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Contact Hours: **2**

Emergency Cardiac Care Guidelines

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COURSE OBJECTIVE: The purpose of this course is to enable healthcare professionals to understand current evidence-based guidelines for emergency cardiac care for adults.

LEARNING OBJECTIVES
Upon completion of this course, you will be able to:

- Describe the practice skills that comprise high-quality cardiopulmonary resuscitation (CPR), also known as Basic Life Support (BLS), in accordance with American Heart Association (AHA) guidelines.
- Describe the appropriate steps of automated electronic defibrillator (AED) usage and the situations when AEDs are necessary.
- List the recommended steps to assist an adult victim with mild and severe airway obstruction.
- Discuss the expansion of the BLS survey into the Advanced Cardiac Life Support (ACLS) survey.
- Differentiate the appropriate care of a patient experiencing a pulseless arrest with a shockable versus a nonshockable dysrhythmia.
- Discuss the care of a patient experiencing a heart rate disturbance.
- Explain the importance of early recognition, assessment, and treatment of acute coronary syndromes.
- Identify modifications of emergency cardiac care techniques appropriate for untrained rescuers.

The *2015 American Heart Association Guidelines Update for Cardiopulmonary Resuscitation (CPR) and Emergency Cardiovascular Care (ECC)* was released in October 2015. These guidelines include updates from the previous editions regarding the assessment sequence, depth and rate of compressions, chest recoil, and avoiding excessive ventilation while performing cardiopulmonary resuscitation (Neumar et al., 2015). High-quality compressions and the use of automated electronic defibrillators remain the framework for successful resuscitation. The basic life support sequence of C-A-B is reinforced, as is achieving full chest recoil and reducing over-inflation and excessive ventilation.

BASIC LIFE SUPPORT (BLS)

Early recognition and treatment are the tenets of good emergency cardiovascular care. Specifically, early initiation of excellent CPR and early defibrillation are associated with significantly improved survival rates (Posner, 2015).

Initial Steps

Prior to initiating BLS care on anyone, the "scene" should be safe. It is important to note one's location in terms of proximity to any potential danger (vehicles, power lines, crime, etc.). For trained healthcare professionals, it is advisable to perform multiple tasks as reasonable, such as using a mobile device to request emergency response while checking the patient's pulse at the location of the carotid artery and whether breathing is present. This can reduce the time until the first compression.

If there are multiple providers, concern for head and neck injury can be addressed. If there are limited numbers of rescuers, it is not advisable to spend time in attempting to protect cervical spine injury (Kleinman et al., 2015).

Pulse Check and Compressions

After activating the emergency response system, the rescuer establishes unresponsiveness using the "shake and shout" method. Once unresponsiveness is established, the rescuer checks for a carotid pulse by using two fingers to find the trachea and sliding his or her fingers into the groove on either side. The recommendation is to feel for a pulse for no longer than ten seconds.

If pulse is absent, chest compressions are begun. (For the situation with a pulse present but no breathing, see below in this course.) For untrained responders, it is preferable to not perform a pulse check but to begin chest compressions immediately (Kleinman et al., 2015).

The importance of delivering high-quality compressions cannot be overemphasized. The compressions should be hard, fast, and deep while also allowing for full recoil of the chest between compressions.

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If the hands remain on the chest and create even minimal continuous pressure, full chest recoil is not achieved. This results in residual intrathoracic pressure, impedes venous return, and reduces coronary perfusion pressure and myocardial blood flow. This could potentially limit positive outcomes during resuscitation (Kleinman et al., 2015). However, since it is also important to maintain correct hand placement, completely removing the hands off the chest is not advised. Interruptions are kept to an absolute minimum.

Proper **hand position** is necessary for effective compressions, and hand placement can affect outcomes during resuscitation. For an adult patient, the rescuer places the heel of one hand on the center of the patient's chest on the lower half of the sternum and the other hand on top, with the fingers linked. The rescuer's body is positioned directly over the patient's chest, with the arms straight and elbows locked, thereby allowing one's body weight to compress (Kleinman et al., 2015).

Compressions are delivered at a **rate** of 100 to 120 per minute. Higher numbers of chest compressions with fewer interruptions yield better chances for a return of spontaneous circulation (ROSC) and survival with good neurologic function (Kleinman et al., 2015).

The *2015 American Heart Association Guidelines* reinforce the **depth** of compressions to at least 2 inches for the adult patient. (Studies are inconclusive regarding deeper compressions.) It is important to compress deep enough to create intrathoracic pressure to pump blood from the heart. It is also vital to limit the amount of interruptions with chest compressions. Between each compression, the chest should fully recoil to its natural state to promote venous return of blood flow through the lungs and heart.

MAINTAINING EFFECTIVE COMPRESSIONS

Delivery of chest compressions can be exhausting, and the emphasis on fast, hard compressions makes it even more so. Research has shown that compressions can become ineffective after only one minute (Posner, 2015). After becoming fatigued, rescuers may mistakenly believe they are still performing effective compressions. In order to get the best and most effective compressions, the American Heart Association recommends that personnel be rotated every two minutes or at the end of every fifth cycle (AHA, 2015).

Airway

The **head-tilt-chin-lift** is the preferred method of opening the airway of an adult patient. The head-tilt-chin-lift is performed by placing one hand on the patient's forehead, with the other hand on the chin and tilting the head back. It is important to resist the temptation to lift up on the neck instead of the chin; this can cause hyperextension of the neck and partially obstruct the airway.

If trauma is involved or suspected, the use of the jaw-thrust method is recommended. However, if ventilations are ineffective using the jaw-thrust maneuver, personnel are limited, or readjustment still does not provide an effective airway, then the head-tilt-chin-lift may be used.

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If the number of personnel allow, untrained rescuers may place one hand on either side of the head to limit movement in a suspected head or neck injury (Kleinman et al., 2015).

Breathing

After the initial 30 compressions are delivered, the airway is opened and the rescuer delivers two breaths. (If two providers are involved in the resuscitation, one maintains chest compressions while the second prepares the airway to give breaths immediately following the 30th compression.) Each breath should be delivered over one second with any delivery method available, such as mouth-to-mask or bag-mask device (with or without oxygen). A good breath will cause the chest to rise. If chest rise is not seen, the airway should be adjusted and ventilation reattempted.

After an advanced airway is placed (such as an endotracheal tube or laryngeal airway), the compressions should not be paused, and ventilations are delivered at a rate of 1 breath every 6 seconds (about 10 breaths per minute) (Kleinman et al., 2015).

In the case of an apneic (respiratory arrest) patient with a detectable pulse, trained and untrained and rescuers alike may consider an opioid overdose as a cause. The *2015 AHA Guidelines* state that for a victim of opioid overdose, it is acceptable to give either intramuscular or intranasal naloxone along with standard BLS care. The guidelines suggest either 2 mg intranasally or 0.4 mg intramuscularly (Kleinman et al., 2015; Lavonas et al., 2015).

COMPRESSION-TO-VENTILATION RATIO

Every time there is a pause in compressions, cerebral perfusion pressure falls. Once compressions begin again, it will increase, but slowly. In order to optimize perfusion of brain, interruptions in compressions should be kept to an absolute minimum and limited to less than 10 seconds. Compressions should be interrupted for ventilations only in the absence of an advanced airway. Each breath should be delivered over one second, and CPR should be resumed quickly (Kleinman et al., 2015).

The recommended compression-to-ventilation ratio for adult patients is 30:2, with a brief pause to deliver the breaths. Five cycles of the 30:2 compressions/ventilations should be delivered, which should take approximately two minutes. All BLS and ACLS interventions are completed around the two minutes of CPR, which helps keep pauses in compressions to an absolute minimum. With the increased rate and depth of compressions, it may be advisable to switch providers prior to completing a two-minute cycle. With organization, compressions can continue seamlessly.

Early in resuscitation, circulation is more important than ventilation, and the more chest compressions a patient receives, the better myocardial perfusion will be maintained. For the untrained rescuer it may be advisable to perform just chest compressions, or “hands-only CPR,” until advanced providers arrive. It has been shown that bystanders without training are more willing to participate in resuscitative measures if they are at limited risk (Kleinman et al., 2015).

