

# ADVANCED CARDIAC LIFE SUPPORT

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## OBJECTIVES

### Introduction

### Airway management and ventilation

### Alternative Airway Devices

### Ventricular Tachycardia (VT)

### Ventricular Fibrillation

### Drugs for VF/ Pulseless VT

### Asystole (Cardiac Standstill)

### Pulseless Electrical Activity (PEA)

### Adult Cardiac Arrest Algorithm

## CONCLUSIONS

## REFERENCES

**Introduction** Heart rhythms associated with cardiac arrest are divided into two groups: shockable rhythms (ventricular fibrillation/pulseless ventricular tachycardia (VF/VT)) and non-shockable rhythms (asystole and pulseless electrical activity (PEA)). The principal difference in the management of these two groups of arrhythmias is the need for attempted defibrillation in those patients with VF/VT. Subsequent actions, including chest compressions, airway management and ventilation, venous access, administration of adrenaline and the identification and correction of reversible factors, are common to both groups.

**Advanced Cardiac Life Support (ACLS).** – These terms refer to attempts at restoration of spontaneous circulation using Basic CPR plus advanced airway management, endotracheal intubation, defibrillation, and intravenous medications.

### Airway management and ventilation

Patients requiring resuscitation often have an obstructed airway, usually secondary to loss of consciousness, but occasionally it may be the primary cause of cardiorespiratory arrest. Prompt assessment, with control of the airway and ventilation of the lungs, is essential. This will help to prevent secondary hypoxic damage to the brain and other vital organs. Without adequate oxygenation it may be impossible to restore a spontaneous cardiac output. These principles may not apply to the witnessed primary cardiac arrest in the vicinity of a defibrillator; in this case, the priority is immediate attempted defibrillation. Airway obstruction Causes of airway obstruction Obstruction of the airway may be partial or complete. It may occur at any level, from the nose and mouth down to the trachea. In the unconscious patient, the commonest site of airway obstruction is at the level of the pharynx. Until recently this obstruction had been attributed to posterior displacement of the tongue caused by decreased muscle tone; with the tongue ultimately touching the posterior pharyngeal wall. Obstruction may be caused also by vomit or blood (regurgitation of gastric contents or trauma), or by foreign bodies. Laryngeal obstruction may be caused by oedema from burns, inflammation or anaphylaxis. Upper airway stimulation

may cause laryngeal spasm. Obstruction of the airway below the larynx is less common, but may arise from excessive bronchial secretions, mucosal oedema, bronchospasm, pulmonary oedema or aspiration of gastric contents.

Recognition of airway obstruction Airway obstruction can be subtle and is often missed by healthcare professionals, let alone by lay people. The 'look, listen and feel' approach is a simple, systematic method of detecting airway obstruction.

- Look for chest and abdominal movements.
- Listen and feel for airflow at the mouth and nose. In partial airway obstruction, air entry is diminished and usually noisy. Inspiratory stridor is caused by obstruction at the laryngeal level or above. Expiratory wheeze implies obstruction of the lower airways, which tend to collapse and obstruct during expiration. Other characteristic sounds include the following:
  - Gurgling is caused by liquid or semisolid foreign material in the main airways.
  - Snoring arises when the pharynx is partially occluded by the soft palate or epiglottis.
  - Crowing is the sound of laryngeal spasm.

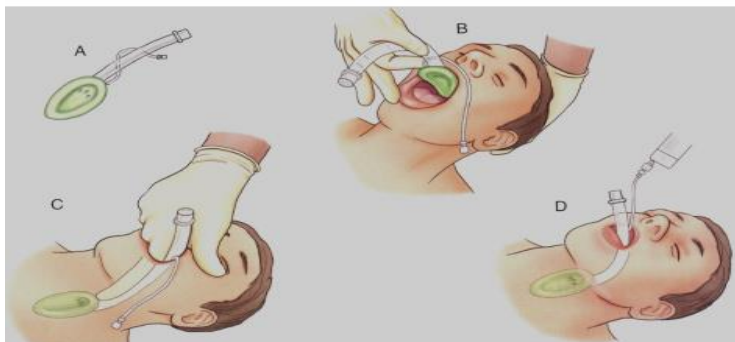
In a patient who is making respiratory efforts, complete airway obstruction causes paradoxical chest and abdominal movement, often described as 'see-saw breathing'. As the patient attempts to breathe in, the chest is drawn in and the abdomen expands; the opposite occurs during expiration. This is in contrast to the normal breathing pattern of synchronous movement upwards and outwards of the abdomen (pushed down by the diaphragm) with the lifting of the chest wall. During airway obstruction, other accessory muscles of respiration are used, with the neck and the shoulder muscles contracting to assist movement of the thoracic cage. Full examination of the neck, chest and abdomen is required to differentiate the paradoxical movements that may mimic normal respiration. The examination must include listening for the absence of breath sounds in order to diagnose complete airway obstruction reliably; any noisy breathing indicates partial airway obstruction. During apnoea, when spontaneous breathing movements are absent, complete airway obstruction is recognised by failure to inflate the lungs during attempted positive pressure ventilation. Unless airway patency can be re-established to enable adequate lung ventilation within a period of a very few minutes, neurological and other vital organ injury may occur, leading to cardiac arrest.

### **Alternative Airway Devices**

- **Laryngeal mask airway**
- **King laringian tube airway**
- **ProSeal LMA**
- **I-gel airway**
- **Esophageal – tracheal Combitube**
- **Tracheal intubation**
- **Cricothyroidotomy and cricothyroidostomy**

**Laryngeal mask airway (LMA)** An LMA is a device that functions intermediately between an OPA and a tracheal tube and does not require direct visualization of the airway for insertion. The LMA is available in sizes for neonates, infants, young children, older children, and small, average, and large adults. The LMA consists of a tube fitted with an oval mask and an inflatable rim. The laryngeal mask airway comprises a wide-bore tube with an elliptical inflated cuff designed to seal around the laryngeal opening. It is easier to insert than a tracheal tube. During CPR, successful ventilation is achieved with the LMA in 72—98% of cases. Ventilation using the LMA is more efficient and easier than with a bag-mask. When an LMA can be inserted without delay it is preferable to avoid bagmask ventilation altogether. When used for intermittent positive pressure ventilation, provided high inflation pressures (>20 cm H<sub>2</sub>O) are avoided, gastric inflation can be minimised. In comparison with bag-mask ventilation, use of a self-inflating bag and LMA during cardiac arrest reduces the incidence of regurgitation. In comparison with tracheal intubation, the perceived disadvantages of the LMA are the increased risk of aspiration and inability to provide adequate ventilation in patients with low lung and/or chestwall compliance. The ability to ventilate the lungs adequately while continuing to compress the chest may be one of the main benefits of a tracheal tube. There are remarkably few cases of pulmonary aspiration reported in the studies of the LMA during CPR.

