

دکتر نازنین نوری رودسری متخصص طب اورژانس استادیار گروہ طب اورژنس دانشگاہ علوم پزشکی گیلان

ADVANCED CARDIAC LIFE SUPPORT (ACLS)

EPIDEMIOLOGY

Every year, approximately 6.8 to 8.5 million persons throughout the world sustain cardiac arrest. About 70% of cardiac arrests occur out of hospital. The proportion of cardiac arrest patients who are treated varies from 54.6% (United States) to 28.3% (Asia). Approximately half of cardiac arrest victims are <65 years old.

Ventricular tachydysrhythmias are the initiating event in approximately 80% of patients with out-of-hospital primary cardiac arrest. During ambulatory ECG monitoring of 157 witnessed cardiac arrests, Bayés de Luna et al documented 70% with ventricular tachycardia (VT) and VF, 13% with torsades de pointes, and 17% with bradydysrhythmias. Untreated VF deteriorates to asystole in approximately 15 minutes. For patients with sudden cardiac arrest, the rate of survival declines rapidly by 7% to 10% for each minute without defibrillation. If delay to

defibrillation exceeds 12 minutes, survival approaches 0% to 5%.

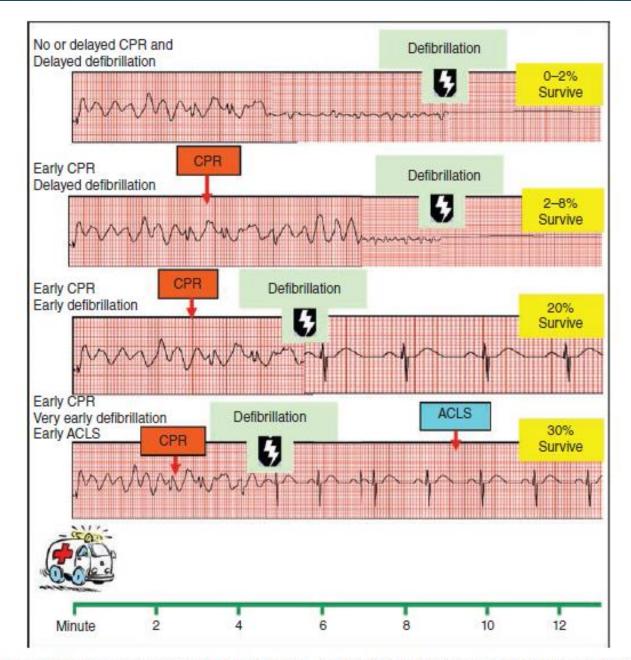


FIGURE 24-1. Incremental survival benefits by the links in the Chain of Survival. ACLS = Advanced Cardiac Life Support. [Reproduced, with permission, from the National Resuscitation Council, Singapore.]







Improved survival can only occur if structured emergency care systems allow trained providers to access the patient rapidly and deliver the appropriate treatment in a timely fashion. Delays in initiating the various links weaken the chain and adversely affect the next link, resulting in a decreased chance of a good outcome for the patient.

Based on the Chain of Survival, cardiac arrest can be divided into three separate timesensitive phases: the electrical, circulatory, and metabolic phases. These phases affect treatment efficacy. The electrical phase is present in the first **5** minutes of arrest, where the cardiac system is most amenable to defibrillation if a shockable rhythm is present. The circulatory system consists of the next 15 minutes after arrest, when high-quality chest compressions and epinephrine provide the greatest benefit. The metabolic phase is >20 minutes after arrest and is marked by electrolyte and acid-base disorders; epinephrine delivered >20 minutes after arrest is associated with decreased cerebral blood flow, worse neurologic outcome, and increased cardiac ischemia.

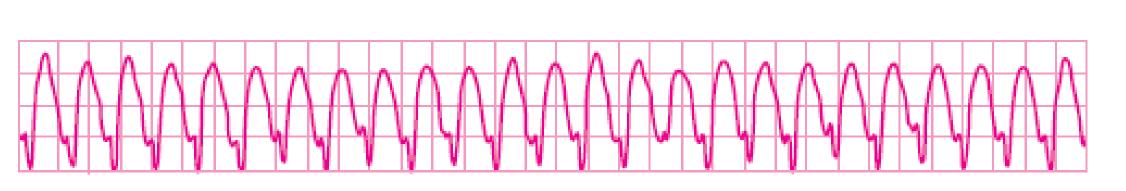


FIGURE 23-2. Ventricular tachycardia.