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THREE PHASES OF CARDIOCEREBRAL RESUSCITATION

Studies into the physiology of cardiac arrest due to ventricular fibrillation have divided the event into three time phases, which have also been termed the "three phases of cardiocerebral resuscitation." Cardiocerebral resuscitation, in addition to uninterrupted chest compressions and early defibrillation, takes into account delayed intubation and the avoidance of overventilation.

The first four minutes are the **electrical phase.** The most important action during this time is defibrillation, and it is during this time that successful resuscitation rates are the highest. High-quality CPR administration while a defibrillator is being obtained improves survival, but if cardiac arrest is left untreated in the first phase, it moves into the circulatory, or metabolic, phase.

The next six minutes of cardiovascular arrest constitute the **circulatory phase**, in which the energy stores in the myocardium fail. At this time both defibrillation and chest compression are essential.

The third phase, defined as greater than 10 minutes of pulselessness, is called the **metabolic phase**. In this phase systemic ischemia, reperfusion injury, and endotoxin release ("sepsis-like syndrome") occurs. The effectiveness of resuscitation and overall prognosis is very poor once a patient has entered into this phase. If the patient does not obtain a perfusing rhythm during this phase, the patient generally does not survive.

If considering the three-phase model, it is important to remember that the above information is theoretical in nature and has no bearing on an actual arrest scenario. It should not determine how resuscitative measures proceed. High-quality chest compressions and early defibrillation should remain the essential framework of successful resuscitation.

Source: Stapczynski, 2014.

Automated External Defibrillators (AED)

When a victim collapses due to a sudden cardiac event, high-quality compressions should be initiated while an AED is retrieved.

In a scenario with only one rescuer, after calling for help, the rescuer gets the AED to the patient and begins CPR. The AED is turned on and used according to its instructions. The AED should be applied immediately without interrupting compressions until it is ready to analyze. Electrode pads are placed by peeling away the backing and placing one pad on the upper-right side of the patient's bare chest (to the right of the sternum directly below the clavicle) and the other to the left of the left nipple a few inches below the left armpit.

During the analysis of the patient's cardiac rhythm, the AED will ask the rescuer to pause compressions briefly. The AED will analyze the rhythm and determine if a shock is appropriate. If needed, a shock will be delivered and chest compressions should immediately follow. There is

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no longer a specified time to wait until the defibrillator is used. It should be set up and turned on as soon as it is available (Kleinman et al., 2015).

DELIVERING A SHOCK

Compressions are continued until a rescuer is ready to deliver a shock, with no more than 10 seconds from the time of the last compression to shock delivery for the most effective outcome. Chest compressions are resumed immediately after the shock, without taking time for a pulse check. Even if the patient has an organized rhythm, the compressions will not harm the patient (Kleinman et al., 2015). If a rhythm is present, cardiac output is still too low to provide adequate perfusion and the patient benefits from the assistance (Posner, 2015).

Biphasic defibrillators are preferred due to the lower energy doses needed for defibrillation. If the electrical output is unknown, the highest available setting is used (Posner, 2015). This timesaving technique has the potential to decrease interruptions in CPR and is supported by a study of out-of-hospital cardiac arrest patients treated with either low or high doses.

When using an AED, the rescuer should remember these factors:

- Some **medication patches** may be located in the vicinity of AED pad placement. If this is the case, carefully remove such patches and place them in an area that will not have contact with other responders.
- Some people have **electronic pacemakers** or automated internal cardioverting devices (AICD). Often, they are placed on the left side beneath the clavicle and are usually discernible under the skin. Infrequently, these devices may be placed on the right side below the clavicle or on the left side below the ribs. These also may be in the way of the AED pads. If this is the case, place the pads no closer in proximity to the devices than two inches.
- On some skin (such as wet or very hairy), AED **pads may not adhere** well. If the skin is wet, dry off briefly. If this is a drowning victim, remove the patient from the water and ensure there is no puddle beneath them. They do not have to be completely dry for the AED to be used successfully. If hair is impeding the adherence, there are a few options. The hair may be shaved off if a razor is immediately available. Or, if spare pads are available, the first set may be placed onto the skin and rapidly removed to remove some hair. Then place the second set and press firmly.

CASE

Susie, a med-surg nurse, was walking back into the hospital after her lunch break and saw her 60-year-old patient, Sam, smoking in the parking lot. As she got closer to him, Sam collapsed on the sidewalk. Susie ran up to Sam, shook him, and shouted, “Are you okay?” Sam did not reply. Susie looked for signs of breathing but did not see any. She quickly scanned the area and saw a bystander and yelled for her to go into the hospital and bring back help.

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Susie checked Sam's carotid pulse but did not find one within 10 seconds. She immediately started compressions hard and fast at the rate of 100 to 120 per minute and at a depth of at least two inches. After 30 compressions she used the head-tilt-chin-lift maneuver and gave two breaths, watching for Sam's chest to rise each time. She then resumed giving compressions and ventilations at a rate of 30:2 until help arrived.

The bystander returned from inside the hospital with a nurse, who carried an AED. The nurse said that the code team had been called. In the meantime, the nurse opened up the AED, turned it on, followed the instructions, and applied the patches. While she was doing this, Susie continued giving compressions and ventilations. The AED announced it was analyzing, and Susie stood clear while a shock was delivered. Susie quickly resumed compressions and could see the code team running to them.

(continues)

Foreign Body Airway Obstruction (FBAO)

A number of arrest situations begin as airway obstructions. The first step in the FBAO sequence is to distinguish between partial or complete airway obstructions. Intervention is indicated only if the obstruction is completely occluding the airway. The signs of complete obstruction include respiratory difficulty, no air exchange, cyanosis, or an inability to cough or talk. The AHA recommends that only one question be asked, and that question is **"Are you choking?"**

If the victim nods yes, the rescuer positions him- or herself behind the patient and wraps the arms around the patient while inserting one foot in between the patient's for stability. The rescuer then makes a fist with one hand and places the thumb (flat side) against the patient's abdomen between the naval and the xiphoid process, then places the other hand on top of the fist. Using both hands with an upward and inward motion, abdominal thrusts are delivered until the foreign body has been dislodged or the patient becomes unresponsive. For obese patients or patients in the last stages of pregnancy, chest thrusts instead of abdominal thrusts are indicated (AHA, 2015).

If the patient becomes unresponsive, a lone rescuer should activate an emergency response before proceeding any further. In the case of multiple rescuers, emergency response is activated as soon as the patient is determined to be in distress.

Management of an unresponsive patient with an airway obstruction is not unlike that of a pulseless arrest situation. Studies show that chest compressions can produce intrathoracic pressures as high or higher than abdominal thrusts. Therefore, CPR is performed as it would be for a pulseless victim, with one exception: as the airway is opened, the rescuer looks inside the patient's mouth for any foreign body. If a foreign body is seen, it should be removed, taking care not to push it farther down the trachea. Blind finger sweeps are not recommended because of the potential damage to the mouth or throat (AHA, 2015).

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ADVANCED CARDIOVASCULAR LIFE SUPPORT (ACLS)

Prior to initiating advanced life saving measures, excellent chest compressions and early defibrillation remain the most important components of resuscitation. Without these simple steps, outcomes are significantly reduced.

Organized team management is also an important facet in advanced measures. Due to the low occurrence of cardiac arrest in situations other than critical-care units, it is often a confusing event. It is therefore important to have a team approach that incorporates collective information and reasoning, with a defined team leader (Posner, 2015).

ACLS Survey

The BLS (primary) survey has already been completed if CPR is being performed. This is the C-A-B plus defibrillation described above in "Basic Life Support."

- **C**irculation. Does the patient have a definite pulse? If not, start compressions.
- **A**irway. Is it open? If not, open it.
- **B**reathing. Is the patient breathing adequately? If not, give rescue breaths.
- **D**efibrillation. Attach an AED or manual defibrillator as soon as it is available and deliver a shock when appropriate
  (Kleinman et al., 2015)

The ACLS survey expands on the BLS survey.

