



Episode 12 – 2010 ACLS Update

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Links in the chain of survival

- Immediate recognition of cardiac arrest and activation of EMS system
- Early CPR with emphasis on good compressions
- Rapid defibrillation
- Effective advanced cardiac life support
- Integrated post-cardiac arrest care (including therapeutic hypothermia)

EMS systems that effectively implement these can achieve witnessed VF cardiac arrest survival of almost 50%

Cardio-cerebral resuscitation (CCR)

New concept emphasizing high quality, minimally-interrupted CPR to maximize blood flow to the brain

C-A-B is the new algorithm, as opposed to the previously taught A-B-C, because of the realization that arterial oxygen saturation remains high for the first 5-10min of a cardiac arrest, that recoil of the chest during chest compressions improves oxygenation, that delaying a definitive airway *increases* survival, and that high quality compressions increases survival

Caveat: because children usually die as a result of asphyxial and respiratory complications, ventilations should be emphasized for them, and so the ABCs are still the most appropriate algorithm for the majority of paediatric cardiac arrests

BLS changes from 2005 Guidelines:

- Compressions **before** giving breaths, and pulse checks 10 seconds or less
- Faster compressions: **at least** 100 compressions/min, i.e. 30-compression cycle in 18 seconds or less
- Deeper compressions: adults – **at least** 2 inches & children/infants **at least** 1/3 the depth of chest

3-phase model of resuscitation

1. Electrical phase – first few minutes after the cardiac arrest: disorganized cardiac activity with minimal ischemia, requiring prompt defibrillation
2. Circulatory phase – 6-10min: ischemic insult and injury developing, requiring uninterrupted compressions before defibrillation is attempted, as the heart needs to be 'primed' for defibrillation with compressions
3. Metabolic phase – 10min and after: therapeutic hypothermia and vasopressors may help in post-cardiac arrest syndrome

Carbon dioxide: Capnography

End-tidal CO₂ (continuous quantitative waveform capnography) serves 3 purposes in initial resuscitation:

1. Helps confirm endotracheal tube placement and monitoring displacement
2. Helps monitor quality of CPR (if ET_{CO}₂ <10mmHg, patient likely needs faster or harder CPR – although epinephrine may lead to decrease in ET_{CO}₂ level)
3. Detects ROSC (return of spontaneous circulation) with abrupt and sustained increase >40mmHg

Pearls

- Bedside ultrasound should only be used in restricted situations during cardiac arrest resuscitation, in order to determine whether the heart is still beating in light of a potentially reversible cause of cardiac arrest (eg, pneumothorax, cardiac tamponade, hypovolemic shock), and which would prompt you to continue resuscitative efforts
- Minimize pre-shock pause between the last compression and defibrillation to increase success
- Ventilation at high respiratory rates (25 breaths per minute) is common during resuscitation from cardiac arrest. The guidelines say to deliver ventilations at a regular rate **1 breath every 6 to 8 seconds (8 to 10 breaths/minute) without interruption** of chest compressions. Ventilating faster will decrease venous return and decrease cardiac output especially in patients with hypovolemia or obstructive airway disease.
- The upper limit of the rate of sinus tachycardia is determined by the 220 minus the age of the patient
- The starting dose for synchronized cardioversion in the setting of atrial fibrillation is now to start at 120-200J (instead of the previous recommendation of 100-120J)
- Remember the Hs and Ts, along with their respective treatment:
 - Hypovolemia (fluid, pressors), hypoxia (oxygen), hydrogen ion (NaHCO₃ only in certain situations), hypo/hyperkalemia (potassium, or calcium, insulin and glucose), hypothermia (re-warming)
 - Tension pneumothorax (needle thoracostomy) cardiac tamponade (pericardiocentesis), toxin (antidote), thrombosis (thrombolytic if pulmonary embolus; thrombolytics or cath lab if myocardial infarction)

Pharmacology

- *Atropine for Asystole*: **not** recommended in asystole and PEA anymore, not due to negative studies, but rather to the realization that the evidence was always weak and therefore shouldn't be considered positive.
- *Atropine for Bradycardia*: may increase ischemia in the setting of an MI, doesn't work in transplant patients and is unlikely to work in patients with 2nd degree type II and 3rd degree block