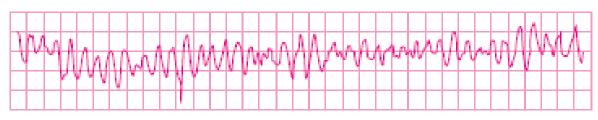


FIGURE 24-5. Ventricular fibrillation.

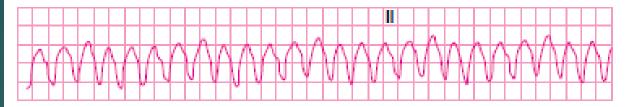


FIGURE 24-6. Pulseless ventricular tachycardia.

FIGURE 24-7. Asystole.

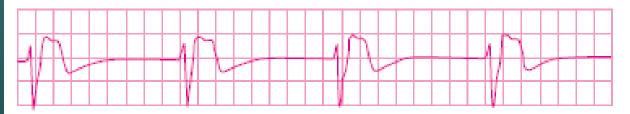
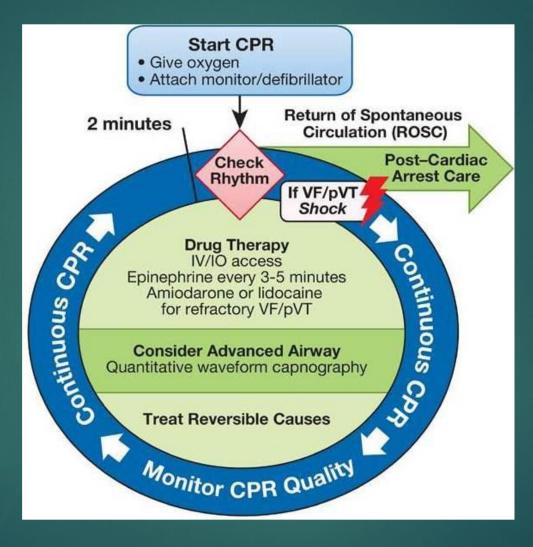
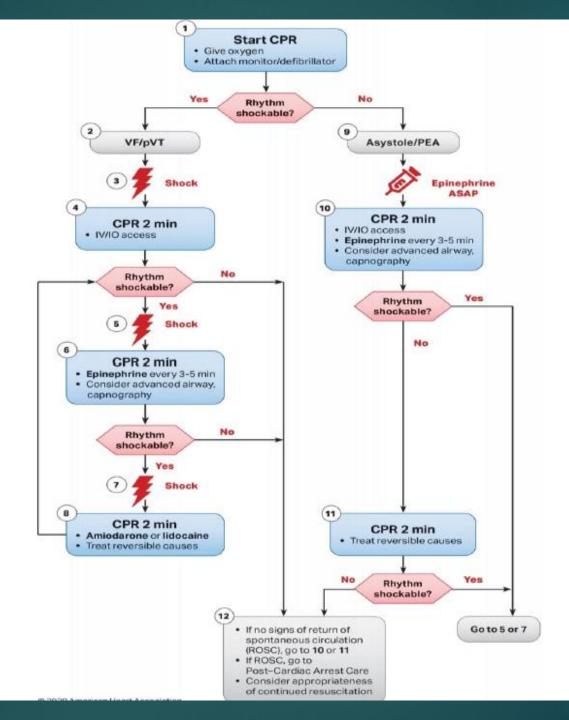


FIGURE 24-8. Pulseless electrical activity.





CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- · Quantitative waveform capnography
 - If PETCO₂ <10 mm Hg, attempt to improve CPR quality.
- Intra-arterial pressure
 - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality.

Shock Energy for Defibrillation

- Biphasic: 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.
- Monophasic: 360 J

Drug Therapy

- Epinephrine IV/IO dose: 1 mg every 3-5 minutes
- Amiodarone IV/IO dose: First dose: 300 mg bolus. Second dose: 150 mg.
 -OR-

Lidocaine IV/IO dose: First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in PETCO₂ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- Hypovolemia
- Hypoxia
- Hydrogen ion (acidosis)
- Hypo-/hyperkalemia
- Hypothermia

- Tension pneumothorax
- Tamponade, cardiac
- Toxins
- Thrombosis, pulmonary
- Thrombosis, coronary

