

Advances in Dosage Forms and Devices for Addressing Cardiac Emergencies: A Comprehensive Review

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Abstract: Cardiac arrest is characterized by the abrupt halt of both spontaneous breathing and circulation. When cardiac arrest occurs, the individual loses consciousness within 15 seconds. Around 30 seconds later, their brain activity, as measured by the electroencephalogram, becomes flat. After approximately 60 seconds, their pupils fully dilate. Significant damage to the brain can begin to occur between 90 and 300 seconds, making immediate action crucial to prevent irreversible harm. Rapid intervention is vital due to the potential for swift and irreversible damage. Cardiopulmonary resuscitation (CPR) is employed to restore natural circulation. This life-saving technique involves several approaches, including prompt defibrillation, consistent and effective chest compressions, advanced airway methods, and, if needed, medications. It's important to prioritize the initial steps such as defibrillation, chest compressions, and ventilation before contemplating drug administration. Although a few drugs offer short-term survival benefits, no specific drug therapy during CPR has demonstrated consistent improvement in survival until hospital discharge.[1] In the initial stages of the COVID-19 pandemic, there were reports of an increased occurrence of sudden cardiac arrests (SCA) and reduced rates of survival.[2] The pandemic had a profound impact on both the frequency of cardiac arrests and the chances of survival.[3] This paper reviews the management and pharmacotherapy of cardiac arrest.

Keywords: Cardiac arrest, CPR, AED, Arrhythmia, COVID-19.

1. Introduction

Cardiac arrest is when the heart stops beating. It is defined as cessation of normal circulation of blood due to failure of the heart to pump effectively. It is a medical emergency that, without immediate medical intervention, will result in cardiac death within minutes. When it happens suddenly, it is called sudden cardiac arrest.[4] Cardiopulmonary resuscitation (CPR) and possibly defibrillation are needed until further treatment can be provided. Cardiac arrest results in a rapid loss of consciousness, and breathing may be abnormal or absent.[5] [6] [7]

A heart attack is different from a sudden cardiac arrest (SCA). A heart attack happens when blood flow to the heart is blocked. During a heart attack, the heart usually doesn't suddenly stop beating. With an SCA, the heart stops beating. Sometimes an SCA can happen after or during recovery from a heart attack.[6]

CPR and defibrillation can reverse a cardiac arrest, leading to return of spontaneous circulation (ROSC), but without such intervention, it will prove fatal.[10] In some cases, cardiac arrest is an anticipated outcome of serious illnesses where death is expected.[11] Treatment for cardiac arrest includes immediate CPR and, if a shockable rhythm is present, defibrillation.[12] Two protocols have been established for CPR: basic life support (BLS) and advanced cardiac life support (ACLS).[13] Among those whose pulses are reestablished, targeted temperature management may improve outcomes.[14][15] In addition, the care team may

initiate measures to protect the patient from brain injury and preserve brain function.[16] In post-resuscitation care, an implantable cardiac defibrillator may be considered to reduce the chance of death from recurrence.[6] In the United States, approximately 535,000 cases occur annually (about 13 per 10,000 people). Of these, 326,000 (61%) experience cardiac arrest outside of a hospital setting, while 209,000 (39%) occur within a hospital.[17] Cardiac arrest becomes more common with age and affects males more often than females.[6] Around 8% survive OHCA with EMS treatment. Fictional portrayals create unrealistic expectations; public often overestimates resuscitation success (40–50%). [18] [19] [21]

1.1 Signs and symptoms

Cardiac arrest is not preceded by any warning symptoms in approximately 50 percent of people.[22] For individuals who do experience symptoms, the symptoms are usually nonspecific to the cardiac arrest.[23] This can present in the form of new or worsening:

- chest pain[24]
- fatigue
- blackouts
- dizziness
- shortness of breath
- weakness
- vomiting[23]

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Assume cardiac arrest, start CPR. Healthcare professional: Check pulse for 10 seconds before CPR. Loss of cerebral perfusion leads to rapid loss of consciousness and breathing cessation. [25][26]

1.2 Risk factors

The risk factors for sudden cardiac arrest (SCA) are similar to those of coronary artery disease and include age, cigarette smoking, high blood pressure, high cholesterol, lack of physical exercise, obesity, diabetes, and family history and Cardiomyopathy of cardiac disease.[27] A prior episode of sudden cardiac arrest also increases the likelihood of future episodes.[28] A statistical analysis of many of these risk factors determined that approximately 50% of all cardiac arrests occur in 10% of the population perceived to be at greatest risk due to aggregate harm of multiple risk factors, demonstrating that cumulative risk of multiple comorbidities exceeds the sum of each risk individually.[29] Previous adverse cardiac events, non-sustained ventricular tachycardia (NSVT), syncope, and left ventricular hypertrophy (LVT) have been shown to predict sudden cardiac death in children.[30] Current cigarette smokers with coronary artery disease were found to have a two to threefold increase in the risk of sudden death between ages 30 and 59. Furthermore, it was found that former smokers' risk was closer to that of those who had never smoked.[22][31]

1.3 Causes and mechanisms



Conduction of the heart. Changes in this pattern can result from injury to the cardiac muscle and lead to non-conducted beats and ultimately cardiac arrest.



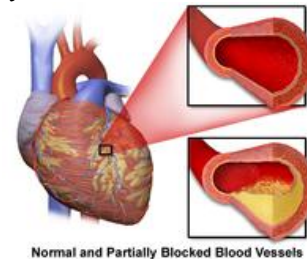
EKG depiction of ventricular fibrillation (no organized rhythm)

Sudden cardiac arrest (SCA), or sudden cardiac death (SCD), occurs when the heart abruptly begins to beat in an abnormal or irregular rhythm (arrhythmia).[32] [33] There are many different types of arrhythmias, but the ones most frequently recorded in sudden cardiac arrest are ventricular tachycardia and ventricular fibrillation.[34][35][36] Less common causes of dysrhythmias in cardiac arrest include

pulseless electrical activity (PEA), bradyarrhythmias, or asystole. [33] Sudden cardiac arrest can result from cardiac and non-cardiac causes including the following:

Cardiac causes

Coronary artery disease



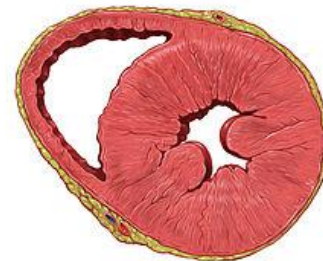
Normal vs blocked coronary artery

Coronary artery disease (CAD), also known as ischemic heart disease, is responsible for 62 to 70 percent of all sudden cardiac deaths. CAD is a much less frequent cause of sudden cardiac death in people under the age of 40.[37] Cases have shown that the most common finding at postmortem examination of sudden cardiac death is chronic high-grade stenosis of at least one segment of a major coronary artery, an artery that supplies the heart muscle with its blood supply. [38] Stenosis is due to artery narrowing from cholesterol plaques and inflammation, marking Atherosclerotic Cardiovascular Disease progression in coronary and systemic blood vessels.[39] A ruptured plaque can block blood flow, causing ischemic injury. This harms tissue, leading to structural changes that disrupt normal heart conduction and alter heart rate.[29]

Non-atherosclerotic coronary artery abnormalities

Abnormalities of the coronary arteries not related to atherosclerosis include congenital coronary artery anomalies (most commonly anomalous origin of the left coronary artery from the pulmonary artery), inflammation known as coronary arteritis, embolism, vasospasm, and mechanical abnormalities related to connective tissue diseases or trauma. These conditions account for 10-15% of cardiac arrest and sudden cardiac death.[29]

Structural heart disease



Short axis view of the heart demonstrating wall thickening in left ventricular hypertrophy. Structural heart diseases unrelated to coronary artery disease account for 10% of all sudden cardiac deaths.[34] [39] Examples of these include: cardiomyopathies (hypertrophic, dilated, or arrhythmogenic), cardiac rhythm disturbances, myocarditis, hypertensive heart disease, and congestive heart failure.[41]



EKG depiction of left ventricular hypertrophy

Left ventricular hypertrophy, often from high blood pressure, can lead to sudden cardiac deaths. Prolonged hypertension causes the heart's main pumping chamber to grow, reducing effectiveness. Detected by echocardiogram and EKG [42] Congestive heart failure increases the risk of sudden cardiac death fivefold.[46]

Inherited arrhythmia syndromes

Arrhythmias not due to structural heart disease account for 5 to 10% of sudden cardiac arrests. These are frequently caused by genetic disorders that lead to abnormal heart rhythms. [33]

Non-cardiac causes

Non-cardiac causes account for 15 to 25% of cardiac arrests.[8][9] The most common non-cardiac causes are trauma, major bleeding (gastrointestinal bleeding, aortic rupture, or intracranial hemorrhage), hypovolemic shock, overdose, drowning, and pulmonary embolism.[9][43][44] Cardiac arrest can also be caused by poisoning like the stings of certain jellyfish or through electrocution like lightning.[33]

Reversible causes

Other non-cardiac causes of cardiac arrest may result from temporary disturbances in the body's homeostasis. This may be the result of changes in electrolyte ratios, oxygen saturation, or alterations of other ions influencing the body's pH.[45]

Mechanism

Cardiac arrest results from abnormal rhythms: tachyarrhythmias (V-fib, V-tach) disrupt circulation. Ventricular fibrillation is chaotic, no meaningful output; Ventricular tachycardia hinders proper heart filling.[47] In ventricular tachycardia, the heart also beats faster than normal, which may prevent the heart chambers from properly filling with blood.[48] [49]

For people who have had COVID-19, lingering COVID-19 heart problems can complicate their recovery. Some of the symptoms common in coronavirus "long-haulers," such as palpitations, dizziness, chest pain and shortness of breath, may be due to heart problems — or, just from having been ill with COVID-19. COVID-19 is primarily a respiratory or lung disease, the heart can also suffer.

Heart Rate and COVID-19

After COVID-19, contact your doctor for rapid heartbeat or palpitations. Dehydration can cause temporary increase. Drink fluids, especially with fever. Watch for symptoms like irregular heartbeat, lightheadedness, and chest discomfort, especially when standing.

Shortness of Breath

Sometimes people are short of breath with exertion after COVID-19 because they have been less active for a long time and need to gradually build their fitness level back up. A diagnosis of heart failure after COVID-19 is rare. But if you have shortness of breath or leg swelling after COVID-19, you should contact your doctor, who may recommend evaluation by a cardiologist if tests indicate you are at risk.

COVID-19 Chest Pain

POTS (postural orthostatic tachycardia syndrome) isn't directly a cardiac problem, but a neurologic one that affects the part of the nervous system that regulates heart rate and blood flow. The syndrome can cause rapid heartbeats when you stand up, which can lead to brain fog, fatigue, palpitations, lightheadedness and other symptoms.

COVID-19 symptoms mimic a heart attack

COVID-19 symptoms can resemble a heart attack, like chest pain and shortness of breath. Echocardiograms and EKGs might show changes, but angiograms often don't reveal major blockages indicating a heart attack. Myocarditis symptoms can also mimic heart attack. Viral infections like COVID-19 can create small blood clots causing pain. Urgent medical attention is vital for heart attack-like symptoms; don't manage them at home. [50]

Diagnosing Cardiac Arrest

During a cardiac event that causes your heart to stop beating efficiently, it's vital to seek medical attention immediately. Medical treatment will focus on getting blood flowing back to your body. [51]

Tests for sudden cardiac arrest often include:

- Blood tests for heart damage markers and chemicals affecting function.
- Electrocardiogram (ECG) checks electrical activity and detects risky heartbeat changes.
- Echocardiogram uses sound waves to visualize heart motion, blood flow, and valve issues.
- Ejection fraction (EF) measures blood pumped per heartbeat; low EF raises sudden cardiac arrest risk.
- Chest X-ray for heart and lung size/shape, possibly indicating heart failure.
- Nuclear scan (with stress test) shows blood flow problems using radioactive tracer.
- Cardiac catheterization identifies heart artery blockages using dye and X-ray guidance

A treatment called balloon angioplasty can be done during this test to treat a blockage. If a blockage is found, the health care provider may treat place a tube called a stent to hold the artery open.[52]

Treatment / Management

A patient in cardiac arrest is treated in multiple different stages. The interventions that have proven to reverse cardiac arrest include early CPR and early defibrillation. The initial step involves identification and basic life-support measures.

Treatment of cardiac arrest depends on rescuer scope of practice:

Lay Rescuer: Treatment includes hands-only CPR and utilization of AED, if available.[53][54] If a patient has had a drowning episode, they can attempt two rescue breaths, since the cause of cardiac arrest is likely from a primary respiratory arrest. If there is no response to rescue breathing, CPR should be initiated. One should continue CPR until the arrival of emergency responders.

Basic Life Support

Treatment for those who are certified to practice basic life support (BLS) includes treatment as above, with the addition of ventilation during active CPR. Current guidelines recommend 2 breaths for every 30 compressions.[53] Providers can also manipulate the airway to aid in airway patency, thus, allowing for proper ventilation. These maneuvers include the head-tilt, chin-lift[55], and the jaw thrust [56]. Oral airway adjuncts including the oral pharyngeal airway (OPA) and the nasopharyngeal airway (NPA) should also be utilized to benefit ventilation.

Sudden cardiac arrest may be treated via attempts at resuscitation. This is usually carried out based on basic life support, advanced cardiac life support (ACLS), pediatric advanced life support (PALS), or neonatal resuscitation program (NRP) guidelines.[57][58]

Cardiopulmonary resuscitation**CPR training on a mannequin**

Early cardiopulmonary resuscitation (CPR) is essential to surviving cardiac arrest with good neurological function.[59] It is recommended that it be started as soon as possible with minimal interruptions once begun. The components of CPR that make the greatest difference in survival are chest compressions and defibrillating shockable rhythms.[60] After defibrillation, chest compressions should be continued for two minutes before another rhythm check. This is based on a compression rate of 100-120 compressions per minute, a compression depth of 5-6 centimeters into the chest, full chest recoil, and a ventilation rate of 10 breath ventilations per minute.[33] Correctly performed bystander CPR has been shown to increase survival; it is performed in fewer than 30% of out-of-hospital cardiac arrests (OHCAs) as of 2007. If high-quality CPR has not resulted in return of spontaneous circulation (ROSC) and the person's heart rhythm is in asystole, discontinuing CPR and pronouncing the person's death is generally reasonable after 20 minutes.[61][62] [63]

High levels of oxygen are generally given during CPR. Tracheal intubation has not been found to improve survival rates or neurological outcomes in cardiac arrest and in the prehospital environment, may worsen it. [64] Endotracheal tubes and supraglottic airways appear equally useful. [65] Mouth-to-mouth as a means of providing respirations to the patient has been phased out due to the risk of contracting infectious diseases from the patient.[66] [67] Mechanical chest compressions (as performed by a machine) are no better than chest compressions performed by hand.



An automated external defibrillator stored in a visible orange mural support Defibrillation is indicated if an electric-shockable heart rhythm is present. The two shockable rhythms are ventricular fibrillation and pulseless ventricular tachycardia. In children, 2 to 4 J/Kg is recommended. [68]

In out-of-hospital arrests, the defibrillation is made by an automated external defibrillator (AED), a portable machine that can be used by any user: it provides voice instructions that guide the process, automatically checks the victim's condition, and applies the appropriate electric shocks. Some defibrillators even provide feedback on the quality of CPR compressions, encouraging the lay rescuer to press the person's chest hard enough to circulate blood.[69] [70]

Advanced Life Support

BLS treatment plus advanced airways (supraglottic devices, intubation) and meds (Epinephrine, Amiodarone) are used. ALS includes rhythm interpretation for faster defibrillation. ACLS teaches resuscitation algorithms for cardiac arrest.

Physician

Providers can use ALS treatment and progress to a wide scope of practice dependent on medical versus traumatic etiology.

Medical

In this paper, we summarize with the experimental and clinical data on the efficacy and safety of drugs during CPR (vasopressors, antiarrhythmics, and other drugs such as sodium bicarbonate, calcium, magnesium, atropine, fibrinolytic drugs, and corticosteroids).

