
INTRODUCTION

The location, etiology, and treatment of cardiac arrest have changed dramatically, but the overall prognosis following return of spontaneous circulation (ROSC) has not improved **(Nolan et al., 2008)**

The majority of research on cardiac arrest over the past half century has focused on improving the rate of ROSC, and significant progress has been made. However, many interventions improve ROSC without improving long-term survival **(Laver et al., 2004).**

The prognosis for post cardiac arrest patients remains very bleak, not only because of anoxic-ischemic neurological damage, but also because of the “post cardiac arrest syndrome,” a phenomenon often severe enough to cause death before any neurological evaluation **(Mongardon et al., 2011).**

This syndrome includes all clinical and biological manifestations related to the phenomenon of global ischemia-reperfusion triggered by cardiac arrest and return of spontaneous circulation. The frequency and intensity of these complications depend largely on the delay of initial treatment,

the efficiency of resuscitation, and the time elapsed between collapse and return of spontaneous circulation (**Adrie et al., 2005**).

As many as 30% of cardiac arrest survivors will suffer from permanent brain injury. The percentage of survivors from the initial cardiac arrest, that subsequently die, is fairly comparable around the world, around 65-75%. Most of these patients die within the first month after return of spontaneous circulation (ROSC) (**Nolan et al., 2010**).

Less than 10% of patients admitted to the hospital after successfully resuscitated out of hospital cardiac arrest (OHCA) will leave the hospital without major neurological impairments. Poor neurological prognosis because two thirds of patients who survive the early phase will subsequently develop neuro-functional sequelae, which sometimes progress toward a post anoxic vegetative state and delayed death (**Nolan et al., 2008**).

Moreover, this is the only medical situation that enables measurement of clinical acute consequences of global ischemia, targeting simultaneously all tissues and organs (**Laver et al., 2004**).

AIM OF THE ESSAY

The aim of this essay was to review the major concerns and problems regarding the post cardiac arrest syndrome, and how to improve its outcome using the most recent therapeutic strategies.

ETIOLOGY OF CARDIAC ARREST

Although the Advanced Life Support (ALS) cardiac arrest algorithm is applicable to all cardiac arrests, additional interventions may be indicated for cardiac arrest caused by special circumstances. The interventions that unquestionably contribute to improved survival after cardiac arrest are prompt and effective bystander basic life support (BLS), uninterrupted, high-quality chest compressions and early defibrillation for VF (Ventricular Fibrillation)/pVT (Pulseless ventricular tachycardia). Although drugs and advanced airways are still included among ALS interventions, they are of secondary importance to early defibrillation and high-quality, uninterrupted chest compressions (*Jacobs et al., 2011*).

The ALS algorithm distinguishes between shockable and non-shockable rhythms. Each cycle is broadly similar, with a total of 2 min of CPR (Cardio-Pulmonary Resuscitation) being given before assessing the rhythm and where indicated, feeling for a pulse. Adrenaline 1 mg is injected every 3–5 min until ROSC (Return Of Spontaneous Circulation) is achieved. In VF/pVT, a single dose of amiodarone 300 mg is indicated after a total of three shocks and a further dose of 150 mg can be

considered after five shocks (figure 1). The optimal CPR cycle time is not known (*Lexow and Sunde, 2007*).