PATIENT SELECTION

The indications for defibrillation include ventricular fibrillation and pulseless ventricular tachycardia Defibrillation is not "jumpstarting the heart" and is thus not indicated for asystole or pulseless electrical activity. It is contraindicated for sinus rhythm, conscious patients with a pulse, or when there is danger to the operator or others (e.g., from a wet patient or wet surroundings). Electrical cardioversion is indicated for patients with ventricular tachycardia, supraventricular tachycardia, atrial flutter, or atrial fibrillation, who are hemodynamically unstable as a consequence of the rhythm. It may also be considered after unsuccessful pharmacologic therapy for the previously mentioned arrhythmias, even if the patient remains hemodynamically stable. Electrical cardioversion should be synchronized, which means the electric impulse will be timed with the patient's intrinsic QRS complexes, to minimize the risk of inducing ventricular fibrillation.

DEFIBRILLATION

Early defibrillation is the most effective modality for return of spontaneous circulation (ROSC).

Defibrillation

is the therapeutic use of electricity in cardiac arrest to depolarize the entire myocardium to eliminate ventricular fibrillation or nonperfusing ventricular tachycardia so that coordinated contractions can resume. It should be performed in close coordination with CPR of the cardiac arrest patient.

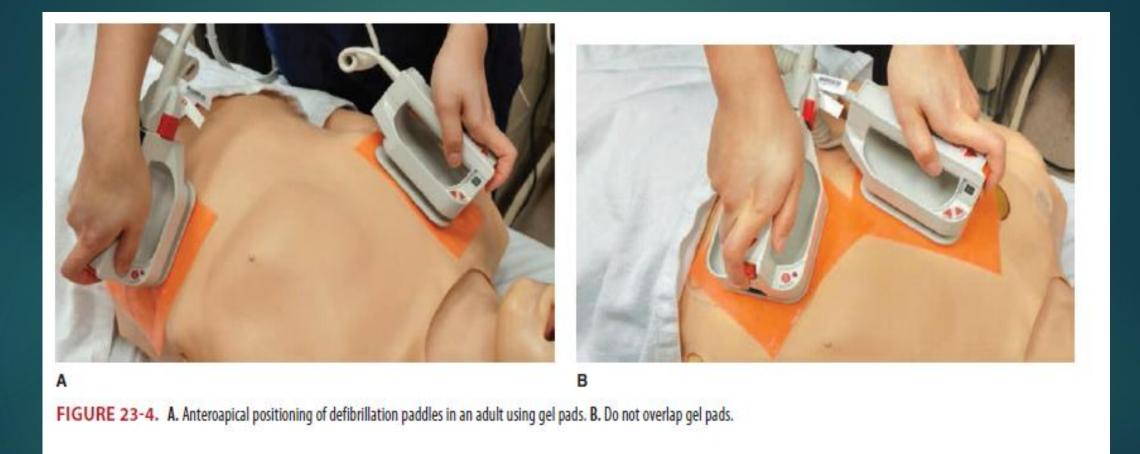
Electrical cardioversion

is the application of a synchronized electrical impulse to convert a still-perfusing tachydysrhythmia back to a normal sinus rhythm. Cardioversion may also be performed pharmacologically or nonpharmacologically for the stable patient.









Defibrillation and Cardioversion

Indications

Defibrillation Ventricular fibrillation Pulseless ventricular tachycardia Cardioversion (usually reserved for unstable rhythms) Ventricular tachycardia with a pulse Supraventricular tachycardia Atrial fibrillation Atrial fibrillation

Contraindications

Defibrillation Presence of a pulse Asystole or pulseless electrical activity Obvious signs of death Valid do-not-resuscitate order Cardioversion Arrhythmias due to digitalis toxicity Sinus tachycardia

Complications

Chest wall burns Shock of a health care worker Myocardial tissue injury

Equipment



Cardiac monitor/defibrillator (other supportive equipment not shown)

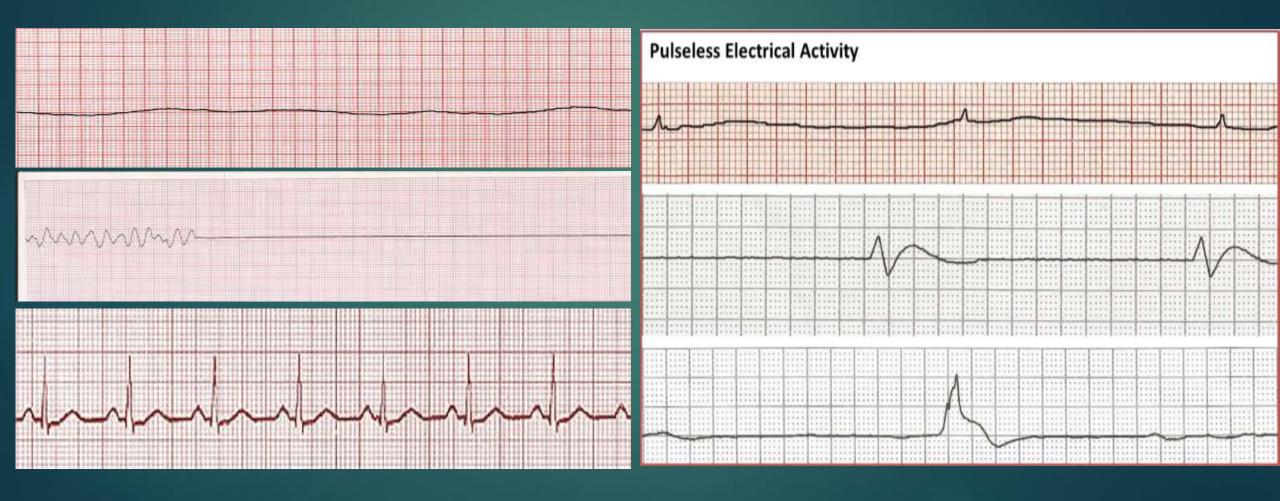
Review Box 12.1 Defibrillation and cardioversion: indications, contraindications, and equipment.

Ventricular flutter and ventricular fibrillation (VF) are arrhythmias that represent severe derangements of the heartbeat that can terminate fatally or produce significant brain damage within 3 to 5 minutes unless corrective measures are undertaken promptly CPR and defibrillation are the only therapies associated with improved survival in patients with VF/pVT.

Asystole

Asystole represents complete cessation of myocardial electrical activity. Although asystole may occur early in cardiac arrest as consequence of progressive bradycardia, asystole generally represents the end-stage rhythm after prolonged cardiac arrest caused by VF or PEA. Because the potential exists for an organized rhythm or VF to appear as asystole in a single lead—if the rhythm vector is completely perpendicular to the lead vector-asystole should be confirmed in at least two limb leads.

Asystole & pea



Although asystole may be difficult to distinguish from extremely fine VF, routine countershock of asystole has not been shown to improve survival. Treatment of asystole requires general resuscitation measures, including CPR, intubation with assisted ventilation, IV access, and repeated administration of vasopressors.

. A, Fine ventricular fibrillation. B, Idioventricular rhythm with wide and slow ventricular response. The absence of pulse confirms PEA. C, Markedly slower idioventricular... Continue Reading



TABLE 8.4

Diagnosis and Treatment of Common Causes of Pulseless Electrical Activity

CAUSE	DIAGNOSIS	PALLIATIVE THERAPY	DEFINITIVE THERAPY
Hypovolemia	Response to volume infusion	Volume infusion	Hemostasis if hemorrhage
Нурохіа	Response to oxygenation	Oxygenation, assisted ventilation	Treat underlying cause
Cardiac tamponade	Echocardiogram; jugular venous distention	Pericardiocentesis	Thoracotomy and pericardiotomy
Tension pneumothorax	Asymmetric breath sounds, tracheal deviation	Needle thoracostomy	Tube thoracostomy
Hypothermia	Rectal temperature		Warm peritoneal or thoracic lavage, venoarterial ECMO
Pulmonary embolus	Risk factors or evidence of deep venous thrombosis	Venoarterial ECMO	Lytic therapy, pulmonary embolectomy
Drug overdose	History of drug ingestion	Drug-specific	Drug-specific
Hyperkalemia	History of renal failure or elevated serum potassium level	Calcium chloride, insulin and glucose, sodium bicarbonate	Hemodialysis
Acidosis	Arterial blood gas	Hyperventilation, sodium bicarbonate	Treat underlying cause



FIGURE 18-21. Two examples of polymorphic ventricular tachycardia. A. Polymorphic ventricular tachycardia. B. Polymorphic ventricular tachycardia of the torsades de pointes subtype, with the characteristic pattern of progressively changing QRS complex amplitude and direction.