### Laryngeal Mask Airway



- A. Laryngeal mask airway (LMA) with the cuff inflated**
- B. LMA placement into the pharynx**
- C. LMA placement using the index finger as a guide**
- D. LMA in place with cuff overlying pharynx**

### The King Laryngeal Tube Airway.

- The king LT-D airway is a single lumen supraglottic airway device.
- It consists of a curved tube with a proximal cuff and a distal cuff.
- Along the tube is an orientation /radiograph line that is used when inserting the device and for locating the device on a radiograph.
- The proximal cuff occludes the oropharynx whereas the distal tube occludes the esophagus.



#### Indications

- Unconscious patient
- Patient with an absent gag reflex
- Failure of less invasive airway measures

Inability to intubate when airway protection is needed

#### Contraindications

Patient with an intact gag reflex

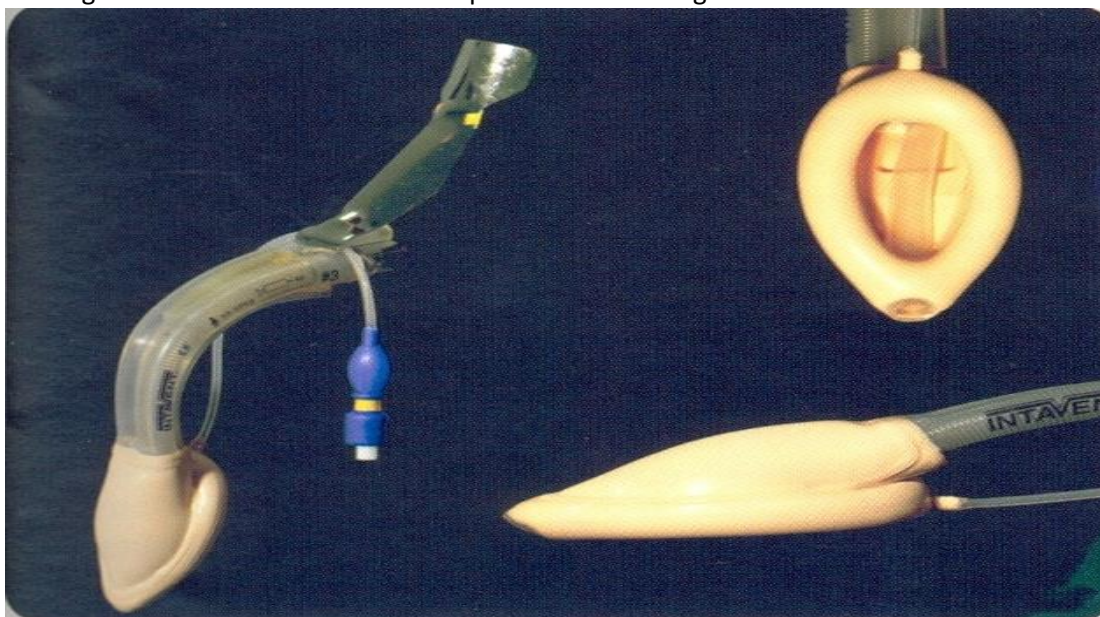
Patient with known or suspected esophageal disease

Patient known to have ingested a caustic substance

Patient with injury to the throat or neck

#### **ProSeal LMA**

The ProSeal LMA has been studied extensively in anaesthetised patients. It has several attributes that, in theory, make it more suitable than the classic LMA for use during CPR: improved seal with the larynx enabling ventilation at higher airway pressures, the inclusion of a gastric drain tube enabling venting of liquid regurgitated gastric contents from the upper oesophagus and passage of a gastric tube to drain liquid gastric contents, and the inclusion of a bite block. The ProSeal LMA has potential weaknesses as an airway device for CPR: it is slightly more difficult to insert than a classic LMA, it is not available in disposable form and is relatively expensive, and solid regurgitated gastric contents will block the gastric drainage tube. Data are awaited on its performance during CPR.





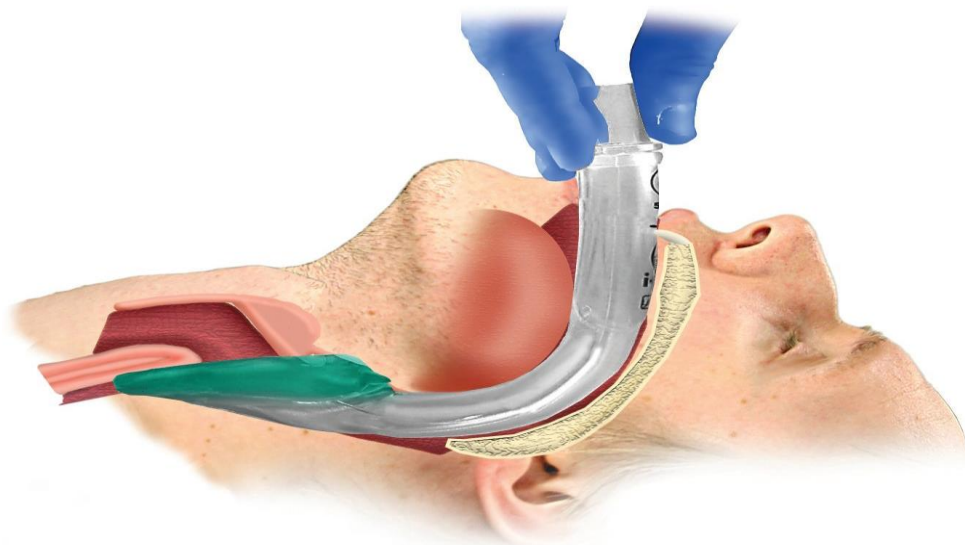
- It has several attributes that, make it more suitable than the original LMA for use during CPR:
  - improved seal with the larynx enabling ventilation at higher airway pressures (commonly up to 35 - 40 cm H<sub>2</sub>O),
  - the inclusion of a gastric drain tube enabling venting of liquid regurgitated gastric contents from the upper oesophagus and passage of a gastric tube to drain liquid gastric contents, and the inclusion of a bite block.
- The higher seal pressures achieved with the PLMA may enable ventilation volume to be maintained during uninterrupted chest compressions.

**Intubating LMA.** The intubating LMA (ILMA) is valuable for managing the difficult airway during anaesthesia, but it has not been studied during CPR. Although it is relatively easy to insert the ILMA, reliable, blind insertion of a tracheal tube requires considerable training and, for this reason, it is not an ideal technique for the inexperienced provider.

### I-Gel Airway

- The I-Gel is a relatively new supraglottic airway.
- The cuff is made of thermoplastic elastomer gel and does not require inflation; the stem of the I-Gel incorporates a bite block and a narrow oesophageal drain tube
- It is easy to insert, requiring only minimal training and a laryngeal seal pressure of 20 - 24 cm H<sub>2</sub>O can be achieved.





### Esophageal-Tracheal Tube

- The esophageal-tracheal tube is an advanced airway alternative to endotracheal intubation. This device provides adequate ventilation comparable to an ET tube.
- The combitube is called a dual-lumen airway because two separate tubes have been joined together with separate airflow passages.

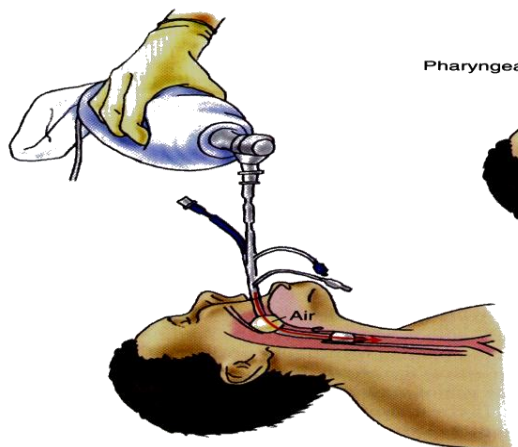
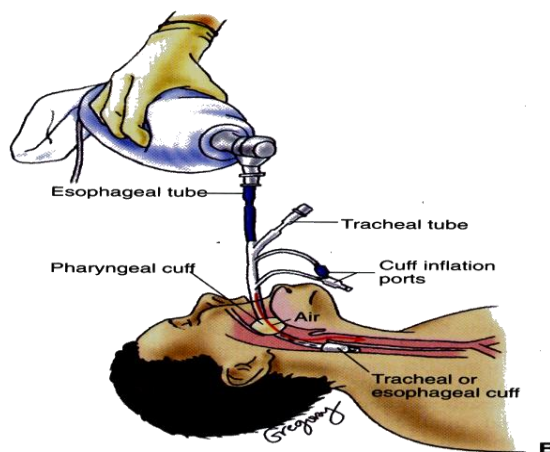
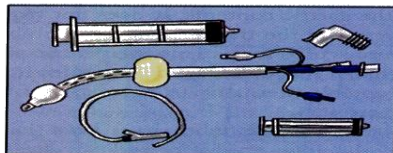
#### Contraindications

Patient with an intact gag reflex

Patient with known or suspected esophageal disease

Patient known to have ingested a caustic substance

Suspected upper airway obstruction due to laryngeal foreign body or pathology



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The Esophageal-Tracheal Combitube, which is commonly called the combitube, allows for the ventilation of the lungs and it reduces the risk of the aspiration of the gastric contents. It does not require the visualization of the vocal cords (i.e., blind insertion) to ventilate the trachea.

It is acceptable to use the esophageal-tracheal tube as an alternative to an ET tube for airway management in cardiac arrest.

## Indications

- Difficult face mask fit (beards, absence of teeth)
- Patient in whom intubation has been unsuccessful and ventilation is difficult
- Patient in whom airway management is necessary but the healthcare provider is untrained in the technique of visualized orotracheal intubation

## Tracheal Intubation

### Indications

- Inability of the patient to protect his or her own airway due to the absence of protective reflex (e.g., coma, respiratory and/or cardiac arrest)
  - Inability of the rescuer to ventilate the unresponsive patient with less invasive methods
  - Present or impending airway obstruction respiratory failure (e.g., inhalation injury, severe asthma, exacerbation of chronic obstructive pulmonary edema, severe flail chest or pulmonary contusion)
  - When prolonged ventilatory support is required.
- Essential equipment for tracheal intubation
- Laryngoscope blades - generally a curved Macintosh blade. Several sizes are available, but a size 3 will be adequate for most patients.
  - Laryngoscope handle - check the light source and battery regularly and just before use .
  - Cuffed tracheal tubes - a selection should be available appropriate to the size of the patient. An 8 mm internal diameter tube is suitable for an adult male and a 7 mm internal diameter tube for a female.
  - Sizes 6, 7 and 8 mm will generally cover the immediate needs of all adults. Availability of smaller tracheal tubes will be helpful for patients with conditions causing narrowing of the upper airway.
  - 10-ml syringe for cuff inflation.
  - Equipment for confirming correct placement of the tracheal tube ( waveform capnography, exhaled CO<sub>2</sub> detector, esophageal detector device).

Water-soluble lubricating jelly; o Magill's forceps;

Introducers: either a gum elastic bougie or a semirigid stylet;

Tape or bandage to secure tube in position;

Suction apparatus with a wide-bore rigid suction end (e.g. Yankauer) and a range of smaller flexible catheters.