- **Airway.** Use a bag-mask to provide respirations. Use a supraglottic airway (laryngeal mask airway, laryngeal tube, Combitube), if available. You may consider the placement of an advanced airway if rescue breathing is not adequate. With any device, placement should be deferred unless it can be performed without disrupting chest compressions. If an advanced airway is inserted, it is important to use waveform capnography, if available, to ensure proper placement of the device. If continuous waveform capnography is unavailable, a CO_2 detector, esophageal airway detector, or ultrasound may be used.
- **Breathing.** Support ventilation as needed and give high-flow oxygen as available. Airway adjuncts, such as nasopharyngeal or oropharyngeal airways, are appropriate to assist with ventilations. When using advanced airways, the recommendation is for 10 breaths per minute (every 6 seconds) with continuous chest compressions while avoiding excessive ventilation.
- **Circulation.** Ensure excellent CPR quality, attach monitor/defibrillator, provide defibrillation/cardioversion, establish IV/IO access, and give appropriate medications (see "Medications" later in this course).

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- **Differential diagnosis.** Treat reversible causes. Look for a medication bracelet or necklace. If patient history is available, use this information for the differential diagnosis. Question bystanders; look for medications. Reversible causes can be recalled from the "Hs and Ts" list below:
    - Hypovolemia
    - Hypoxia
    - Hydrogen ion (acidosis)
    - Hypo/hyperkalemia
    - Hypothermia
    - Tension pneumothorax
    - Tamponade, cardiac
    - Toxins (overdose/medication)
    - Thrombosis (pulmonary/cardiac)
      (Link, 2015)

Pulseless Arrest Algorithms

The ACLS sequence builds on the BLS guidelines and their emphasis on continuous and excellent CPR. As discussed earlier in this course, chest compressions are the most important component of survivability.

Adding in electrical therapy and medications are the next important steps. With such interventions, it is important to be able to distinguish between the various critical dysrhythmias, which include: ventricular fibrillation/pulseless ventricular tachycardia, asystole, and pulseless electrical activity.

SHOCKABLE PULSELESS RHYTHMS

Ventricular fibrillation (VF) is a common rhythm seen in the out-of-hospital cardiac arrest patient. It is a non-perfusing, chaotic rhythm with no organization. The foci, from the ventricles, are firing randomly and with no conduction, ultimately causing the heart to quiver uselessly. No pumping action occurs, and there is no cardiac output. However, there is electrical activity.

VF may be described as coarse or fine. Coarse VF is characterized by more electrical activity than fine VF. If the patient remains in VF, the electrical activity will gradually decrease because of tissue ischemia. Without intervention, it will deteriorate to asystole (see rhythm strip under "Nonshockable Rhythms" below) (Posner, 2015).

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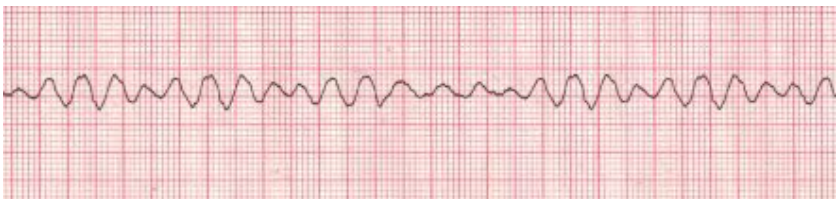
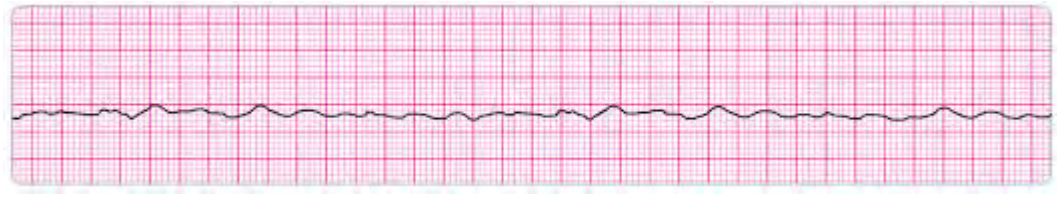


Coarse ventricular fibrillation (VF).

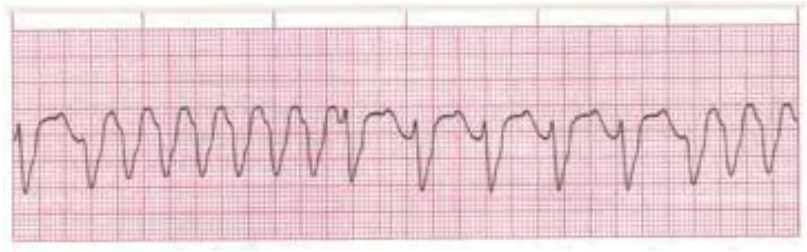


Fine ventricular fibrillation (VF).

Ventricular tachycardia (VT) originates in one of the ventricles of the heart. It may present itself in one of two ways—monomorphic or polymorphic. With **monomorphic VT** all of the electrical complexes will have the same morphology, indicating that they originate from the same location in the ventricles. **Polymorphic VT** originates from multiple locations, causing the complexes to take on different shapes and sizes. Torsades de pointes (a polymorphic ventricular tachycardia, a "twisting of the points," that shifts above and below the isoelectric line) is one of the more commonly recognized polymorphic VTs.

Patients in VT may or may not have a pulse by physical examination, so the pulse check should be done with care. If pulses are clearly palpable, the tachycardia algorithm should be used. If pulses are not clearly felt, the VT is treated as a pulseless arrest.

Monomorphic ventricular tachycardia (VT).



Polymorphic ventricular tachycardia (VT).

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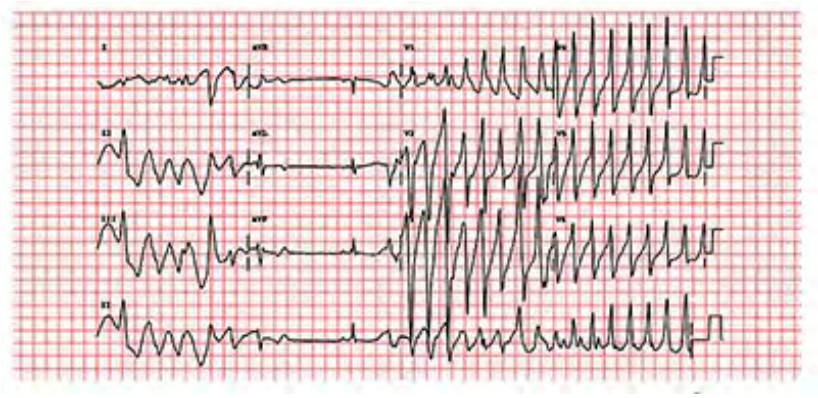

Torsades de pointes (polymorphic VT). (Source: EKG World Encyclopedia, courtesy of Michael Rosengarten.)

The **treatment** of choice for VF/pulseless VT is defibrillation followed by excellent CPR. Defibrillation is at the appropriate energy level, per manufacturer instructions, usually between 120 to 200 joules with a biphasic defibrillator. If using a monophasic machine, defibrillation starts between 200 to 360 joules, as recommended. Chest compressions begin immediately after defibrillation; valuable time should not be spent on a pulse check (Link, 2015).

Several activities take place during a two-minute cycle of CPR. If not already in place, IV or IO access is established. A pulse and rhythm check is done at the end of the cycle. Next come preparations to defibrillate and preparation for medications. (If the rhythm is no longer shockable, the rescuer goes to the appropriate algorithm.)

At the end of a two-minute cycle, if the rhythm is still VF or pulseless VT, defibrillation is done again with the manufacturer's recommendation or higher than the previous dose. After the shock, CPR is resumed immediately without an additional pulse or rhythm check. Epinephrine, 1 mg of the 1:10,000 solution, should be given while compressions proceed. Epinephrine may be administered every 3 to 5 minutes. Vasopressin is no longer recommended (Link, 2015).

Intubation or the insertion of an advanced airway should be considered at this point, however the need for intubation should be weighed against the need for continuous chest compressions (Link, 2015). If the patient is intubated, it is important to use quantitative waveform capnography. Normal values of the partial pressure of the end-tidal CO_2 ($PETCO_2$) measurement should be between 35 mm HG and 45 mm Hg. If the $PETCO_2$ is less than 10 mm Hg, attempts should be made to improve CPR quality. (See also the explanation of $PETCO_2$ later in this course.)

At the end of the next two-minute cycle, if the rhythm is still VF or pulseless VT, defibrillation is done at the highest setting (Link, 2015). CPR is resumed for two minutes, and then an antiarrhythmic medication is administered. A 300 mg bolus of amiodarone is given during chest compressions. Reversible causes (the Hs and Ts) should be investigated and treated.

The cycle is repeated by alternating shocks followed by vasopressors and antiarrhythmics as long as the patient remains in VF or pulseless VT. All subsequent shocks should be delivered at the highest energy level. If adequate airway and ventilations are achieved by bag-mask, advanced airways may be deferred until the patient is more stable. CPR with a supraglottic advanced

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airway or endotracheal intubation should include 10 breaths per minute with continuous chest compressions (Link, 2015).

CASE *(continued)*

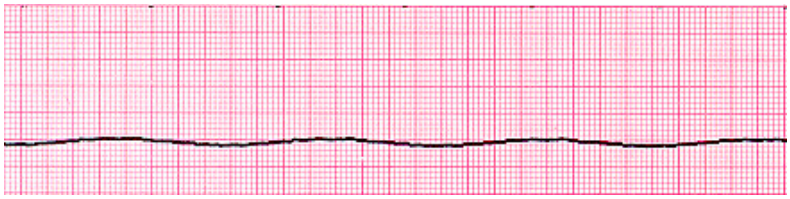
As the nurse Susie was delivering compressions to Sam in the hospital parking lot, the emergency department (ED) team arrived on the scene. One ED nurse took over compressions while another started giving breaths with the bag-mask in the same ratio Susie had been using (30:2). During this transition, Sam was moved onto a gurney and transported into the ED.

Once inside, another nurse cut open Sam's gown and set up the cardiac monitor, which showed ventricular fibrillation (VF). The code team was now assembled and a team leader was chosen. The team then prepared to defibrillate. A shock was delivered to Sam and chest compressions were immediately continued. While compressions were being delivered, IV access was obtained. After two minutes of CPR, VF was still noted. Another shock was delivered and chest compressions resumed.

Epinephrine, 1 mg IV, was given and an advanced airway was considered. However, it was determined to be too risky to take a break to intubate. After another two minutes of chest compressions, a sinus rhythm was noted. Sam's carotid pulse was checked and found. A return of spontaneous circulation had occurred, and the team began Sam's post-resuscitation care.

NONSHOCKABLE RHYTHMS

Asystole is the complete absence of demonstrable electrical and mechanical cardiac activity; it is most often irreversible. There is no electrical activity occurring from either the atria or the ventricles. Confirmation of this rhythm should be made by viewing it in at least two different leads. At times, very fine VF can be mistaken for asystole. For treatment, see "Pulseless Electrical Activity" below. It is imperative that, if not already begun, chest compressions are initiated and that reversible causes are sought (Posner, 2015).



Asystole (flatline).

Pulseless electrical activity (PEA) is defined as "any one of a heterogeneous group of organized electrocardiographic rhythms without sufficient mechanical contraction of the heart to produce a palpable pulse or measurable blood pressure" (Posner, 2015). There is organized electrical activity seen on the monitor that could be expected to produce a pulse, but there is no actual mechanical activity occurring, and so no pulses are being generated. Because of its nature it is treated like asystole.

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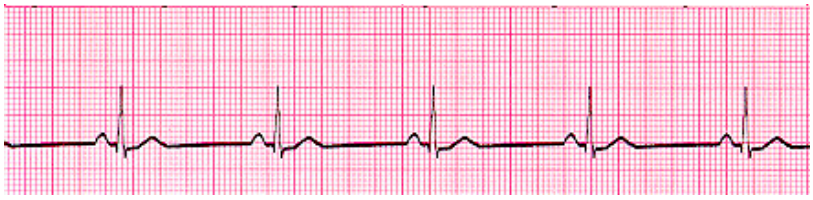

Pulseless electrical activity (PEA) can be described as any electrical rhythm without a pulse.

An early consideration with asystole or PEA is whether this is a treatable event. The Hs and Ts need to be brought to attention early. This is often easier to determine in a hospital setting, where medical history and advanced directives are more readily available. Information from family members, advanced directives, hospice workers, and local protocols should be considered in determining whether any resuscitation should be attempted.

If asystole or PEA is present and the determination has been made to resuscitate, CPR is begun and reversible causes are immediately considered. An IV or IO line is established as soon as possible. A dose of 1 mg epinephrine may be given as soon as venous access is in place.

In the event of asystole or PEA, the clinician must address the Hs and Ts in the differential diagnosis. By identifying the cause, a cure may be found. Often, the cause is irreversible.

After two minutes of CPR, rescuers perform a pulse and rhythm check. If asystole or a PEA is still present, CPR is immediately continued. Epinephrine continues to be administered every three to five minutes.

The cycle of epinephrine and CPR is repeated as long as the patient remains in asystole or PEA or until death is determined. Intubation or placement of an advanced airway should take place as soon as it is feasible. Again, the benefit of continuous compressions is assessed and postponing placement is considered if good ventilation can be obtained with a bag-mask.

Because of the absence of electrical activity, there is no benefit to defibrillation. Pacing is also not effective in either of these nonshockable dysrhythmias (Link, 2015).

Medications

DELIVERY ROUTES

Peripheral IV access should be established as soon as possible for the administration of medications and fluids. If access is difficult and results in delayed therapy, the adult intraosseous (IO) route may be used. IO has the advantage of being quick and easy to place. Any medications or IV fluids that go through an IV line can be delivered through an IO line, and uptake is almost as rapid. The location and technique of placement depends on which device is used, but possible sites include the distal femur, humeral head, tibial plateau, and the manubrium.

Should attempts at establishing an IV or IO access fail, endotracheal (ET) administration can be considered as a last resort or while attempting to establish IV or IO access. The IV or IO route is

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greatly preferred because it will provide a more predictable drug delivery and pharmacological effect. ET doses should be 2 to 2.5 times the standard IV dose and diluted in 5 mL to 10 mL of sterile water or normal saline (AHA, 2015).

CLASSES OF MEDICATIONS

Medications given in pulseless arrests fall into two general categories. Vasopressors are given for their alpha-adrenergic properties in hopes that they will increase perfusion to the brain and heart. This makes a return of spontaneous circulation more likely. Antiarrhythmics are used to convert nonperfusing and hypoperfusing rhythms. Neither class of drugs has been shown to improve survival rates to discharge home (Posner, 2015).

Vasopressors

A vasopressor should be the first medication considered after oxygen in any pulseless arrest. There is evidence that vasopressors can facilitate the return of spontaneous circulation (ROSC) during a code, even though they have not been shown to increase the rate of neurologically intact survival to hospital discharge. The recommended vasopressor in cardiac arrest is epinephrine (Posner, 2015).

Epinephrine is the most familiar and commonly given medication during an emergency cardiac event. It is thought to have a stimulatory effect on adrenergic receptors causing vasoconstriction and increased myocardial and cerebral blood flow during CPR. The dosing for epinephrine is 1 mg IV/IO of the 1:10,000 solution given every 3 to 5 minutes. High-dose epinephrine has not been shown to improve survival rates and is not recommended (Posner, 2015).

Antidysrhythmics

Amiodarone is a preferred antidysrhythmic for VF/pulseless VT. In studies, amiodarone increased ROSC rates, but it has not increased survival to hospital discharge, nor does any other antidysrhythmic studied. Even so, the *2015 AHA Guidelines* are supportive in its use in certain situations (Posner, 2015).

Amiodarone has multiple effects on the myocardium, affecting the sodium, calcium, and potassium channels. It also has alpha- and beta-adrenergic blocking properties. It has many side effects and interactions with other drugs and should only be given by those very familiar with its administration.

The VF/pulseless VT dose for amiodarone is a one-time 300 mg rapid IV/IO push. If the rhythm has not converted after subsequent shocks and epinephrine administration, a second dose of 150 mg is given (Link, 2015).

Lidocaine can be considered an alternative for VF/pulseless VT if amiodarone is not available. However, in studies lidocaine was found to be less effective than amiodarone

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(Posner, 2015; Link, 2015). Lidocaine decreases the automaticity of the myocardium, which helps to reduce fibrillation, especially in ischemic tissue. In a pulseless patient, the dose is 1 to 1.5 mg/kg IV/IO initially. If the arrhythmia persists, doses of 0.5–0.75 mg/kg IV/IO push can be administered at 5 to 10 minute intervals to a maximum dose of 3 mg/kg.

Electrolyte replacement (such as calcium and potassium) may be considered after blood analysis and should not be given routinely. Magnesium can be given after defibrillation in a patient with torsades de pointes or suspected hypomagnesemia (Link, 2015). If given, the magnesium dose is 2 g by IV/IO; this can be followed by a maintenance infusion (Posner, 2015).

Advanced Airways

The placement of an advanced airway can take significant time, which may cause a prolonged interruption of CPR. Healthcare providers should weigh the need for compressions against the need for the advanced airway. Advanced airway management "must never be made at the expense of performing excellent CPR and early defibrillation" (AHA, 2015).

If adequate ventilations can be accomplished by a bag-mask device and no immediate risk for occlusion or aspiration is seen, it may be prudent to delay placement of an advanced airway until several cycles of CPR have been performed and several shocks have been delivered (Link, 2015). This requires maintenance of excellent bag-mask skills.

Advanced airways include **endotracheal tube**, **laryngeal mask airway**, or a **laryngeal tube**. Whichever device is chosen, it should be placed by the most experienced person available who has mastered the skill through practice, frequency of placement, and education. Chest compressions can continue while some advanced airway devices—such as a laryngeal mask airway, laryngeal tube, or esophageal-tracheal tube—are placed.

If an attempt to place an advanced airway fails, a cycle of CPR and bag-mask ventilation should be performed before any subsequent attempts are made. All equipment should be checked and close at hand before compressions are stopped for the placement attempt. Once placed, an ET tube must be confirmed by waveform capnography, secured, and monitored continuously (Link, 2015).

RATE DISTURBANCES

Bradycardia

Bradycardia (slow heart rate) is generally considered to be a resting heart rate of fewer than 60 beats per minute. Many times, a slow heart rate may be a completely normal presentation in some patients. The bradycardia algorithm (see below) covers symptomatic bradycardia as well as atrioventricular (AV) blocks (which are not discussed in detail in this course) (AHA, 2015).

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The first rule of cardiac care (and all patient care assessments): **treat the patient, not the monitor.** Although the monitor shows a slow heart rate, it does not mean that the patient will need treatment.

If bradycardia is accompanied by signs and symptoms of poor perfusion, such as chest pain, shortness of breath, altered mental status, hypotension, or shock, the rhythm should be treated immediately. If the patient has adequate perfusion, observation may be all that is needed until a physician can do full work-up.

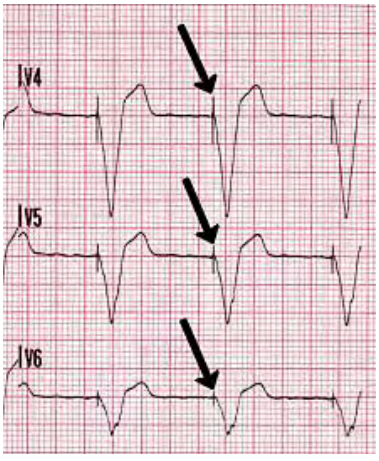
For symptomatic bradycardia, a 0.5 mg bolus of atropine is administered. It may be repeated every 3 to 5 minutes to a maximum of 3 mg. If atropine is ineffective, preparation should be made for transcutaneous pacing or a dopamine or epinephrine infusion (AHA, 2015).

PACING

Transcutaneous pacing should be considered along with dopamine or epinephrine infusions for symptomatic bradycardia if atropine is ineffective (AHA, 2015). To set up for pacing, the skin is cleaned and excess hair is clipped to enhance adhesion and conduction. Pacemaker pads are placed in the recommended position on the anterior chest overlying the heart and posteriorly overlying the heart. Monitoring leads should also be in place. Pacing can be very painful, so sedation and analgesia should be considered if they can be accomplished quickly.

The pacemaker rate should be set at a heart rate for an adult patient, usually 60 to 80 bpm, per policy and expert consultation. When setting the current, it is started low and increased until capture is seen on the monitor. This is characterized by pacemaker spikes consistently followed by a wide QRS complex. The pulse is checked for correlation with the monitor rhythm using a site other than the carotid. Sedation should be considered if it was not done before starting. A check is made for signs of improved perfusion. An unresponsive patient may become responsive and very uncomfortable with successful pacing.

If transcutaneous pacing fails, transvenous pacing may need to be initiated with expert consultation (AHA, 2015; Posner, 2015).



Paced rhythm, with the pacemaker spikes indicated by arrows. (Source: Adapted from Glenlarson.)

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MEDICATIONS

Atropine is one of the first medications to consider for sinus bradycardia as well as first-degree or second-degree type I AV blocks. An anti-cholinergic and muscarinic, **atropine** interrupts the parasympathetic nerve impulses in the central and autonomic nervous systems, allowing an increase in the heart rate, systemic vascular resistance, and blood pressure.

Atropine is rarely effective on high-degree blocks, such as second-degree type II or third-degree, and may cause further deterioration in condition. Be aware that patients with transplanted hearts most likely will not respond to atropine because of vagal denervation.

The suggested dose of atropine is 0.5 mg IV every three to five minutes to a maximum dose of 3 mg for symptomatic bradycardia (AHA, 2015).

If atropine fails and the patient remains hypotensive, transcutaneous pacing or a **dopamine** or **epinephrine** infusion can be considered. The dosing of both dopamine and epinephrine is 2 to 10 mcg/min. Both are titrated to the patient's response.

Both dopamine and epinephrine have vasoconstricting as well as chronotropic effects. Dopamine is a catecholamine agent with dose-related dopaminergic and beta- and alpha-adrenergic agonist activity. The effect is rate dependent. At lower doses (2 to 10 mcg/kg/min) it has a greater effect on inotropy and heart rate; at higher doses (>10 mcg/kg per minute) it also has vasoconstrictive effects and the rate should be titrated as needed (AHA, 2015).

Tachycardia

Tachycardia (fast heart rate) is generally considered to be a resting heart rate over 100 beats per minute in an adult. Fast rates may be benign or they can be very dangerous. This will depend on the patient's age, underlying heart condition, and duration and morphology of the arrhythmia. The faster the rate, the less likely it is that it will be tolerated for any length of time.

As with all advanced care, the patient examination begins with the basics of circulation, airway, and breathing and correcting any problems. An initial electrocardiogram (ECG) reading is obtained as soon as possible. If the rate is less than 150, the patient's symptoms may not be related to the rate, in which case it is important to look for other causes such as hypovolemia, pain, and fear.

EVALUATING THE RHYTHM

Tachycardias are evaluated on the basis of the stability of the patient, heart rate, width of the QRS complex, and regularity of the rhythm. The stability of the patient is determined during the BLS (primary) and ACLS (secondary) survey.

If the patient has signs of poor perfusion—such as chest pain, hypotension, shortness of breath, or altered mental status—immediate **synchronized cardioversion** should be considered. If IV

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access is in place, sedation may be considered if it can be done without significant delay. For synchronized cardioversion, the energy doses are as follows:

- Narrow regular: 50 to 100 joules (J)
- Narrow irregular: 120 to 200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (not synchronized)
  (AHA, 2015)

If the patient is stable and tolerating the tachycardia well, there is more time for decision making. The clinician establishes an IV if one is not already in place; obtains a 12-lead ECG for a better look at the dysrhythmia; and determines the width of the QRS complexes. This will determine the next steps of treatment. If the QRS complex is <0.12 seconds, it is considered narrow; if it is ≥0.12 seconds, it is considered wide (AHA, 2015).

NARROW-COMPLEX TACHYCARDIAS

If the QRS complex is narrow in any lead, it is important to determine whether the rhythm is regular or irregular.

With a narrow, regular rhythm, **vagal maneuvers** can be considered, either as a treatment or as a diagnostic tool. They are generally simple to perform and are successful in about 20% of cases. However, they should not be performed if the patient has severe coronary artery disease, has had a recent heart attack, or has a reduction in blood volume. A patient with any of these conditions could experience detachment of blood clots, resulting in stroke (cerebrovascular accident, or CVA), vertigo, cardiac arrhythmias, or even arrest.

Valsalva maneuver has fewer complications than other methods, but it depends on the patient's cooperation and ability to perform the maneuver. Some treatment methods to try may include having the patient: bear down (as if having a bowel movement), forcibly exhale while keeping their mouth and nose closed, blow into a large syringe against the plunger, or blow into an occluded straw. Unilateral carotid sinus massage can be performed, but only by a trained professional, as carotid plaques can be dislodged, causing a CVA.

One of the most common causes of tachycardic rhythms is hypoxemia. If the patient shows signs of an increased work of breathing, **supplementary oxygen** is provided. If vagal maneuvers and supplementary oxygen do not reduce the rate and discomfort, medications may be considered (AHA, 2015).

In regular, narrow-complex tachycardias (less than 0.12 seconds), such as re-entrant tachycardia, **adenosine** may be considered. Adenosine blocks conduction through the AV node and interrupts the re-entrant aberrancy. When giving adenosine, delivery method and speed are key considerations. The half-life of this medication is only about 5 seconds, so it needs to get from the IV injection site into central circulation very quickly. The IV

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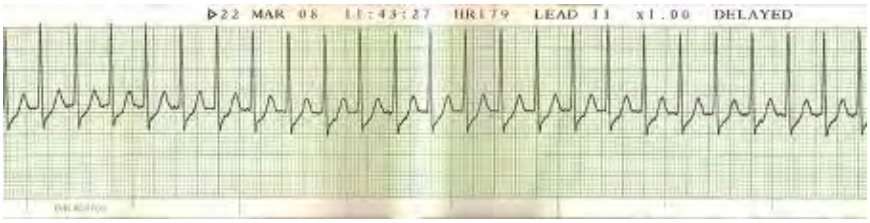
site should be proximal to the patient, preferably in the antecubital, with the largest catheter possible. It is also preferable to elevate the extremity to ensure rapid infusion.

Adenosine is administered at 6 mg by rapid IV bolus injection followed immediately by 20 ml of normal saline flush by rapid bolus injection. If the tachycardia has not converted, a second dose of adenosine, 12 mg, is given. A repeat dose of 12 mg may be considered.

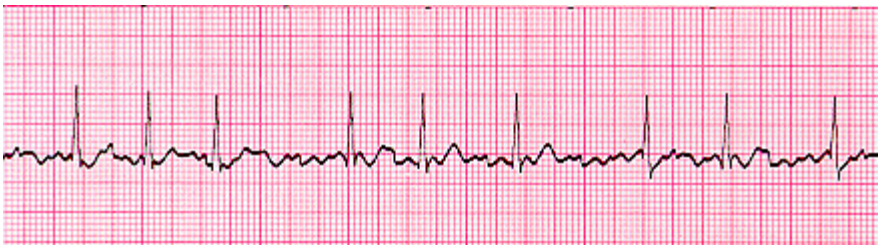
The side effects of this medication are sometimes severe, and the patient should be warned about them. These may include chest pain, shortness of breath, and sometimes a feeling of impending doom. If possible, some sedation may be given prior to administration of adenosine (Posner, 2015). Adenosine may be used in pregnancy.

Some medications may interact with adenosine. Larger doses may be needed if the patient takes theophylline, caffeine, or theobromine. Smaller doses may be needed if the patient takes dipyridamole or carbamazepine, in those with heart transplants, or if given by central venous access (AHA, 2015).



Supraventricular tachycardia (re-entrant tachycardia).

Should cardioversion attempts fail with adenosine, other supraventricular rhythms are considered, such as atrial fibrillation and atrial flutter. In these narrow-complex tachycardias, calcium channel blockers (such as diltiazem or verapamil) and beta blockers (such as metoprolol, atenolol, esmolol, and labetalol) are routinely used (Posner, 2015).



Atrial fibrillation.

WIDE-COMPLEX TACHYCARDIAS: REGULAR

The most common form of regular wide-complex tachycardias is **monomorphic** ventricular tachycardia. Ventricular filling time is short and cardiac output can drop quickly. Usually, adult patients will quickly become unstable in this arrhythmia. In a patient who is stable initially, treatment is with **antiarrhythmics.**

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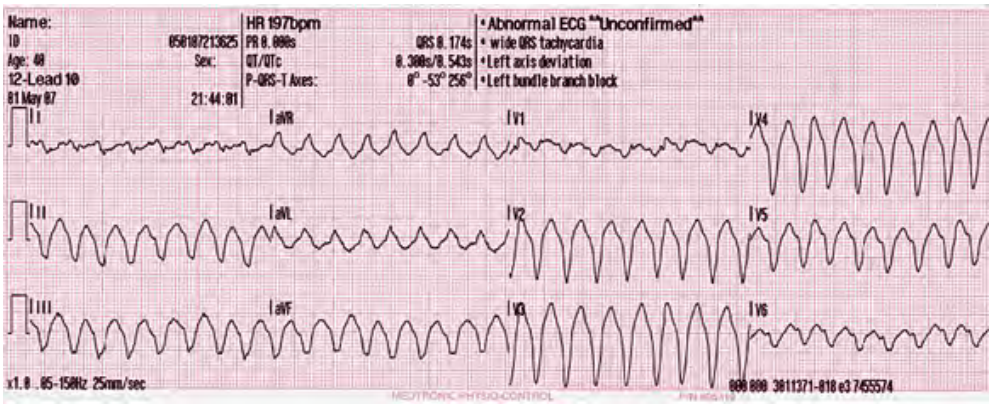
Adenosine may be used as a diagnostic tool for regular wide-complex tachycardia with an assumed monomorphic QRS complex (see dosing above). Adenosine may slow the aberrancy long enough to visualize the underlying rhythm. Adenosine should not be administered for polymorphic wide-complex tachycardias (such as torsades de pointes), as it may cause degeneration of the arrhythmia to VF (AHA, 2015).

If the patient has a diagnosed stable wide-complex tachycardia, treatment may include procainamide, amiodarone, or sotalol. Procainamide or sotalol should not be administered if there is a prolonged QT interval. The patient should be monitored closely and, if at any time they become unstable, cardioversion is undertaken (AHA, 2015; Posner 2015).



Monomorphic ventricular tachycardia.

Procainamide, though not a medication considered for VF/pulseless VT, can be considered for stable wide-complex VT without a prolonged QT interval (AHA, 2015). It reduces automaticity in all pacemakers and slows intraventricular conduction. It also has vasodilatory effects, especially with rapid administration and high doses. Procainamide is administered at a rate of 20 mg/min to 50 mg/min IV until the arrhythmia is suppressed, hypotension occurs, the QRS widens by >50% from baseline, or the maximum dose of 17 mg/kg is given. If the arrhythmia is suppressed by procainamide, maintenance infusion is begun at 1 mg/minute to 4 mg/minute (AHA, 2015).

For stable wide QRS VT, 150 mg of amiodarone is administered over 10 minutes. The dose is repeated as needed until the rhythm converts or the patient becomes unstable. The maximum dose is 2.2 g in a 24-hour period (Posner, 2015; AHA, 2015).

Sotalol may be considered as an alternate antiarrhythmic for VT with a wide QRS. The first dose is 100 mg (1.5 mg/kg) IV over 5 minutes, which is repeated as needed if VT recurs. Sotalol should be avoided if the QT interval is prolonged (Posner, 2015; AHA, 2015).

WIDE-COMPLEX TACHYCARDIAS: IRREGULAR

The three most common irregular wide-complex tachycardias are polymorphic VT (such as torsades de pointes), aberrantly conducted atrial fibrillation (such as atrial fibrillation with

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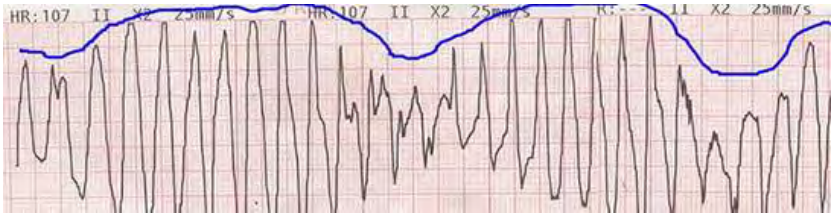
bundle branch block), or pre-excited atrial fibrillation (such as atrial fibrillation with Wolf Parkinson White syndrome) (Posner, 2015).

A **polymorphic VT** is one in which different foci of the heart are initiating the impulse. It tends to have a poorer prognosis than monomorphic VT and usually deteriorates quite quickly into a pulseless arrhythmia. It is often associated with a prolonged QT interval prior to collapse. If this has been documented, any medications that may prolong the QT interval should be stopped. Then problems such as electrolyte imbalance or drug overdose should be corrected.

In the case of irregular tachycardia of unclear etiology, it is important to **not** deliver AV nodal blockade, such as adenosine. This can precipitate VF and is contraindicated (Posner, 2015).



Polymorphic VT.

If the patient has a rhythm of torsades de pointes, documented hypomagnesemia, or documented long QT interval, magnesium may be considered (Medscape, 2016). A dose of 2 g IV is administered and followed by a maintenance infusion. Slower rates are preferred in the stable patient. If it is a polymorphic VT other than torsades, it is treated as a regular wide-complex tachycardia with amiodarone.

Patients in polymorphic VT become unstable quickly, so it is important to be prepared to defibrillate and to start compressions after defibrillation. This rhythm may be too irregular to cardiovert, as most monitors cannot synchronize to it. It should be treated as a pulseless rhythm.

If the rhythm is a new onset **atrial fibrillation,** it is treated as an irregular narrow-complex tachycardia, with the initial focus on rate control. Conversion of the rhythm can be done later under more controlled circumstances, unless the patient is unstable. If the patient has a documented **pre-excitation syndrome** such as Wolff-Parkinson-White (WPW), certain medications should not be administered: those that block the AV node, calcium channel blockers, or beta blockers. These drugs can actually increase the rate.

If the atrial fibrillation has a duration of greater than 48 hours, the patient is at a greater risk for cardioembolic events. In this case, cardioversion, either by medication or electrical therapy, should be avoided unless the patient is unstable. If the patient is stable, it is preferred to treat with anticoagulant therapy and perform a transesophageal echocardiograph (TEE) to ensure that no clot exists in the left atrium (AHA, 2015).

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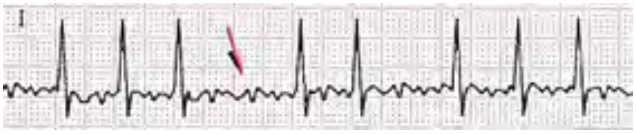

Atrial fibrillation. (Source: J. Heuser.)

Post-Resuscitation Care

In a cardiac arrest patient who has a return of spontaneous circulation, there are important goal-oriented interventions, led by an experienced multidisciplinary team, for his/her post-resuscitation care:

- Optimizing cardiopulmonary function and perfusion of vital organs
- Managing acute coronary syndromes
- Implementing strategies to prevent and manage organ system dysfunction (Posner, 2015)
- Considering the use of targeted temperature management (TTM) between 32 °C and 36 °C for 12 to 24 hours post arrest if remaining unresponsive (Callaway, 2015)

Termination of Resuscitative Efforts

The decision to stop resuscitation efforts may be difficult. Posner (2015) recommends considering the following based on physician survey data and clinical practice guidelines:

- Duration of resuscitative effort >30 minutes without a sustained perfusing rhythm
- Initial electrocardiographic rhythm of asystole
- Prolonged interval between estimated time of arrest and initiation of resuscitation
- Patient age and severity of comorbid disease
- Absent brainstem reflexes
- Normothermia

There are other objective data that can be considered at this stage of the event, such as the use of end-tidal carbon dioxide measurement ($PETCO_2$). Normal end-tidal CO_2 measurements reflect arterial blood gas measurements but will read slightly lower (normal arterial $PaCO_2$ = 35 to 45 mm Hg, $PETCO_2$ = 30 to 35 mm Hg). If the $PETCO_2$ level is <10 mm Hg with a prolonged resuscitation (>20 minutes), this shows an absence of circulation and a clear indication of tissue death. It is also imperative to know if the endotracheal tube has been dislodged. ET tube displacement may also cause low $PETCO_2$ readings and must be corrected prior to terminating resuscitative efforts (Posner, 2015).

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CASE

Joe is a patient of Agnes, a nurse on the medical-surgical floor. While Agnes was down the hall in the hospital pharmacy, the telemetry tech called to let her know that Joe's cardiac monitor was reading tachycardia at a rate of 167 beats per min. Agnes rushed to Joe's room, where he was sitting up in bed talking with his family. Agnes took Joe's vital signs and found him to be otherwise stable.

While she was glad to see he was not symptomatic, Agnes still called the rapid response team (the house nursing supervisor, a designated ICU nurse, and a respiratory therapist) for help and requested the code cart be brought to the bedside, knowing Joe could deteriorate. When the team arrived, Agnes explained to Joe the rationale for what she had done as she placed him on the bedside cardiac monitor and attached a BP cuff and pulse oximeter. The team confirmed a continuing tachycardia, with a wide and regular QRS complex. Agnes told Joe to prepare for a strange sensation before the ICU nurse administered adenosine, 6 mg, by rapid IV push, followed with a NS flush.

After receiving the adenosine, Joe was still tachycardic. His level of consciousness (LOC) became altered, and he began to grasp at his chest, becoming hypotensive and hypoxemic with a weak pulse. Since Joe's condition was now unstable, the code team was called. The team physician quickly sedated him and then proceeded with cardioversion. With synchronized cardioversion, Joe returned to a sinus rhythm.

ACUTE CORONARY SYNDROMES (ACS)

Most cases of sudden cardiac death are immediately preceded by acute myocardial infarction, or heart attack; therefore, early recognition and prompt, aggressive care of this condition is very important.

Early Recognition, Assessment, and Treatment

The overall goal that directs all therapies and protocols is for the patient to get the treatment they need in the shortest time possible.

The interval between onset of symptoms and delivery of definitive care has many stages where delay may occur. The first and longest delay is from the time of onset of symptoms to the decision by or for the patient to seek care.

It is very important to call 911 early, and public education in this matter should be aggressive and ongoing. Emergency dispatchers should be trained to ask a series of questions while dispatching an ambulance. If they determine that the patient is having signs or symptoms of an acute myocardial infarction, they should advise the patient to immediately chew an aspirin (160 mg to 325 mg) as long as the patient does not have an aspirin allergy or recent gastrointestinal bleeding (AHA, 2015).

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Prehospital emergency medical service (EMS) personnel need to rapidly recognize an acute coronary syndrome and deliver the patient to an appropriate facility in a timely manner. EMS should also have protocols in place to initiate care and notify the receiving facility to prepare for a cardiac patient. Prehospital 12-lead ECG can significantly shorten the time to definitive care and is becoming the gold standard in many areas (AHA, 2015).

Finally, the hospital emergency department should be ready to rapidly evaluate the patient and facilitate definitive care.

The first healthcare provider to encounter any patient with signs and symptoms of ACS should begin with general assessment and treatment (as outlined earlier in this course in the BLS and ACLS surveys). Further assessment covering cardiac history, signs and symptoms, risk factors for cardiac disease, and screening for the administration of fibrinolytics should be completed in less than 10 minutes. A 12-lead ECG should be obtained as quickly as possible.

Specific care for ACS includes the mnemonic ***MONA,*** which stands for morphine, oxygen, nitroglycerin, and aspirin.

- **Oxygen** delivery should begin first and should only be used for patients who are dyspneic, hypoxemic, or have obvious signs of heart failure, titrated to keep the patient's oxygen saturation (SaO_2) levels at or above 94%.
- Non-enteric coated **aspirin,** 160 mg to 325 mg, should be chewed as soon as possible. This may be deferred in the presence of recent GI bleed or true aspirin allergy.
- **Nitroglycerin** should be administered by sublingual tablet or spray as long as the patient has a systolic BP over 100 mm Hg and a pulse between 50 and 100. Specific facility guidelines on the administration of nitroglycerin should be followed. Typically, a dose of 0.4 mg is given every three to five minutes while the symptoms persist and the vital signs remain stable.
- Once three doses of nitroglycerin have been given, **morphine** may be given for persistent symptoms.

  (AHA, 2015)

Once the patent has been assessed, they will be stratified into one of three categories—ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and indeterminate—and this will determine subsequent treatment.

STEMI

If the patient has a new left-bundle branch block, or if the patient has ST-segment elevation >1 mm (0.1 mV) in two or more contiguous precordial leads or two or more adjacent limb leads, they are classified as having an ST-segment elevation MI, or STEMI. Most patients in this category have blockage of a coronary artery.

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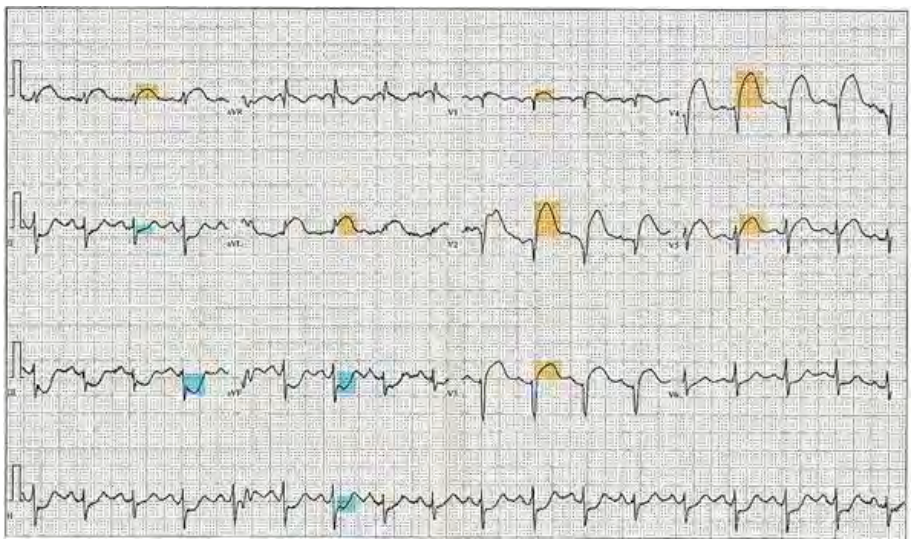

ST-segment elevation myocardial infarction (STEMI).

The goal of treatment is early reperfusion to eliminate or minimize necrosis of the heart muscle. This can be done with **fibrinolytics** or by performing **primary percutaneous coronary intervention (PPCI)**. In many instances, PPCI is superior to fibrinolytic administration in restoration of vessel patency and has a lower rate of reocclusion.

The *2015 AHA Guidelines* state that:

1. If initial symptom onset is <12 hours and time to deliver patient to a hospital with PPCI is <120 minutes, PPCI is recommended.
2. If there is a delay of >60 minutes to a facility that can perform PPCI and the patient presents within 2 hours of onset, immediate fibrinolytics are a reasonable treatment.
3. If the delay to a tertiary facility is between 60 to 120 minutes and the patient presents within 2 to 3 hours of symptom onset, either fibrinolytics or PPCI are a reasonable treatment.
4. If the delay to intervention is >120 minutes and the patient presents between 3 to 12 hours, PPCI is preferred over fibrinolysis.

It is important to note that fibrinolytics become less effective the longer the symptoms have persisted (O'Connor et al., 2015).

NSTEMI

Patients demonstrating ischemic ST-segment depression of at least 0.5 mm (0.05 mV) or dynamic T-wave inversion with chest pain or discomfort are classified as non–ST-segment elevation MI (NSTEMI). High-risk patients with unstable angina are also included in this category, as are patients who have ST elevation of more than 0.5 mm but lasting less than 20 minutes.

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Generally, patients with ST-segment depression do not have a complete coronary artery blockage and may not be having an ACS. However, patients with chest pain and positive or elevated serum cardiac markers, diffuse or widespread ECG abnormalities, or heart failure have an increased risk of major acute cardiac event (MACE). Patients displaying ST-segment depression may be having a posterior MI; this should be confirmed with diagnostic tests.

Treatment options for these patients can include **beta-adrenergic blockers, clopidogrel, heparin therapy,** and/or **glycoprotein IIb/IIIa inhibitors** as well as early **PCI**. These patients should not receive fibrinolytics (AHA, 2015).

Normal or Nondiagnostic ECG

If the patient has normal or nonspecific ECG changes, he or she is considered indeterminate pending further evaluation. Evaluation will include cardiac markers and possibly a stress test. Therapies should be considered on the basis of risk versus benefit (AHA, 2015).

CONCLUSION

The information in this course covers the early, basic care of most patients with emergency cardiac conditions. Many factors can complicate care—such as drug overdose, hypothermia, trauma, or pregnancy—and are beyond the scope of this course. Providers who can expect to encounter such conditions regularly require additional education appropriate to their situation. Guidelines for cardiac care are just that: guidelines. Local protocol should be the ultimate authority for any care given.

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TEST

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1. While performing cardiopulmonary resuscitation (CPR), the rescuer allows the patient's chest to fully recoil between chest compressions in order to:

a. Force blood out of the heart chambers.
b. Reduce cardiac output.
c. Allow for blood return to the lungs and heart.
d. Reduce rescuer fatigue.

2. CPR chest compressions for the adult patient should be given at a rate of:

a. 60 per minute and at a depth of 1-1/2 to 2 inches.
b. 75 per minute and at a depth of at least 2 inches.
c. 90 per minute and at a depth of 1-1/2 to 2 inches.
d. 100 to 120 per minute and at a depth of at least 2 inches.

3. The *2015 AHA Guidelines* recommend adult cardiopulmonary resuscitation with a compression-to-ventilation ratio of:

a. 15:2.
b. 20:1.
c. 15:1.
d. 30:2.

4. During basic life support, an automated external defibrillator (AED) is used:

a. In lieu of high-quality compressions.
b. During very brief interruption of high-quality compressions.
c. After two complete cycles of CPR (4 minutes) is completed.
d. Before attaching the device to the patient.

5. Chest compressions should begin for patients with foreign body airway obstruction when:

a. The patient indicates he or she is choking.
b. Five abdominal thrusts are ineffective.
c. Pulselessness is well established.
d. The patient becomes unresponsive.

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6. What is the rationale for including differential diagnosis in the advanced cardiac life support (ACLS) survey?

a. It helps to activate the emergency response system.
b. It is a reminder to obtain an automatic external defibrillator.
c. It is a method to evaluate patient response level.
d. It can help to discern reversible causes and contributing factors.

7. Immediately after defibrillation of the patient, the code team's action is to:

a. Resume chest compressions.
b. Place an advanced airway.
c. Give medications.
d. Defibrillate with stacked shocks.

8. Asystole is a heart rhythm that most often is:

a. Treated with defibrillation.
b. An indicator of regular ventricular activity.
c. Treated with slower compressions rates.
d. Irreversible.

9. Amiodarone is a preferred antidysrhythmic medication for ventricular fibrillation/pulseless ventricular tachycardia because it has:

a. Few side effects and interactions with other drugs.
b. Shown increased patient return of spontaneous circulation.
c. Shown increased patient survival rates to hospital discharge.
d. Singular treatment effect on myocardial function.

10. Bradycardic rhythms should be treated:

a. When the patient's heart rate falls below 60 beats/minute.
b. If the patient shows signs of poor perfusion.
c. With early defibrillation.
d. After hypotension and shock have been managed.

11. Treatment for tachycardia depends on whether the patient:

a. Is showing signs of hemodynamic instability.
b. Is less than 35 years of age.
c. Has a pulse of 120 beats/min.
d. Has had a 12-lead ECG.

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12. When administering IV adenosine to a patient with tachycardia, the nurse first:

a. Educates the patient about possible adverse side effects.
b. Explains the need for slow infusion.
c. Stops the patient's supplementary oxygen administration.
d. Ensures a female patient is not pregnant.

13. Patients with wide-complex tachycardias are closely evaluated due to their:

a. Longer than normal ventricular filling times.
b. Increased cardiac output status.
c. Poor medical treatment prognosis.
d. Potential to rapidly become unstable.

14. Sudden cardiac death is most often preceded by which condition?

a. Blunt trauma
b. Acute myocardial infarction
c. Near drowning
d. Severe hypovolemia

15. Patients with ST-segment elevation myocardial infarction (STEMI) are treated:

a. Primarily in the home setting.
b. With early reperfusion therapy.
c. With catecholamines and antiarrhythmics.
d. Only after diagnostic findings are confirmed by a stress test.

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