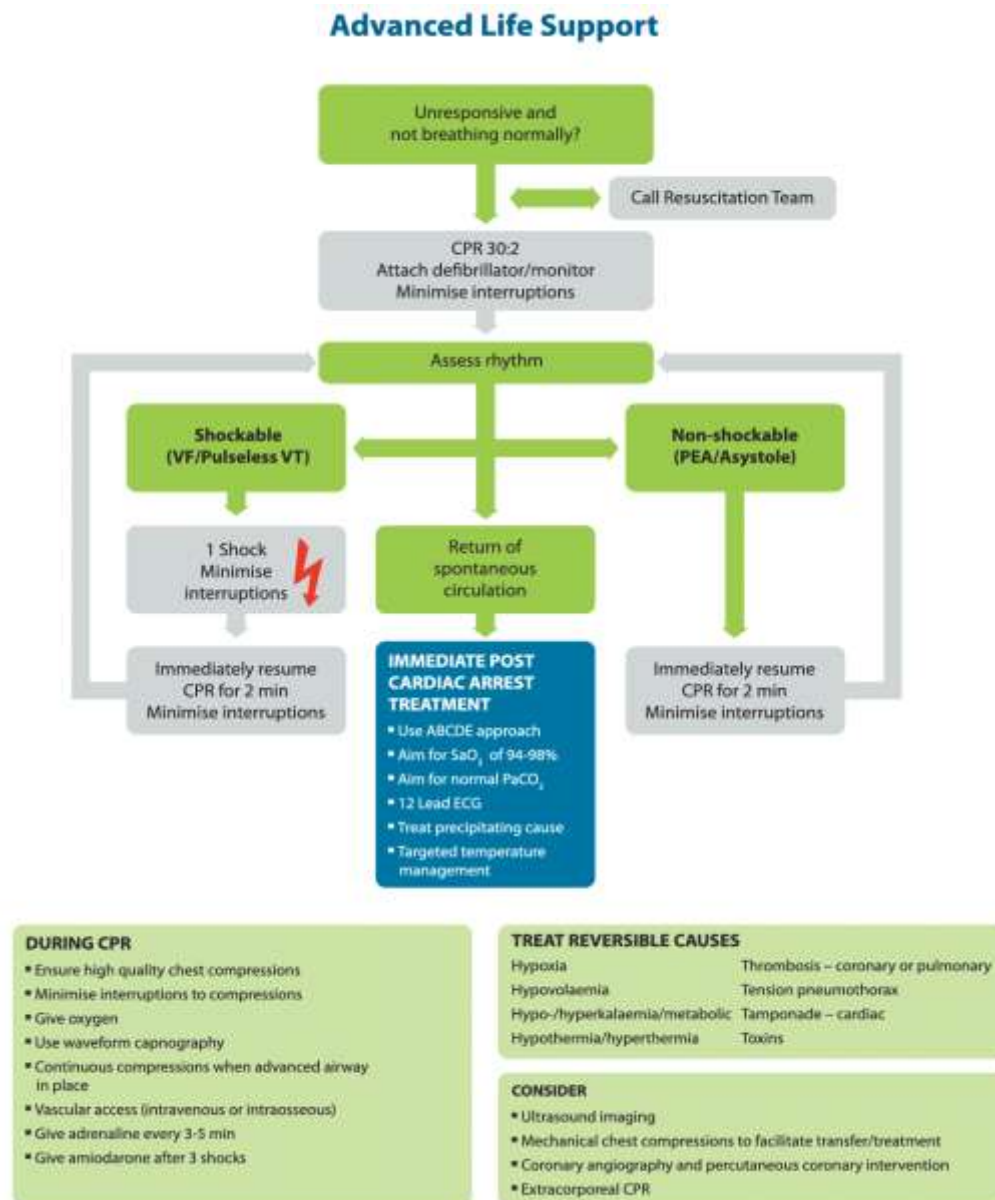


Figure (1): Algorithm of advanced life support (*Monsieurs et al., 2015*)

In most cases of cardiac arrest, the etiology determines the therapy. A focused history and physical examination are performed and diagnostic studies are obtained in order to identify possible causes for the arrest and on-going or imminent threats to life. Cardiovascular disease is the most common cause of Sudden Cardiac Arrest (SCA), but a broad differential diagnosis should be considered (*Dumas et al., 2010*).

The potentially reversible causes of cardiac arrest must be identified or excluded during any resuscitation. They are divided into two groups of four – 4Hs and 4Ts: hypoxia; hypo-/hyperkalemia and other electrolyte disorders; hypo-/hyperthermia; hypovolemia; tension pneumothorax; tamponade (cardiac); thrombosis (coronary and pulmonary); toxins (poisoning) (*Soar et al., 2015*).

The four ‘Hs’:

(1) Hypoxia:

Most cardiac arrests of non-cardiac origin have respiratory causes, such as drowning and asphyxia. Rescue breaths as well as chest compressions are critical for successful resuscitation of these victims (*Kitamura et al., 2010*).

Minimize the risk of hypoxia by ensuring that the patient's lungs are ventilated adequately with the maximal possible inspired oxygen during CPR (Cardio-Pulmonary Resuscitation). Make sure there is adequate chest rise and bilateral breath sounds. Check carefully that the tracheal tube is not misplaced in a bronchus or the esophagus (*Chamberlain et al., 2001*).

There are special situations of cardiac arrest due to hypoxia such as asthma and acute respiratory distress syndrome. The majority of asthma related deaths occur before admission to hospital. Cardiac arrest in a person with asthma is often a terminal event after a period of hypoxemia. Modifications to standard ALS guide-lines include considering the need for early tracheal intubation. If dynamic hyperinflation of the lungs is suspected during CPR, compression of the chest while disconnecting tracheal tube may relieve air trapping (*Monsieurs et al., 2015*).

Until the 1990s, most studies reported a 40-70% mortality rate for ARDS. However, later studies suggested much lower mortality rates, in the range of 30-40%. Possible explanations for the improved survival rates may be better understanding and treatment of sepsis, recent changes in the

application of mechanical ventilation, and better overall supportive care of critically ill patients (*Phua et al., 2009*).

Most deaths in ARDS patients are attributable to sepsis (a poor prognostic factor) or multiorgan failure rather than to a primary pulmonary cause. Mortality in ARDS increases with advancing age. The study performed in King County, Washington, found mortality rates of 24% in patients between ages 15 and 19 years and 60% in patients aged 85 years and older. The adverse effect of age may be related to underlying health status (*Stapleton et al., 2005*).

(2) Hypovolemia:

Pulseless electrical activity caused by hypovolemia is usually due to severe hemorrhage. This may be precipitated by trauma, gastrointestinal bleeding or rupture of an aortic aneurysm. Intravascular volume should be restored rapidly with warmed fluid, coupled with urgent surgery to stop the hemorrhage (*Brenner et al., 2013*).

Perioperative cardiac arrest is a special situation of cardiac arrest due to hypovolemia. The commonest cause of anesthesia related cardiac arrest involves airway management. Cardiac arrest caused by bleeding had the highest mortality in non-cardiac surgery, with only small percent of these patients

surviving to hospital discharge. Patients in the operating room are normally fully monitored and, as such, there should be little or no delay in diagnosing cardiac arrest (*Ellis et al., 2014*).

(3) Hypo-/hyperkalemia and other electrolyte disorders:

Hyperkalemia, hypokalemia, hypocalcemia, acidemia and other metabolic disorders are detected by biochemical tests (usually by using a blood gas analyzer) or suggested by the patient's medical history, e.g. renal failure. Intravenous calcium chloride is indicated in the presence of hyperkalemia, hypocalcemia and calcium channel-blocker overdose (*Monsieurs et al., 2015*).

Cardiac arrest in a dialysis unit is a special situation of cardiac arrest due to hypo-/hyperkalemia. Sudden cardiac death is the most common cause of death in hemodialysis patients and is usually preceded by ventricular arrhythmias. Hyperkalemia contributes to 2–5% of deaths amongst hemodialysis patients. A shockable rhythm (VF/pVT) is more common in patients undergoing hemodialysis. Most hemodialysis machine manufacturers recommend disconnection from the dialysis equipment prior to defibrillation (*Lafrance et al., 2006*).

(4) Hypo/Hyperthermia:

Hypothermia should be suspected based on the history such as cardiac arrest associated with drowning (**Gruber et al., 2014**). Hyperthermia occurs when the body's ability to thermoregulate fails and core temperature exceeds that normally maintained by homeostatic mechanisms. Hyperthermia is a continuum of heat-related conditions, starting with heat stress, progressing to heat exhaustion, heat stroke and finally multiple organ dysfunction and cardiac arrest. The main stay of treatment is supportive therapy and rapidly cooling the patient. Start cooling in the pre-hospital setting if possible. The aim is to rapidly reduce the core temperature to approximately 39°C (**Bouchama and Knochel, 2002**).

One of the most common causes of cardiac arrest due to hypothermia is drowning. Drowning is a common cause of accidental death. The Drowning Chain of Survival describes five critical links for improving survival from drowning. Bystanders play a critical role in initial attempts at rescue and resuscitation. The International Liaison Committee (ILCOR) reviewed specific prognostic indicators and noted that submersion durations of less than 10 min were associated with a very high chance of favorable outcome. Age, emergency

medical services (EMS) response time, fresh or saltwater, water temperature, and witness status were not useful for predicting survival. Submersion in ice-cold water may prolong the window of survival and justify extended search and rescue activities. The BLS sequence in drowning (Fig.2) reflects the critical importance of rapid alleviation of hypoxia (*Dyson et al., 2013*).



Figure (2): Basic life support for drowning (*Monsieurs et al., 2015*)

The four ‘Ts’:

(1) Thrombosis:

Coronary thrombosis associated with an acute coronary syndrome or ischemic heart disease is the most common cause of sudden cardiac arrest. An acute coronary syndrome is

usually diagnosed and treated after restoration of spontaneous circulation (ROSC). If an acute coronary syndrome is suspected, and ROSC has not been achieved, urgent coronary angiography should be considered when feasible and if required percutaneous coronary intervention. Mechanical chest compression devices and extracorporeal CPR can help facilitate this (*Mowry et al., 2014*).

On initial heart-rhythm analysis, about 25–50% of SCA victims have VF. The recommended treatment for VF cardiac arrest is immediate by standard CPR and early electrical defibrillation (*Ringh et al., 2009*).

The commonest cause of thromboembolic or mechanical circulatory obstruction is massive pulmonary embolism. The treatment of cardiac arrest with known or suspected pulmonary embolism includes the use of fibrinolysis, surgical or mechanical thrombectomy and extracorporeal CPR (*Konstantinides et al., 2014*).

Cardiac arrest following major cardiac surgery is relatively common in the immediate post-operative phase, with a reported incidence of 0.7–8%. Emergency resternotomy is an integral part of resuscitation after cardiac surgery, once all other reversible causes have been excluded. Once adequate

airway and ventilation has been established, and if three attempts at defibrillation have failed in VF/pVT, undertake resternotomy without delay. Emergency resternotomy is also indicated in asystole or PEA, when other treatments have failed, and should be performed within 5 min of the cardiac arrest by anyone with appropriate training (*LaPar et al., 2014*).

Cardiac arrest in a cardiac catheterization laboratory is commonly VF and may occur during percutaneous coronary intervention (PCI) for ST-elevation myocardial infarction (STEMI) or non-STEMI, but it may also be a complication of angiography. In this special setting with immediate response to monitored VF, defibrillation without preceding chest compressions is recommended. If needed for failed defibrillation or immediately recurring VF, immediate defibrillation may be repeated up to two times. If VF persists after the initial three shocks or return of spontaneous circulation (ROSC) not immediately established with certainty, chest compressions and ventilations must be initiated without further delay and a cause for the unresolved problem sought with further coronary angiography (*Ringh et al., 2009*).

On an angiography table with the image intensifier above the patient, delivering chest compressions with adequate depth

and rate is almost impossible and exposes the rescuers to dangerous radiation. Therefore, early transition to the use of a mechanical chest compression device is strongly recommended. If the problem is not rapidly resolved, very low quality evidence suggests that the use of extracorporeal life support (ECLS) can be considered as a rescue strategy if the infrastructure is available, and probably to be preferred over intra-aortic balloon pump (IABP) (*Tsao et al., 2012*).

(2) Tension pneumothorax:

A tension pneumothorax may be the primary cause of pulseless electrical activity (PEA) and may be associated with trauma or follow attempts at central venous catheter insertion. The diagnosis is made clinically or by ultrasound. Decompress rapidly by thoracostomy or needle thoracocentesis, and then insert an intercostal tube (**Kleber et al., 2014**). In the context of cardiac arrest from major trauma, consider bilateral thoracostomies for decompression of a suspected tension pneumothorax (*Massarutti et al., 2006*).

(3) Tamponade:

Cardiac tamponade is difficult to diagnose because the typical signs of distended neck veins and hypotension are usually obscured by the arrest itself. Cardiac arrest after

penetrating chest trauma is highly suggestive of tamponade and is an indication for resuscitative thoracotomy. The use of ultrasound will make the diagnosis of cardiac tamponade much more reliable (*Leis et al., 2013*).

Traumatic cardiac arrest:

Traumatic cardiac arrest (TCA) carries a very high mortality, but in those where ROSC can be achieved, neurological outcome in survivors appears to be much better than in other causes of cardiac arrest. In cardiac arrest caused by hypovolemia, cardiac tamponade or tension pneumothorax, chest compressions are unlikely to be as effective as in normovolemic cardiac arrest. For this reason, chest compressions take a lower priority than the immediate treatment of reversible causes, e.g. thoracotomy, controlling hemorrhage etc. (*Zwingmann et al., 2012*). (Fig.3).

(4)Toxins:

In the absence of a specific history, the accidental or deliberate ingestion of therapeutic or toxic substances may be revealed only by laboratory investigations. Where available, the appropriate antidotes should be used, but most often treatment