- *Adenosine for Wide-complex Tachycardias*: on top of stable *narrow*-complex regular tachycardia, the 2010 guidelines recommend using it in stable *wide*-complex regular (i.e. monomorphic) tachycardia as well as a diagnostic approach, given that it might unmask SVT with aberrancy but won't do much for VT
 - Beware of the irregular (i.e. polymorphic) wide-complex tachycardia, which may mask an alternative pathway such as Wolff-Parkinson-White's, in which case adenosine may precipitate VF
 - Reduce the dose of adenosine (to 3mg) in post-cardiac transplant patients, those taking dipyridamole or carbamazepine, and when administered via a central vein access
- *Amiodarone or procainamide for Stable Vtach*: the former has been shown in small studies to terminate dysrhythmias in only about 30% of cases, and latter, as much as 80%; patients receiving either should be monitored for QT prolongation and hypotension, and should never receive both medications due to the risk of refractory bradycardia and/or hypotension
- *NaHCO₃ (sodium bicarbonate) in cardiac arrest*: should not be used routinely because of resultant paradoxical intracellular acidosis, but may be considered in known or suspected hyperkalemia (eg, dialysis patient) TCA overdose, or documented pre-existing metabolic acidosis as a cause for cardiac arrest
- *Inotropes for Bradycardia*: dopamine (10µg/kg/min) or epinephrine (2-10µg/min) drips may now be given as an alternative to transthoracic pacing, as a bridge to transvenous pacing

Post-cardiac arrest care: "Getting the pulse back is just the beginning"

- Avoid SpO₂ of 100% because it leads to increased production of harmful free radicals and increases mortality – aim instead for 94%
- Avoid hyperventilation, which leads to hypocarbia and resultant cerebral vasoconstriction, which will worsen brain injury due to the decreased cerebral blood flow; also avoid hypotension
- Activate emergency PCI for post-arrest STEMI, and strongly consider for suspected high-risk NSTEMI; PCI and therapeutic hypothermia can and should be done together; thrombolysis should be considered on an individual basis when PCI is not available; there is a theoretical increased bleeding risk when thrombolysis is combined with therapeutic hypothermia
- Therapeutic hypothermia (TH) – a good resource: www.NYChypothermia.org
 - Reduces or stops destructive pathways such as cerebral edema and free radicals formation, which are caused by reperfusion after ROSC; has been shown to increase survival to hospital discharge with good neurological outcome (NNT of 6!)
 - Although strong evidence (i.e. RCTs) is available only for VF/VT as presenting rhythms, the 2010 guidelines recommend TH in PEA and asystole based on positive observational studies
 - TH should not be done in cardiac arrests resulting from trauma because it will worsen coagulopathy in a hypovolemic and bleeding patient, as well as in vasopressor-resistant severe cardiogenic shock (controversial) because the cold will make the myocardium less responsive to drugs

- Cooling should be started as soon as possible in the ED (not in the ICU) with cooled IV saline boluses (30cc/kg or 2L) with pressure bags, and ice packs to the axillae, groin and neck, even if the patient will go to the cath lab; goal of 32-34°C within 6-8hrs
- Patients need to be adequately sedated (midazolam, fentanyl), but paralysis should only be used to prevent shivering (which often stops below 33.5°C) because it may mask seizure activity, which is rendered more likely by ischemic brain injury
- Consider the following initial ventilation settings: Tidal volume of 6-8mL/kg with rate adjusted to obtain an end-tidal CO₂ of 35-40mmHg and FiO₂ adjusted to obtain arterial saturation of 94%; also prevent hyperventilation and hypocarbia
- Prognostication cannot be accomplished until 72hrs after the cardiac arrest, which is later than what used to be done in the pre-cooling era

Key live-saving steps in 2010 Guidelines: The 5 C's of ACLS

1. Compressions (hard, fast & uninterrupted)
2. Cardiovert (defibrillate early)
3. Capnography (CO₂ monitoring)
4. Cooling (therapeutic hypothermia)
5. Cath (PCI)

Interesting articles

- *For the guidelines, please refer to The American Heart Association, 2010 Guidelines for CPR and ECC, Circulation, Volume 122, Issue 18, supplement 3, November 2, 2010*
- IV ALCS drugs (vs. no IV drugs) does not lead to increased survival to hospital discharge – Olasveengen TM et al. Intravenous drug administration during out-of-hospital cardiac arrest: a randomized trial. JAMA 2009 Nov 25;302(20):2222-9
- Hands-on defibrillation may be coming in a hospital near you – Lloyd, M. Hands-On Defibrillation: An Analysis of Electrical Current Flow Through Rescuers in Direct Contact With Patients During Biphasic External Defibrillation. Circulation 2008;117:2510-2514
- Standard cardiopulmonary resuscitation versus active compression-decompression cardiopulmonary resuscitation with augmentation of negative intrathoracic pressure for out-of-hospital cardiac arrest: a randomized trial. Lancet Jan 19th, 2011