Bag-mask device with supplemental oxygen and reservoir

Bite-block or oral airway

- There are two types of laryngoscope blades: straight blade is also referred to as the Miller, Wisconsin, or Flagg blade.
- Curved Macintosh blade

### Post-intubation procedures

- After successful intubation, connect the tracheal tube (via a catheter mount if necessary) to a ventilating device, e.g. self-inflating bag, and ventilate with the highest oxygen concentration available.
- Inflate the cuff of the tracheal tube just sufficiently to stop an air leak during inspiration.

- Confirm correct placement of the tracheal tube using clinical assessment AND a technique for secondary confirmation - waveform capnography is the most reliable secondary technique (see below).
- Continue ventilation with a high concentration of oxygen until ROSC and oxygen saturations are recordable.
- Secure the tube with a bandage or tie. Adhesive tape is not reliable if the face is moist.
- An oropharyngeal airway may be inserted alongside the tracheal tube to maintain the position of the tube, and prevent damage from biting when consciousness returns.

#### Confirmation of Correct Tracheal Tube Placement

- Unrecognised oesophageal intubation is the most serious complication of attempted tracheal intubation.
- Routine use of primary and secondary techniques to confirm correct placement of the tracheal tube will reduce this risk.
- Methods that are used to verify the proper placement of a tracheal tube include the following:
  - Visualizing the passage of the tracheal tube between the vocal cords
  - Auscultating the presence of bilateral breath sounds
  - Determining the absence of sounds over the epigastrium during ventilation
  - Observing adequate chest rise with each ventilation
  - Observing absence of the vocal sounds after the placement of the tracheal tube
  - Measuring the level of end-tidal carbon dioxide (waveform capnography is preferred)
  - Verifying tube placement with the use of an esophageal detector device
  - Obtaining a chest radiograph
- Do not rely exclusively on one method or device to detect and monitor for inadvertent esophageal intubation.

#### Esophageal Detector Device

- The Esophageal detector device creates a suction force at the tracheal end of the tracheal tube, either by pulling back the plunger on a large syringe or releasing a compressed flexible bulb.
- Air is aspirated easily from the lower airways through a tracheal tube placed in the cartilage-supported rigid trachea. When the tube is in the oesophagus, air cannot be aspirated because the oesophagus collapses when aspiration is attempted.
- Carbon dioxide (CO<sub>2</sub>) detector devices measure the concentration of exhaled carbon dioxide from the lungs.
- The persistence of exhaled CO<sub>2</sub> after six ventilations indicates placement of the tracheal tube in the trachea or a main bronchus.
- Confirmation of correct placement above the carina will require auscultation of the chest bilaterally in the mid-axillary lines. Broadly, there are three types of carbon dioxide detector device Disposable colorimetric end-tidal carbon dioxide (ETCO<sub>2</sub>) detectors use a litmus paper to detect CO<sub>2</sub>, and these devices generally give readings of purple (ETCO<sub>2</sub> <0.5%), tan (ETCO<sub>2</sub> 0.5 - 2%) and yellow (ETCO<sub>2</sub> > 2%).
- Non-waveform electronic digital ETCO<sub>2</sub> devices generally measure ETCO<sub>2</sub> using an infrared spectrometer and display the results with a number; they do not provide a waveform graphical display of the respiratory cycle on a capnograph.
- End-tidal CO<sub>2</sub> detectors that include a waveform graphical display (capnograph) are the most reliable for verification of tracheal tube position during cardiac arrest. Studies of waveform capnography to verify tracheal tube position in victims of cardiac arrest demonstrate 100% sensitivity and 100% specificity in identifying correct tracheal tube placement.



### **Cricoid Pressure (Sellick Maneuver)**

Cricoid pressure in nonarrest patients may offer some measure of protection to the airway from aspiration and gastric insufflation during bag-mask ventilation. However, it also may impede ventilation and interfere with placement of a supraglottic airway or intubation.

Cricoid pressure is applying firm pressure to the cricoid cartilage with the thumb and index finger, just lateral to the midline.

### **Procedure for surgical cricothyroidotomy**

- Place the patient supine with the head extended if possible.
- Identify the cricothyroid membrane as the recess just above the cricoid cartilage and below the thyroid cartilage,
- Incise the skin over the membrane and extend the incision through the cricothyroid membrane. Make a vertical incision in the skin and a horizontal one into the cricothyroid membrane; this avoids the superiorly positioned cricothyroid artery.
- Use the reversed handle of a scalpel or tissue expanding forceps to open up the incision in the cricothyroid membrane.
- Insert a suitably sized tracheal tube into the trachea and inflate the cuff, Do not insert the tube too far into the trachea: the carina is not far from here.
- This may occur in patients with extensive facial trauma or laryngeal obstruction caused by edema, e.g. anaphylaxis, or foreign material. In these circumstances, it will be necessary to create a surgical airway below the level of the obstruction.
- Substantial bleeding can occur, surgical cricothyroidotomy provides a definitive airway that can be used to ventilate the patient's lungs until semi-elective intubation or tracheostomy is performed.
- Needle cricothyroidotomy is a much more temporary procedure providing only short-term oxygenation. It requires a wide-bore, non-kinking cannula, a high-pressure oxygen source and may cause serious barotrauma.

### **Ventilation Rates**

<b>Airway device</b>	<b>Ventilation during Cardiac Arrest</b>	<b>Ventilation During Respiratory Arrest</b>
<b>Bag- mask</b>	<b>2 ventilation after every 30 compressions</b>	<b>1 ventilation every 5 to 6 seconds (10 to 12 breaths per minute)</b>
<b>Any advanced airway</b>	<b>1 ventilation every 6 to 8 seconds (8 to 10 breaths per minute)</b>	

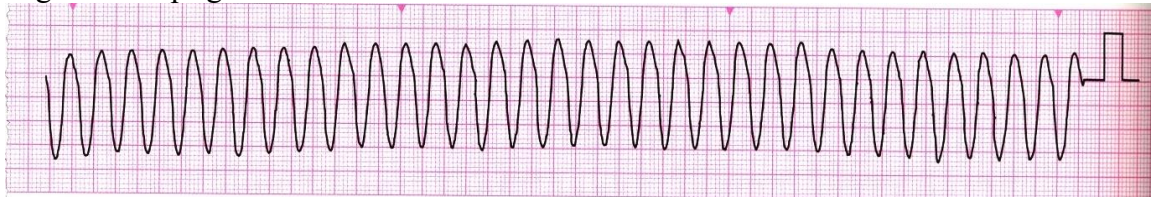
- During CPR the compression-to-ventilation ratio is 30:2. But once an advanced airway is in place (ie, laryngeal mask airway, laryngeal tube, esophageal-tracheal tube, or ET tube), chest compressions are no longer interrupted for ventilations.
- When ventilating through a properly placed advanced airway, give 1 breath every 6 to 8 seconds (approximately 8 to 10 breaths per minute) without trying to synchronize breaths to compressions. Ideally deliver the breath during chest recoil between compressions. Continuously reevaluate compressions and ventilations. Be prepared to make modifications if either is ineffective.
- In this case the patient has a pulse, and compressions are not indicated. Give 1 breath every 5 to 6 seconds (10 to 12 breaths per minute).

### **Ventricular Tachycardia (VT)**

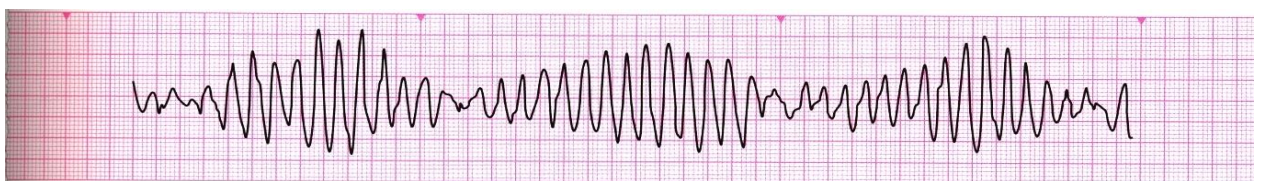
VT exists when three or more ventricular complexes occur in immediate succession at a rate greater than 100beats/min.VT may occur with or without pulses,and the patient may be stable or unstable with this rhythm.

**Common causes of VT include:**

- **Acid-base imbalance**
  - **Acute coronary syndromes**
  - **Cardiomyopathy**
  - **Cocain abuse**
  - **Digitalis toxicity**
  - **Electrolyte imbalance (e.g., hypokalemia, hyperkalemia, hypomagnesemia)**
  - **Mitral valve prolapse**
  - **Trauma (e.g., myocardial contusion, invasive cardiac procedures)**
  - **Tricyclic antidepressant overdose**
  - **Valvular heart disease**
- **Pulseless VT is a medical emergency and treated with immediate defibrillation.**
  - **VT can be classified according to its duration:**
  - **Non –sustained VT is the term used if the arrhythmia lasts < 30 seconds before self-terminating.**
  - **Sustained VT is if the arrhythmia lasts >30 seconds**
  - When the QRS complexes of VT are of the same shape and amplitude, the rhythm is called monomorphic.
  - When the QRS complexes of VT vary in shape and amplitude from beat to beat, the rhythm is called polymorphic VT.
  - In polymorphic VT, the QRS complexes appear to twist from upright to negative or negative to upright and back.

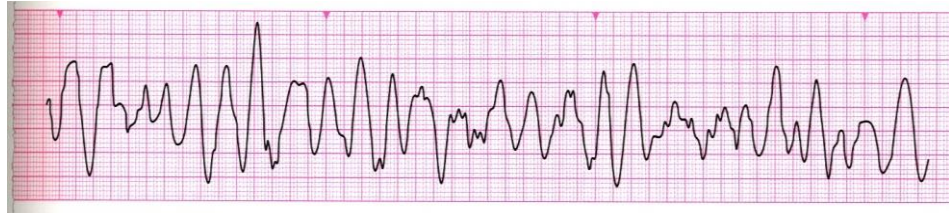


Monomorphic Ventricular Tachycardia QRS complexes are the same shape and amplitude



Polymorphic Ventricular Tachycardia - Torsade de Pointe QRS complexes appear to twist from upright to negative or negative to upright and back

- **Ventricular Fibrillation**  
VF is a chaotic rhythm that begins in ventricles. In VF, there is no organized depolarization of the ventricles. The ventricular muscle quivers, as a result, there is no effective myocardial contraction and no pulse.
- Factors that increase the susceptibility of the myocardium to fibrillate include:
  - Acute coronary syndromes
  - Antiarrhythmics and other medications
  - Dysrhythmias
  - Electrolyte imbalance
  - Environmental factors (e.g., electrocution)
  - Increased sympathetic nervous system activity
  - Vagal stimulation



Coarse ventricular fibrillation - ventricular fibrillation (VF) with waves that are 3 mm high or more is called “coarse” VF.



Fine ventricular fibrillation - ventricular fibrillation (VF) with low amplitude waves (less than 3 mm) is called “fine” VF.

The major points about CPR really haven’t changed. Keep going with good compressions at 30:2, maximizing compression time, with no pauses longer than 10 seconds. However, they have Successful defibrillation depends on the electrical and metabolic state of the myocardium. From a metabolic perspective VF depletes more of the cardiac energy stores—adenosine triphosphate (ATP)— per minute than does normal sinus rhythm.

Prolonged VF will exhaust the energy stores of ATP in the myocardium, particularly- in the cardiac pacemaker cells. The longer VF persists, the greater the myocardial deterioration as energy stores become exhausted. In a heart stunned into electrical silence by a defibrillatory shock, no spontaneous contractions will resume if the fibrillating myocardium has consumed all its energy stores.

When ATP is depleted and cellular functions are disrupted, shocks are more likely to convert VF to asystole than to a spontaneous rhythm because no "fuel" remains to support spontaneous depolarization in the pacemaker tissues or the contracting myocardium. With depleted reserves of energy, any postshock *asystole* or *agonal rhythms* will be permanent, not temporary.

VF of short duration is much more likely to respond to a shock delivered soon after VF starts. An important objective for resuscitation and any effort to improve outcome is to shorten the interval between the onset of VF and the first shock. In *Emergency Cardiovascular Care (ECC) Guidelines 2000*, the American Heart Association (AHA) recommended that all patients who have a VF arrest out of hospital receive shocks in <5 minutes; a goal for patients who have an in-hospital VF arrest is < 3minutes.

### **Drugs for VF/ Pulseless VT**

This case involves these drugs:

- Epinephrine
- Amiodarone
- Lidocaine
- Magnesium sulfate

### **Epinephrine**



- IV/IO dose: 1mg (10ml of 1:10000 solution) administered every 3 to 5 minutes during resuscitation. Follow each dose with 20ml flush, elevate arm for 10 to 20 seconds after dose
- Higher dose: higher doses (up to 0.2 mg/kg) may be used for specific indications ( $\beta$ -blocker or calcium channel blocker overdose)
- Continuous infusion: initial rate: 0.1 to 0.5 mcg/kg per minute, titrate to response
- Profound Bradycardia or Hipotension 2 to 10 mcg per minute infusion, titrate to patient response

**Amiodarone** is an antiarrhythmic that blocks sodium channels, inhibits symphatetic stimulation, and bloks potassium channels as well as calcium channels.

- Amiodarone is the first antiarrhythmic given during cardiac arrest and has been demonstrated to improve the rate of return of spontaneous circulation (ROSC) and hospital admission in adults with refractory VF/pulseless VT.
- Amiodarone 300 mg IV/IO bolus, then consider an additional 150 mg IV/IO once.
- If amiodarone is not available, may administer lidocaine.

### Lidicaine

- Cardiac Arrest From VF/VT
- Lidocaine may be considered if amiodarone is not available.
- Initial dose: 1 to 1.5 mg/kg IV/IO first dose, then 0.5 to 0.75 mg/kg IV/10 at 5- to 10-minute intervals, to a maximum dose of 3 mg/kg

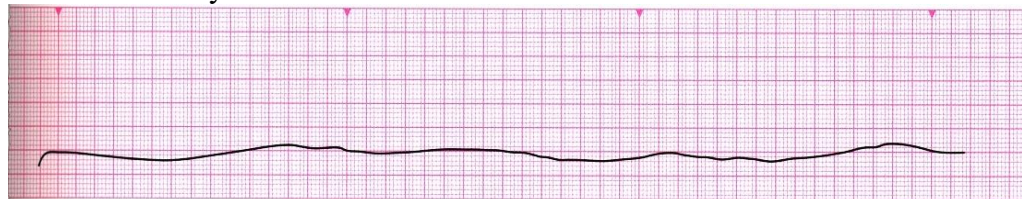
For refractory VF may give additional 0.5 to 0.75 mg/kg IV push, repeat in 5 to 10 minutes; maximum 3 doses or total 3 mg/kg.

**Magnesium sulfate** for torsades de pointes associated with a long QT interval, loading dose 1 to 2 g IV/IO diluted in 10 mL of D5W given as IV/10 bolus, typically over 5 to 20 minutes

- Routine administration of magnesium sulfate in cardiac arrest is not recommended unless torsades de pointes is present.
- Search for and treat any treatable underlying cause of cardiac arrest.

### Asystole (Cardiac Standstill)

- Asystole, which is also called ventricular asystole, is a total absence of ventricular electrical activity. There is no ventricular rate or rhythm, no pulse, and no cardiac output.
- Some atrial electrical activity may be evident. If atrial electrical activity is present, the rhythm is called “P wave” asystole or ventricular standstill.
  - When asystole is observed on a cardiac monitor, confirm that the patient is unresponsive and has no pulse, and then begin CPR.
  - Additional care includes establishing vascular access, considering the possible causes of the arrest, administering epinephrine, and possibly inserting an advanced airway.



Asystole



**„P wave” asystole or ventricular standstill (atrial electrical activity is present)  
Pulseless Electrical Activity (PEA)**

- PEA is a clinical situation, not a specific dysrhythmia. PEA exists when organized electrical activity (other than VT) is observed on the cardiac monitor, but the patient is unresponsive and not breathing, and the pulse cannot be felt. PEA was formerly called electromechanical dissociation.
- The term was changed from EMD to PEA because research using ultrasonography and indwelling pressure catheters revealed that the electrical activity seen in some of these situations is indeed associated with mechanical contractions; however, the contractions are simply too weak to produce a palpable pulse or measurable blood pressure.



the rhythm shown is a sinus tachycardia; however, if no pulse is associated with the rhythm, the clinical situation is termed pulseless electrical activity (PEA) .

PEA has a poor prognosis unless the underlying cause can be rapidly identified and appropriately managed. Treatment includes high-quality CPR, establishing vascular access, the administration of epinephrine, and an aggressive search for possible causes of the situation and the possible insertion of an advanced airway.

- Not too fast. Maximum compression rate of 120. They don't want compressions going too fast, as there is evidence that quality decreases with more than 120 compressions per minute. The new target is 100-120 compressions a minute (instead of *at least* 100)
- Not too deep. Maximum compression depth 6 cm. The new target is 5-6cm in adults (instead of *at least* 5cm)
- 10 breaths a minute. If an advanced airway (endotracheal tube, LMA, etc) is in place, everyone gets just 10 breaths a minute. This applies to children and infants as well

CAB is the alphabet. No change, just a statement of support. Start with compressions to reduce the delay to first compression.



Compression only CPR is not endorsed. If you are a trained provider, keep giving rescue breaths. They state, “Our confidence in the equivalence between chest compression-only and standard CPR is not sufficient to change current practice”

Give epinephrine early in non-shockable rhythms. Based on one observational study, they say if you are going to give epinephrine, you should probably get epinephrine on board as soon as possible in non-shockable rhythms. (For a full review of the evidence for epinephrine, see this post.)

Naloxone added to the guidelines. In patients with known or suspected opioid addiction who are not breathing normally but have a pulse, it is reasonable for trained lay rescuers and BLS providers to administer naloxone. The doses listed are 2mg intranasally or 0.4mg IM. They suggest standard following the standard ALS algorithm if the patient does not have a pulse, but state that providing a dose of naloxone may be reasonable based on the possibility that the patient may be in respiratory distress.

