2) – 160 to 325 mg*, to reduce platelet aggregation, may chew NOT in that order, however. See numbers above.
4. Get a 12 lead ECG. Look for ST-segment elevation, Q wave or inverted T wave.
5. Draw blood for cardiac markers
6. Monitor BP—Avoid hypotension with Nitro & Morphine, evaluate hypertension that may exclude fibrinolytic therapy BP > 185 systolic or > 110 diastolic
7. The goal is to start fibrinolytic therapy within 30 minutes (Door-to-needle), but it may be started up to 12 hours following the event
8. The goal is to perform percutaneous coronary intervention (PCI) within 90 minutes (Door-to-balloon inflation),* but this also may be considered up to 12 hours following the MI

Post-Resusitation Care
Patients display a wide spectrum of responses to resuscitation. Following ROSC (return of spontaneous circulation), patients may respond by becoming awake and alert with adequate spontaneous respirations and hemodynamic stability. Others will remain comatose with an unstable circulation and no spontaneous breathing. They may require 24 to 48 hours of invasive hemodynamic monitoring for optimal management after resuscitation.

Your immediate goal is to provide cardio-respiratory support to optimize ventilation, oxygenation and perfusion, particularly to the brain. This is accomplished by assessing and treating the primary and secondary ABCD surveys:
A **Airway** = Secure the airway and confirm tracheal tube placement with primary assessments, including waveform capnography and secondary assessment with a chest x-ray.

B **Breathing** = Avoid excessive ventilation (do not ventilate too fast or too much) which can potentially cause oxygen toxicity. Start at 10 breaths/min and titrate to target PETCO₂ of 35-40 mmHg. When feasible, titrate FIO₂ to minimum necessary to achieve SpO₂ between 94% and 99%.*

C **Circulation** = Treat hypotension (systolic BP < 90mm Hg)*, administer 1 to 2 liters of normal saline or lactated Ringer’s IV and monitor urine output to reflect tissue perfusion. Epinephrine, Dopamine, or Norepinephrine IV infusions can also be used to treat hypotension.

- Epinephrine 0.1 – 0.5 mcg/kg per minute (in a 70 kg adult: 7-35 mcg per minute)
- Dopamine 5 – 10 mcg/kg per minute
- Norepinephrine 0.1 – 0.5 mcg/kg per minute

D **Differential Diagnosis** = Search for specific cause for the arrest. Review the chest x-ray, 12-lead ECG, history, and serum electrolytes.

The following problems may develop:

- Hostile environment for the brain – control seizures that increase cerebral oxygen requirements. Elevate the head 30 degrees to decrease intracranial pressure.
- Hypotension – Even mild hypotension can impair recovery of cerebral function. Administer fluids.
- Recurrent VF/Pulseless VT – consider administration of an infusion of the antiarrhythmic used during resuscitation.
- Post-resuscitation tachycardia – rapid SVTs that may develop in the immediate post-resuscitation period are best treated by leaving them alone.
- Post-resuscitation bradycardia – poor ventilation and oxygenation play a major role in post-resuscitation bradycardia.
- Post-resuscitation PVCs – improved oxygenation over time may eliminate the ectopic beats.

Post-resuscitation care includes support of the myocardial function with anticipation that myocardial “stunning” may be present, requiring vasoactive support. A healthy brain is the primary goal of cerebral and cardiopulmonary resuscitation. This may be accomplished by Targeted Temperature Management (TTM).*

- TTM—Unconscious adult patients with ROSC should be cooled to 32 to 36 degrees C* for at least 24 hours* when the initial rhythm was VF. Similar therapy may be beneficial for patients with non-VF arrest. In multiple randomized clinical trials, TTM resulted in improved survival and neurologic outcome in adults who remained comatose after initial resuscitation from out-of-hospital VF cardiac arrest.

To ensure the success of post-cardiac arrest care, healthcare providers must:

- Optimize the patient's hemodynamic and ventilation status
- Initiate TTM
- Provide immediate coronary reperfusion with PCI*
- Institute glycemic control
- Provide neurologic care and prognostication and other structured interventions
10 Required Scenarios
(With Key Points)

1. Respiratory Arrest – CAB, if pulse, support respiration, intubate if necessary
2. VF treated with CPR and AED – CAB, defib. as soon as AED arrives, follow AED prompts
3. Ventricular Fibrillation/Pulseless VT – CAB, defibrillate, resume CPR (2 min.), give a drug, draw a drug up, repeat steps. Start with Epi. q 3 – 5 min., then Amiodarone
5. Asystole – same as PEA
6. Acute Coronary Syndromes – MONA, STEMI or not, fibrinolytic therapy, cath lab
7. Symptomatic Bradycardia – Atropine, 2nd or 3rd degree blocks = transcutaneous pacing, chronotropic drug infusion
8. Unstable Tachycardia – Cardiovert
9. Stable Tachycardia – meds:
   • narrow complex/regular = Vagal, then Adenosine
   • narrow/irregular = Calcium channel blockers or Beta blockers
   • wide complex/regular = Adenosine, Amiodarone
   • wide complex/irregular = Consult cardiologist—depends upon rhythm
10. Acute Stroke – check glucose, non-contrast CT of head read by radiologist, fibrinolytics within 3 hrs if BP O.K.

“*” Used throughout the course outline indicates that there is a test question related to the noted material.

Credits: