

ACLS Guidelines 2015 Update – Cardiac Arrest Controversies Part 1

- The recommended *chest compression rate* is 100-120 per minute which is updated from the at least 100/min.
- The recommended *chest compression depth* is 5-6cm or just over 2 inches, but not more than 6cm as too deep can be harmful.
- Use Audiovisual devices such as metronomes and compression depth analyzers which can be used to optimize CPR quality.
- The routine use of impedence threshold devices (ITDs) alone or mechanical chest compression devices alone are not recommended, however in out of hospital situations where manual compressions are difficult due to physical space limitations, mechanical devices may be useful.
- A recent RCT suggests that the use of the ITD plus active compression decompression CPR is associated with improved neurological intact survival for patients with out of hospital cardiac arrest.

- *ECMO or ECPR* may be considered for selected patients with refractory cardiac arrest where a reversible cause of cardiac arrest is suspected.
- *Vasopressin* has been removed from the algorithm altogether, and an emphasis on EARLY administration of epinephrine is stressed.
- Ultrasound has been added as an additional method for helping to confirm ROSC and for confirming ETT placement.
- Use maximum inspired oxygen during CPR and then after ROSC, titrate oxygen to an oxygen saturation of 94% rather than continuing maximum oxygen delivery.
- A low end tidal CO2 in intubated patients after 20 minutes of CPR is associated with a very low likelihood of survival, and this factor should be used in combination with other factors to help determine when to terminate resuscitation.
- The routine use of lidocaine after ROSC is not recommended, however the initiation or continuation of lidocaine may be considered immediately after ROSC from ventricular fibrillation or pulseless ventricular tachycardia.
- *Emergency PCI* is recommended for all patients with STEMI *AND* for hemodynamically or electrically unstable patients without ST elevation for whom a cardiovascular lesion is suspected.

• Do Targeted Temperature Management recommendations have been updated with new evidence suggesting that a range of temperatures between 32 and 36 may be acceptable to target in the post cardiac arrest period, and an emphasis on fever prevention and treatment is stressed.

CCR rather than CPR, CAB rather than ABC

Before we jump into the controversies it's important to understand the basic theory of how best to achieve meaningful survival in the cardiac arrest patient. The concept of Cardiocerebral Resuscitation rather than Cardiopulmonary Resuscitation emphasizing high quality, minimally-interrupted chest compressions to maximize blood flow to the brain, an important theoretical determinant of long term survival.

Circulation-Airway-Breathing as opposed to the classic Airway-Breathing-Circulation is how we should approach these patients 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 continuous compressions improves survival.

In Toronto, the survival rate overall for cardiac arrest has improved from 2% in 2004 to 13% in 2015, probably related to improved early defibrillation and improved quality of chest compressions. The survival rate for Ventricular Fibrillation in Toronto has risen from 8% in 2004 to 35% in 2015. Caveat: because children usually die as a result of asphyxia and respiratory complications, early ventilations should be emphasized in the pediatric population.

Determinants of Good Quality CCR

The recommended rate of chest compressions of 100-120 per minute, aiming for 110 per minute with audiovisual feedback such as a metronome (which is easily activated on most defibrillators), and the recommended depth of compressions is 5-6cm with visual feedback (which is also easily activated on most defibrillators). These recommendations are based on observational studies from the **Resuscitation Outcomes Consortium** (ROC) papers.



The guidelines suggest a compression to breath ratio of 30:2, however in Seattle, the survival rates for cardiac arrest overall have remained around 19-20%, and for Ventricular Fibrillation are 44%, the highest in the world, possible, in part due to a revision of the compression to breaths ratio. Rather than the recommended 30:2 compressions:breath ratio, in Seattle, the paramedics provide continuous chest compressions. On the 10th compression, the provider handling the breathing gives a single 500cc breath, in between compressions, without any pause in compressions. The only time that Seattle paramedics pause compressions is to shock the patient, and even then, once they have determined that the patient is in ventricular fibrillation, while continuing compressions, they count down out loud and pause compressions for only a single, hands-Off beat to re-administer a shock every 15 compressions until signs of ROSC are apparent. Experimental data out of England shows that, using gloves, the maximal energy delivered to a provider providing hands-on compressions without any pause for defibrillation is less than the average house hold electrical current, suggesting that hands-on defibrillation may be safe.