### **Capnography**

Waveform capnography receives a little more attention than in the past. They say:

- Waveform capnography is the most reliable method to confirm and continuously monitor tracheal tube placement
- An end-tidal less CO<sub>2</sub> than 10 mmHg after 20 minutes is associated with extremely low chance of survival, but should not be used alone in the decision to stop resuscitation
- Waveform capnography can be used to monitor the ventilation rate
- Waveform capnography can be used to monitor the quality of CPR. (High quality compressions should produce an end-tidal CO<sub>2</sub> of at least 12-15 mmHg).

**A rise in end-tidal CO<sub>2</sub> can be used as an early indication of ROSC**

### **Special circumstances**

Pregnancy

- No more tilting the patient. It is no longer recommended to use a wedge or attempt to laterally tilt the patient because this will interfere with the quality of CPR. Just manually displace the uterus to the left. (Most people have been teaching this already)
- Perimortem C-section is still recommended after 4 minutes of CPR with no ROSC. However, if the mother will clearly not survive, such as in non-survivable trauma, they recommend starting the c-section immediately

**A**

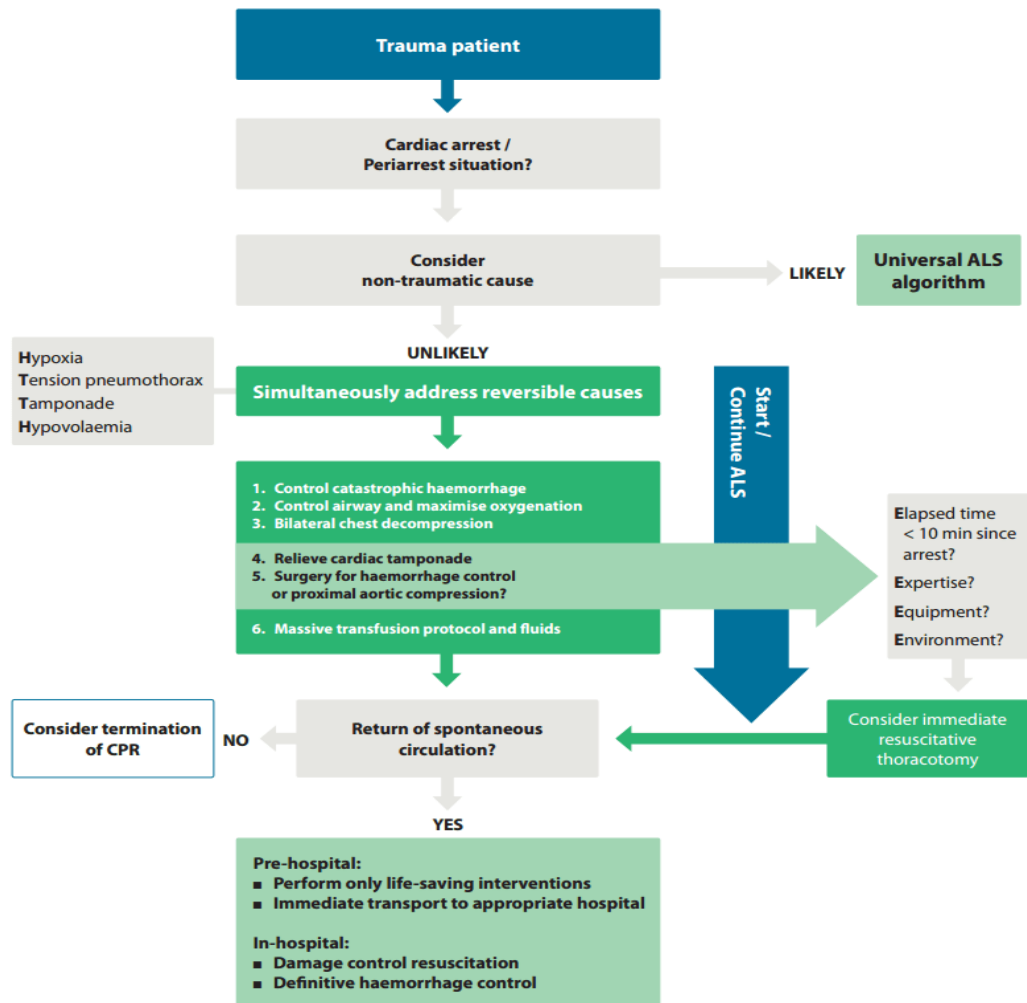


**B**



- Trauma
- They have added a specific algorithm for the traumatic arrest. The immediate actions are addressing the key reversible causes: hypoxia, tension pneumothorax, tamponade, and hypovolemia.

