ISSN 1998 – 409X

REVIEW ARTICLE

# Cardiac Arrest: Comprehensive Review of Etiology and Evidence-based Management Strategies

**Anju K. Abraham, S. Haritha, S. K. Anwar Bhasha**

*Department of Pharmacy Practice, Aditya College of Pharmacy, Surampalem, Andhra Pradesh, India*

**Abstract**

Cardiac arrest is a life-threatening emergency characterized by the sudden cessation of effective cardiac activity, resulting in loss of circulation and consciousness. It remains a major cause of mortality worldwide and requires immediate intervention to prevent irreversible organ damage and death. This review summarizes the various cardiac and non-cardiac causes of cardiac arrest, including ischemic heart disease, heart failure, cardiomyopathies, inherited arrhythmia syndromes, pulmonary embolism, electrolyte disturbances, and trauma. The underlying pathophysiological mechanisms contributing to cardiac arrest are discussed, along with current evidence-based management strategies. Particular emphasis is placed on the role of basic life support, advanced life support, early defibrillation, and post-resuscitation care in improving survival outcomes. Preventive approaches, including cardiovascular risk reduction and appropriate use of implantable cardioverter-defibrillators, are also highlighted as key measures for reducing sudden cardiac death.

**Key words:** Advanced life support, arrhythmias, cardiac arrest, cardiopulmonary resuscitation, cardiovascular disease, defibrillation, prevention, sudden cardiac death

## INTRODUCTION

Cardiac arrest involves cessation of cardiac mechanical activity as confirmed by absence of signs of circulation (e.g., detectable pulse, unresponsiveness, and apnea). Sudden cardiac death is further described as a death from a cardiac cause within 1 h from the onset of symptoms in a person without any prior condition that would appear fatal.[1]

Cardiac arrest should be used to signify an event as described above that is reversed, usually by cardiopulmonary resuscitation (CPR) and/or defibrillation or cardioversion or cardiac pacing. Most cardiac arrests are of cardiac origin (65%), but in 15% of patients the cause is pulmonary.[2]

This medical emergency is distinct from a heart attack (myocardial infarction), as it involves the failure of the heart's electrical system rather than a blockage in blood flow.[3]

The condition is a leading cause of death worldwide, with survival rates heavily dependent on timely intervention. Without immediate resuscitation efforts, such as CPR and defibrillation, irreversible brain damage or death can occur within minutes.[4]

## ETIOLOGY

Cardiac arrest typically occurs due to underlying structural problems in the heart. About 70% of cardiac arrest cases are linked to ischemic coronary disease, which is the main cause of cardiac arrest. Other structural causes can include congestive heart failure, left ventricular hypertrophy (LVH), congenital coronary artery abnormalities, arrhythmogenic right ventricular dysplasia (ARVD), hypertrophic obstructive cardiomyopathy, and cardiac tamponade.

Non-structural causes of cardiac arrest include conditions such as Brugada syndrome, Wolff–Parkinson–White syndrome, and congenital long QT syndrome (CLQTS).

**Address for correspondence:**
S. Haritha, Department of Pharmacy Practice,
Aditya College of Pharmacy, Surampalem,
Andhra Pradesh, India. Phone: +91-9959113723.
E-mail: sagaramharitha@gmail.com

**Received:** 03-07-2026
**Revised:** 07-09-2026
**Accepted:** 16-09-2026

© 2026 Author(s). This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY 4.0)

There are also many non-cardiac causes of cardiac arrest such as intracranial hemorrhage, pulmonary embolism, pneumothorax, primary respiratory failure, toxic ingestions including drug overdose, electrolyte imbalances, severe infection (sepsis), hypothermia, or trauma.[5]

Causes of maternal cardiac arrest include:[6]

- Hemorrhage
- Pre-eclampsia/eclampsia
- Pulmonary embolism
- Amniotic fluid embolism
- Sepsis
- Magnesium toxicity
- Total spinal anesthesia
- Local anesthetic toxicity

Hyperkalemic cardiac arrest can occur as a complication of a massive transfusion in children. In athletes who are 35 years or older, most sudden cardiac death events are caused by atherosclerotic coronary artery disease (CAD). In individuals under 35, genetic and other acquired cardiovascular issues are more common.[7]

## PATHOPHYSIOLOGY

### Ischemic Coronary Disease

According to the conventional ischemic heart disease (IHD) pathophysiological point of view, an obstructive plaque that inhibits blood flow through the coronary artery triggers myocardial ischemia. CAD is defined by the presence of an obstructive atherosclerotic plaque, which causes a reduction in blood flow to the myocardium.[8]

Atherosclerotic plaque buildup can cause narrowing of the coronary arteries and reduce myocardial blood flow, as shown in Figure 1.

### LVH

LVH is a compensatory but ultimately abnormal increase in the mass of the myocardium of the left ventricle induced by a chronically elevated workload on the heart muscle. Another cause is increased filling of the left ventricle, inducing diastolic overload, which is the underlying mechanism for eccentric LVH in patients with regurgitant valvular lesions such as aortic regurgitation or mitral regurgitation and is also seen in dilated cardiomyopathy.[9]

Left ventricular hypertrophy is characterized by an increase in the thickness of the left ventricular myocardium and may occur in conditions such as chronic hypertension, as illustrated in Figure 2.

### Congestive Heart Failure

Although the etiology of congestive heart failure is complex, a disturbance of myocardial function due to IHD or various forms of cardiomyopathy is by far the leading causative factor. In the early stages of failure, various cardiac compensating mechanisms, such as hypertrophy, an increase in contractility, and the Frank–Starling mechanism, prevent a major reduction in function. With ongoing failure, myocardial muscle function further deteriorates because of a disturbance in myocardial energy utilization, abnormalities in sympathetic neurotransmitter metabolism, a reduction in P-receptor density, and, more importantly, a derangement of intracellular calcium transport. The subsequent reduction

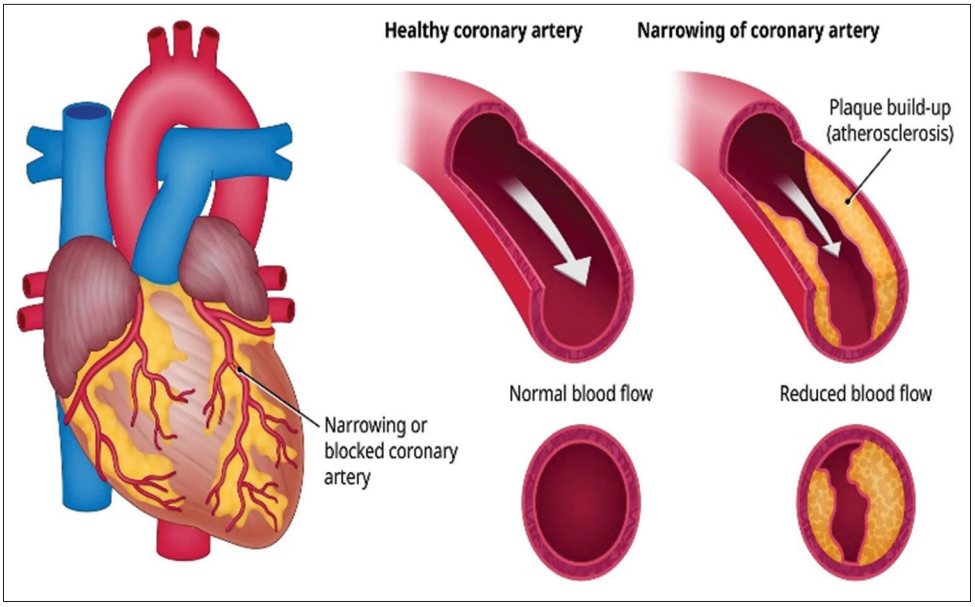



**Figure 1:** Coronary artery disease and myocardial ischemia

in cardiac output (CO) leads to activation of peripheral neurohumoral mechanisms, including sympathetic stimulation, an increase in circulating norepinephrine, stimulation of the renin–angiotensin–aldosterone system, and increased arginine vasopressin production, which result in arterial and venous vasoconstriction, redistribution of tissue blood flow, and an increase in circulating blood volume. The adjustments, however, lead to a vicious circle, where heart function is further depressed by the increase in afterload, whereas the changes in the venous bed and the increase in circulating volume ultimately result in congestion.[10]

In congestive heart failure, impaired cardiac function can result in inadequate tissue perfusion and accumulation of fluid in the lungs and peripheral tissues, as shown in Figure 3.