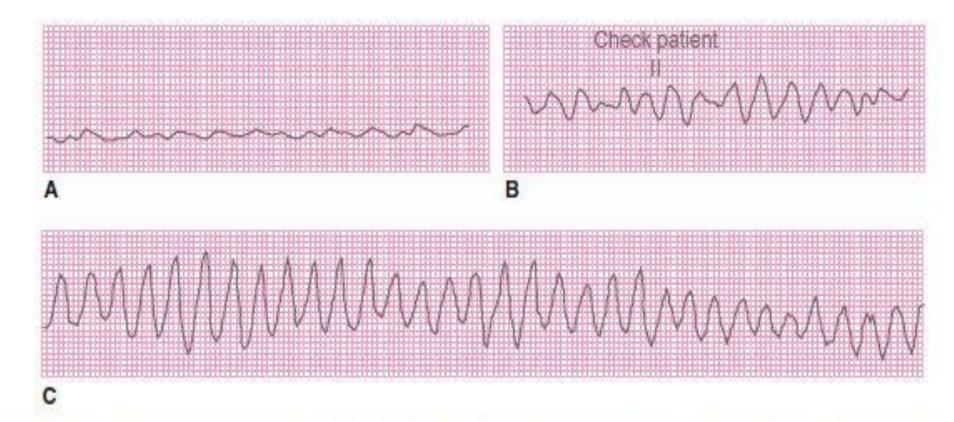
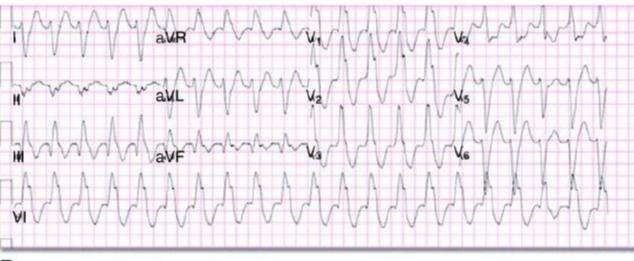


FIGURE 18-23. Three examples of ventricular fibrillation. A. Fine amplitude. B. Coarse amplitude. C. Coarse amplitude mimicking ventricular tachycardia.

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В

A

Figure 12-1 Ventricular dysrhythmias. A, Ventricular fibrillation. B, Ventricular tachycardia.





Figure 12.4 High-quality cardiopulmonary resuscitation is essential in the resuscitation of victims of sudden cardiac arrest. Push hard to a depth of 2+ inches and +fast at a rate of 100 compressions per minute. Minimize interruptions and avoid overventilating the patient. Allow full recoil of the chest between compressions.

CHEST COMPRESSIONS

• The recommended chest compression rate is 100 to 120 per minute, updated from 100 per minute.

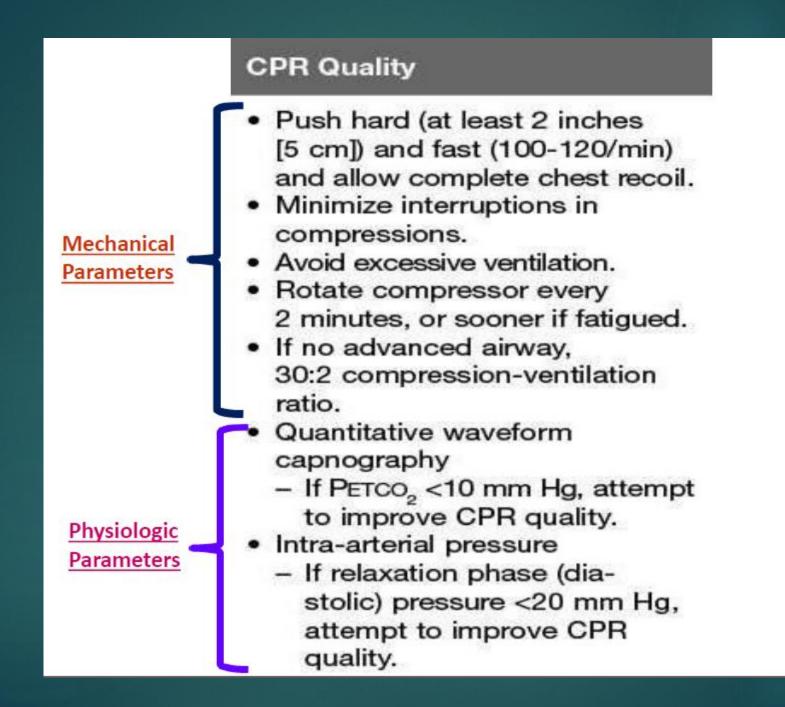
• Chest compression depth should be 5 to 6 cm, not >6 cm. Chest compression ratio should be >60 and as close to 90 as possible.