1) Vasopressors

Cardiac arrest is characterized by global ischemia, tissue hypoxia, and acidosis. CPR aims to improve the oxygen supply-demand ratio, in order to reverse tissue hypoxia.

Epinephrine

Epinephrine stimulates α - and β -Adrenergic Receptors. β -receptor activation triggers Gs-proteins, adenylyclase, and cyclic adenosine monophosphate (cAMP) production. Beta-adrenergic effects can enhance coronary and cerebral blood flow, but also raise oxygen use, risk ventricular arrhythmias, hypoxemia, shunting, impaired microcirculation, and post-ROSC heart failure.[71][72]

Vasopressin

Epinephrine's risky effects led to studying vasopressors. Higher vasopressin levels in cardiac arrest survivors. Lab models found vasopressin + epinephrine improved coronary perfusion. Yet, human trials showed no clear vasopressin benefit over epinephrine in treating cardiac arrest.

2) Antiarrhythmics

Antiarrhythmic drugs block heart ion channels (sodium, potassium, calcium) to modify action potential. Different ion currents in five phases create cardiac action potential. Drug impact on action potential and refractory period influences clinical outcomes.

- Amiodarone: Potent Class III antiarrhythmic, inhibits potassium current, diverse cardiovascular effects like coronary artery and peripheral vasodilation. Major adverse effects are hypotension and bradycardia.
- Lidocaine: Once vital for refractory VF/pulseless VT, now secondary option. Lowers ventricular automaticity, blocks sodium channels more during myocardial ischemia. Toxicity signs include paraesthesia, confusion, and convulsions. Safe dose <3 mg/Kg over first hour.
- Magnesium: Emerging antiarrhythmic, sodium/potassium pump agonist. Effects: blocks atrial calcium channels, prolongs refractory period, suppresses ventricular after-depolarizations.

Other Drugs**a) Atropine**

Atropine, an anticholinergic drug, blocks vagus nerve effects on heart nodes, boosting automaticity and conduction. Given IV (0.6–3.0 mg) to treat bradycardia with hypotension or vagal stimulation. Dose-related side effects, low dose (<0.5 mg) can lead to bradycardia.

b) Calcium

Calcium ions are involved in cellular excitation, excitation-contraction coupling, and muscle contraction in cardiac, skeletal, and smooth muscle cells. Increased extracellular calcium increases intracellular calcium concentrations, and the force of contraction of cardiac myocytes and vascular smooth muscle cells.[75]-[77]

c) Sodium Bicarbonate

During cardiac arrest and CPR, combined respiratory and metabolic acidosis arises from the carbon dioxide retention as pulmonary gas exchange ceases and from the reduction in cellular oxygen availability which leads to the development of anaerobic metabolism with lactic acidosis. Severe acidosis inhibits myocardial contractility and also reduces the responsiveness to catecholamines. [78–80].

d) Fibrinolytic Drugs

Thrombolytic therapy during CPR has two major effects. First, it can be effective in acute myocardial infarction (AMI) or massive pulmonary embolism (PE), which are common causes of cardiac arrest. Second, it may improve microcirculatory reperfusion after ROSC. Cardiac arrest and CPR are associated with a marked activation of coagulation without adequate fibrinolysis [81].

e) Corticosteroids

Relative to other stress states, cardiac arrest is associated with lower cortisol levels during and after CPR [82]. This adrenocortical dysfunction results in hypotension and shock. The KG Hospital, Coimbatore has been given three drugs as a loading dose (atorvastatin 80mg- No, clopidogrel 150mg - 2 No, disprin 350mg -1no.)

2. Conclusion

The conclusion of the article on cardiac arrest management and pharmacotherapy may vary depending on the specific content and findings of the article. However, in general, articles on this topic typically emphasize the critical importance of timely and effective interventions during cardiac arrest situations. Pharmacotherapy, such as administering medications like epinephrine, plays a crucial role in the advanced cardiac life support (ACLS) guidelines to improve the chances of successful resuscitation. Proper management and coordination among healthcare professionals are crucial in providing the best possible outcome for patients experiencing cardiac arrest.

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