In order to **minimize compression pauses for pulse checks**, our experts recommend two approaches:

1. Have a *dedicated experienced* provider feeling for a manual femoral pulse *during* chest compressions so that as soon as a pause in chest compressions to assess for cardiac rhythm occurs at the end of 2 minutes of compressions, the pulse can be rapidly assessed along with the cardiac rhythm in less than 5 seconds (as apposed to less than 10 seconds as recommended in the guidelines).

2. Do not check for the pulse at all! Chest compressions should continue until the patient shows signs of life such as respiratory effort, or until your end tidal CO2 monitor shows *persistent* elevations. The argument here is that palpating for a pulse has been shown to be insensitive, not specific and have poor inter-rater reliability. One study showed that rescuer pulse palpation was only 78% accurate. Even if a pulse is felt, this does not guarantee adequate perfusion to vital organs. Dr. Morrison suggests stopping chest compressions *only* to assess for defibrillation and to defibrillate.

Simulation Training and Cardiac Arrest Teams

Simulation training plays an important role in optimizing performance of cardiac arrest teams. Like NASCAR tire replacement teams, the cardiac arrest team should be proficient at performing their specific dedicated tasks. Observational data from the Resucitation Outcomes Consortium (ROC) show that the larger the cardiac arrest team the better the outcome. Where as previous guidelines focused on developing the skills of the leader of the resuscitation, current trends are to develop skills of each individual on the cardiac arrest team.

The Role of Capnography in Cardiac Arrest

ETCO2 capnography is *mandated* in The Guidelines to be used to:

1. Confirm endotracheal intubation with an increase in ETCO2

2. Assess the quality of chest compressions with an ETCO2 goal of 12-15; an ETCO2 of less than 10 may be an indicator of sub-optimal chest compressions

3. An increase of ETCO2 35-40 may indicate adequate tissue oxygenation and can be used to confirm return of spontaneous circulation (ROSC), however our experts recommend continuing CPR after the initial spike of ETCO2.

Pitfall: stopping chest compressions immediately after an ETCO2 of 35-40 is recognized

The Role of Chest Compression Devices in Cardiac Arrest

In multiple randomized control trials chest compression devices have *not* shown superiority to good quality manual chest compressions. The Guidelines recommend considering the use of compression devices in environments where manual compressions may be difficult or impossible due to physical constraints (eg: small cabins of some rescue helicopters).

Impedance Threshold Devices (ITD) are circulatory devices which are applied to the endotracheal tube (ETT) or the bag-valve-mask (BVM) and increase perfusion to the right side of the heart. The ROC performed a study where they randomized patients to ITD vs sham ITD which showed no superiority of the ITD.

Antiarrhythmic Medications in Refractory Ventricular Fibrillation

There has never been an antiarrhythmic medication that has shown any long term survival benefit in cardiac arrest

Amiodarone 300mg IV bolus is still recommended as the antiarhythmic of choice for refractory ventricular fibrillation. In one study from the 1990's comparing Amiodarone to placebo in refractory ventricular fibrillation, a higher rate of ROSC was achieved. In a study from the NEJM out of Toronto, pre-hospital ventricular fibrillation patients were randomized to amdiodarone vs lidocaine, and amidorone showed a higher rate of ROSC compared to lidocaine, however the death rate in hospital in the amiodarone group was higher than that of the lidocaine group. Overall, survival at discharge from hospital was similar in both groups. The Amiodarone vs Lidocaine vs Placebo (ALPS) Trial is currently ongoing with a projected release in early 2016.

Dual Shock Therapy for Refractory Ventricular Fibrillation

Dual Shock Therapy involves defibrillating patients who are refractory to multiple defibrillations with 2 defibrillators at the same time or within seconds of each other, one with pads set up in the traditional anterior positions and the other set up with anteriorposterior pad positions.



A recent observational case series showed improved rates of ROSC with dual shock therapy with one patient surviving to hospital discharge. An RCT in 1989 by Bardy randomized patients with refractory ventricular fibrillation to dual vs single shock therapy and found no significant difference in ROSC. It is unknown based on current evidence whether the improved rates of ROSC with dual shock therapy are due to chance alone or due to the increase in energy or multiple vectors of energy delivered to the heart.

Dual shock therapy for refractory ventricular fibrillation is not recommended in The Guidelines.

Despite the lack of evidence that dual shock therapy improves survival, our experts recommend attempting it when other avenues have been exhausted in the patient with refractory ventricular fibrillation.

Epinephrine and Vasopressin in Cardiac Arrest

There has never been a vasopressor medication that has shown any long term survival benefit in cardiac arrest

Epinephrine has been shown in RCTs to improve rates of ROSC and admission to hospital, however one large pre-hospital study of Japan, while also showing improved rates of ROSC, showed *worse* 1 month survival rates with epinephrine.

Optimizing Epinephrine in Cardiac Arrest

Multiple factors may contribute to the effectiveness of epinephrine in cardiac arrest:

- 1. Timing
- 2. Dose and frequency of administration
- 3. Underlying rhythm and suspected diagnosis

Timing

The Guidelines recommend administering epinephrine as early as possible in the resuscitation. Observational data suggests that the first dose of epinephrine is administered at 22 minutes on average after cardiac arrest. Some experts believe that earlier administration in the first 4 minutes during the 'circulatory phase' of cardiac arrest

may improve long term outcomes, but there is only observational data for this in the literature.

One observational study of cardiac arrest with non-shockable rhythm compared epinephrine given at 1 to 3 minutes with epinephrine given at 3 later time intervals (4 to 6, 7 to 9, and greater than 9 minutes). The study found an association between early administration of epinephrine and increased ROSC, survival to hospital discharge, and neurologically intact survival.

Dose and Frequency of Administration

Some experts believe that the dose of 1mg every 3-5 minutes that is recommended in the guidelines is too high a dose, is too frequently given and may be detrimental. Epinephrine, in the doses used in cardiac arrest, causes cerebral vasoconstriction that may impair tissue oxygenation, brain perfusion and compromise neurological recovery.

Scott Weingart describes titrating epinephrine in cardiac arrest to hemodynamic parameters and end tidal CO2. While end tidal CO2 is an excellent marker for chest compression quality and determining ROSC, titrating epinephrine to end tidal CO2 goals have never been shown to be associated with long term outcomes.

A physiologic measure of brain oxygenation called near-infrared spectroscopy may be a better marker to titrate epinephrine to in cardiac arrest, but currently there is no evidence in the literature to support its use.

Underlying rhythm and suspected diagnosis

In most patients in ventricular fibrillation the primary cause is an MI. Some experts believe that we should lower the dose of epinephrine in patients with ventricular fibrillation as a the primary rhythm during cardiac arrest to minimize the vasoconstriction of the coronary arteries caused by epinephrine given at 1mg every 3-5 minutes.

In patients with non-shockable rhythms (PEA, asystole), early administration of epinephrine may be more beneficial than in patients' whose presenting rhythm is ventricular fibrillation.

Epinephrine vs Vasopressin in ACLS Guidelines 2015 - Cardiac Arrest

Vasopressin is difficult to draw up compared to the pre-filled epinephrine syringes and has not been shown to be superior to epinephrine in terms of survival benefit in cardiac arrest patients for out of hospital cardiac arrest. It has therefore been removed from The Guidelines.

The Guidelines do state that vasopressin may provide some benefit when administered with steroids and epinephrine in treating inhospital cardiac arrests. However, routine use of this bundle is not recommended. This was based on a controversial study in 2013 out of JAMA in Greece with 268 patients showed that the combination of vasopressin, epinephrine and steroids significantly improved survival to discharge with good neurological outcome compared to only epinephinre and placebo. However, our experts suggest that due to methodological flaws in the study, they cannot recommend its routine use as per The Guidelines.

Key References

The full AHA Guidelines in Circulation

2015 AHA Guidelines Highlights

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