• There does not appear to be an advantage to asynchronous or interpolated breaths as long as the highest possible compression ratio is maintained.

• The compression-to-breath ratio is 30:2. If an adjunctive or definitive airway is in place, provide <u>10 breaths per minute</u>.

BMV without an advanced airway device may not allow adequate ventilation in all patients during resuscitation from cardiac arrest and does not protect against pulmonary aspiration of orogastric secretions.

Either BMV or an advanced airway strategy may be considered during CPR for adult cardiac arrest in any setting



DEFIBRILLATION



Assess patient responsiveness, breathing, and circulation. Check for a pulse for <10 seconds. Call for help and have the code cart delivered to the bedside.



If there is no pulse, begin CPR at a rate of >100 compressions per minute with a ratio of 30 compressions to 2 ventilations. Avoid interruptions in CPR, PUSH HARD, and PUSH FAST.



Apply the electrodes to the patient's chest. Place the sternal electrode below the clavicle, to the right of the sternum. Place the apical electrode in the midaxillary line at the fifth intercostal space.

Check the rhythm on the monitor. If there is a shockable rhythm (i.e., VF or pulseless VT), prepare for immediate defibrillation.





Select the appropriate energy for the initial shock. For biphasic defibrillators, a default energy of 200 J is appropriate (see text for details). For monophasic machines, use 360 J. Make sure that "SYNC" is turned off.

As the "CHARGE" button is depressed, loudly announce "I'm clear, you're clear, everybody's clear!" and make sure that no caregivers are in contact with the patient.



Once the energy has been selected and the decision to defibrillate confirmed, press the "CHARGE" button on the defibrillator.



Check once again to make sure that everyone is clear, and then depress the "SHOCK" button to defibrillate the patient.



Resume CPR immediately and continue for 5 cycles/ 2 minutes. Additional interventions such as IV/IO access and airway management may be pursued but should not interfere with continuous CPR.



After 2 minutes of CPR, reassess the patient and the rhythm. Refer to the text and algorithms in this chapter for additional information.

Figure 12.11 Defibrillation. CPR, Cardiopulmonary resuscitation; IO, intraosseous; IV, intravenous; VF, ventricular fibrillation; VT, ventricular tachycardia.

•Shock-refractory VF/pVT refers to VF or pVT that persists or recurs <u>after ≥ 1 shocks</u>.

• An antiarrhythmic drug alone is unlikely to pharmacologically convert VF/pVT to an organized perfusing rhythm

Amiodarone **or** lidocaine may be considered for VF/pVT that is unresponsive to defibrillation.

OXYGENATION AND CAPNOGRAPHY

• Provide 100% fraction of inspired oxygen (Fio2) during cardiac arrest, with oxygen saturation titrated to >94% after ROSC.

• End-tidal carbon dioxide (CO2) can be used to monitor for ROSC. Low end-tidal

CO2 (<10 mm Hg) after 20 minutes is associated with low likelihood of survival.

• Give amiodarone 300 mg intravenously; a further dose of 150 mg may be given after five defibrillation attempts.

•Lidocaine 100 mg intravenously; An additional bolus of lidocaine 50 mg may also be given after five defibrillation attempts

There is insufficient evidence to support or refute the routine use of a β -blocker early (within the first hour) after ROSC.

□There is insufficient evidence to support or refute the routine use of lidocaine early (within the first hour) after ROSC.

TABLE 8.3

Indicators of Inadequate Blood Flow During Cardiopulmonary Resuscitation

MONITORING TECHNIQUE	INDICATOR
Carotid or femoral pulse	Not palpable
CPP	<15 mm Hg
Arterial relaxation (diastolic) pressure	<20–25 mm Hg
PETco ₂	<10 mm Hg
Scv0 ₂	<40%

CPP, Coronary perfusion pressure; *PETco*₂, partial pressure of CO_2 in exhaled air at the end of expiration; *Scvo*₂, central venous oxygen saturation.

•• Amiodarone or lidocaine (lignocaine) may not provide added benefit to defibrillation.

•• Vasopressin has been removed from the Advanced Cardiac Life Support algorithm.

• Routine use of β-blockers after cardiac arrest is not recommended, with benefits for ROSC only demonstrated through animal studies and case reports.

•• Steroids may provide some benefit when bundled with vasopressin and epinephrine in in-hospital cardiac arrest, although routine use is not recommended.

از توجه شما سپاسگزارم