# Traumatic Cardiac Arrest

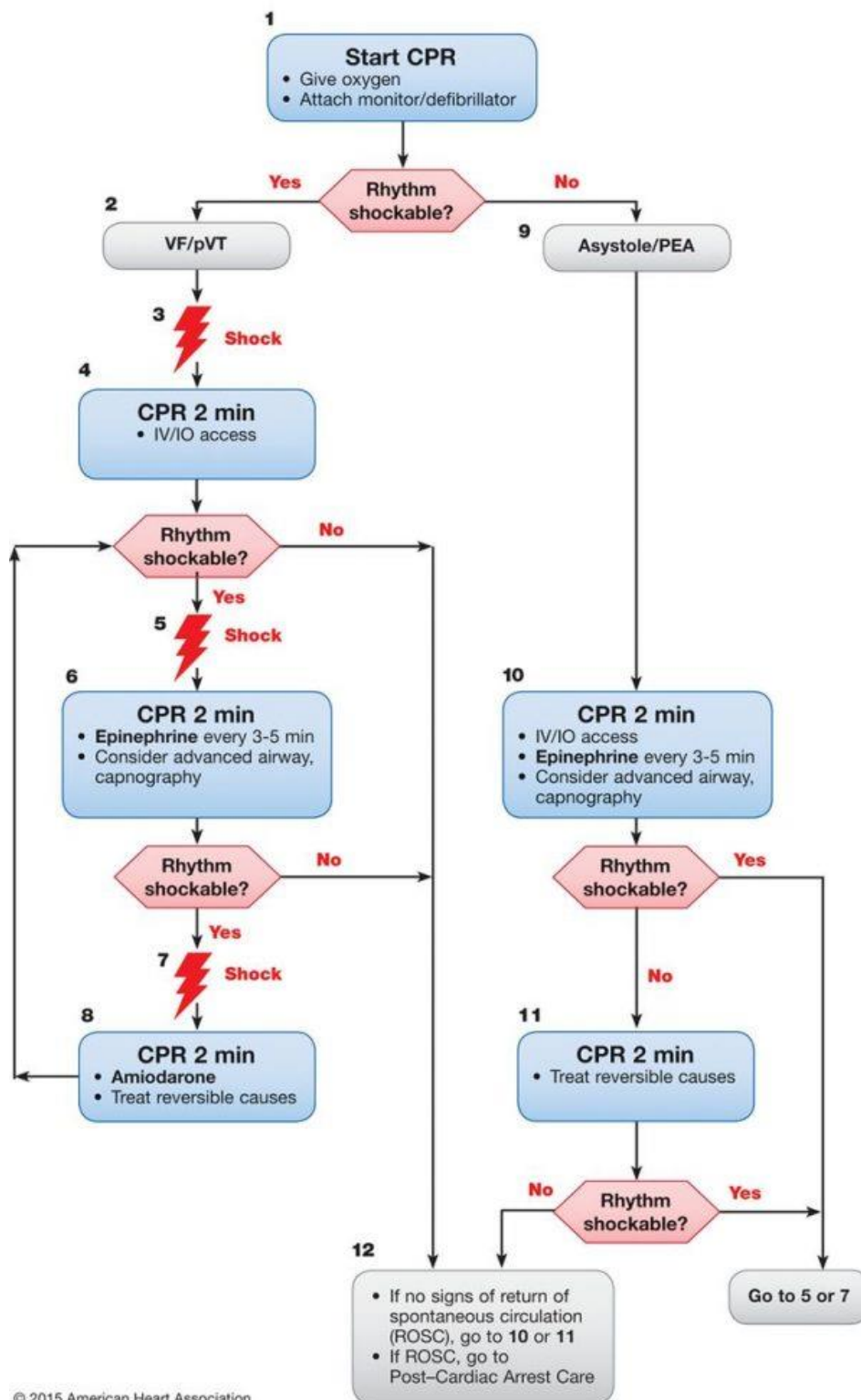


## Pediatrics

CPR should be 15:2 if multiple providers are available, but 30:2 if there is only a single provider.

Do not use compression only CPR. Stick with standard CPR (with rescue breaths) because of high the rate of asphyxia. However, if the rescuer is unwilling to provide rescue breaths, advise compression only CPR. When an advanced airway in place, give 10 breaths a minute (same as adults) no matter what the patient's age.

## Adult Cardiac Arrest Algorithm—2015 Update



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CPR Quality
<ul style="list-style-type: none"> <li>• Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.</li> <li>• Minimize interruptions in compressions.</li> <li>• Avoid excessive ventilation.</li> <li>• Rotate compressor every 2 minutes, or sooner if fatigued.</li> <li>• If no advanced airway, 30:2 compression-ventilation ratio.</li> <li>• Quantitative waveform capnography               <ul style="list-style-type: none"> <li>- If PETCO<sub>2</sub> &lt;10 mm Hg, attempt to improve CPR quality.</li> </ul> </li> <li>• Intra-arterial pressure               <ul style="list-style-type: none"> <li>- If relaxation phase (diastolic) pressure &lt;20 mm Hg, attempt to improve CPR quality.</li> </ul> </li> </ul>
Shock Energy for Defibrillation
<ul style="list-style-type: none"> <li>• <b>Biphasic:</b> Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.</li> <li>• <b>Monophasic:</b> 360 J</li> </ul>
Drug Therapy
<ul style="list-style-type: none"> <li>• <b>Epinephrine IV/IO dose:</b> 1 mg every 3-5 minutes</li> <li>• <b>Amiodarone IV/IO dose:</b> First dose: 300 mg bolus. Second dose: 150 mg.</li> </ul>
Advanced Airway
<ul style="list-style-type: none"> <li>• Endotracheal intubation or supraglottic advanced airway</li> <li>• Waveform capnography or capnometry to confirm and monitor ET tube placement</li> <li>• Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions</li> </ul>
Return of Spontaneous Circulation (ROSC)
<ul style="list-style-type: none"> <li>• Pulse and blood pressure</li> <li>• Abrupt sustained increase in PETCO<sub>2</sub> (typically ≥40 mm Hg)</li> <li>• Spontaneous arterial pressure waves with intra-arterial monitoring</li> </ul>
Reversible Causes
<ul style="list-style-type: none"> <li>• Hypovolemia</li> <li>• Hypoxia</li> <li>• Hydrogen ion (acidosis)</li> <li>• Hypo-/hyperkalemia</li> <li>• Hypothermia</li> <li>• Tension pneumothorax</li> <li>• Tamponade, cardiac</li> <li>• Toxins</li> <li>• Thrombosis, pulmonary</li> <li>• Thrombosis, coronary</li> </ul>

## CONCLUSIONS

Advanced Cardiac Life Support (ACLS). -These terms refer to attempts at restoration of spontaneous circulation using basic CPR plus advanced airway management, endotracheal intubation, defibrillation, and intravenous medications.

The Cardiac Arrest Algorithm is the most important algorithm to know for adult resuscitation.

The algorithm consists of the 2 pathways for a cardiac arrest:

- A shockable rhythm (VF/pulseless VT) displayed on the left side of the algorithm
- A nonshockable rhythm (asystole/PEA) displayed on the right side of the algorithm.

## **REFERENCES**

American Heart Association. Advanced Cardiovascular Life Support 2016

European Resuscitation Council Guidelines for Resuscitation 2015, section 3.

Gh.Ciobanu. Resuscitarea Cardiorespiratorie și Cerebrală, 2015, Vol.1-2,