## ARVD

ARVD is a new form of cardiomyopathy probably more frequent than commonly reported. It is a rare but important cause of sudden arrhythmic death in young, otherwise healthy persons, as well as a subtle cause of congestive heart failure. It may lead to temporary incapacitation with catastrophic consequences.[11] It is a disorder in which normal myocardium is replaced by fibrofatty tissue. This disorder usually involves the right ventricle (RV), but the left ventricle and septum also may be affected.[12]

In ARVC, progressive loss of right ventricular myocardium and replacement by fibrofatty tissue can result in right ventricular dilation and dysfunction, as shown in Figure 4.

## Hypertrophic Obstructive Cardiomyopathy

Hypertrophic obstructive cardiomyopathy is an inherited myocardial disease defined by cardiac hypertrophy (wall thickness ≥15 mm) that is not explained by abnormal loading conditions and left ventricular obstruction greater than or equal to 30 mm Hg. Typical symptoms include dyspnea, chest pain, palpitations, and syncope.[13]

Hypertrophic obstructive cardiomyopathy is characterized by abnormal thickening of the left ventricular myocardium, particularly the interventricular septum, which can narrow the left ventricular outflow tract and impair cardiac output, as illustrated in Figure 5.

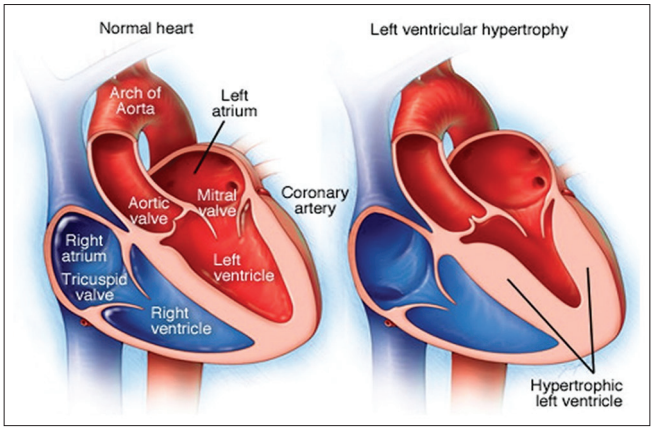



**Figure 2:** Left ventricle hypertrophy

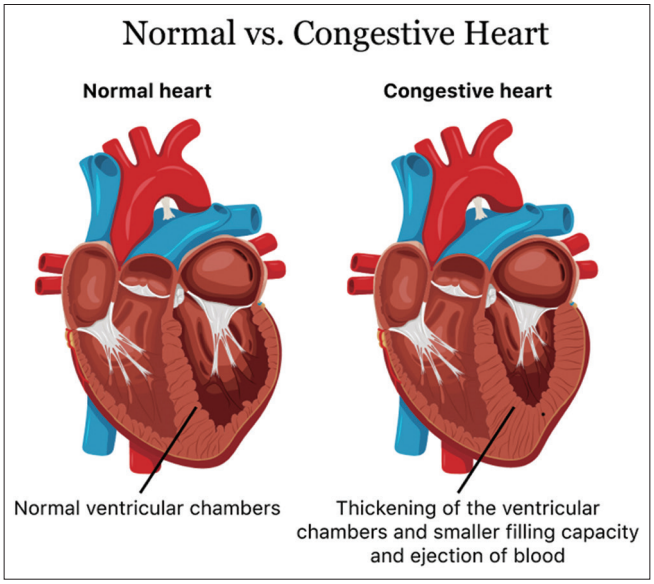



**Figure 3:** Congestive heart failure

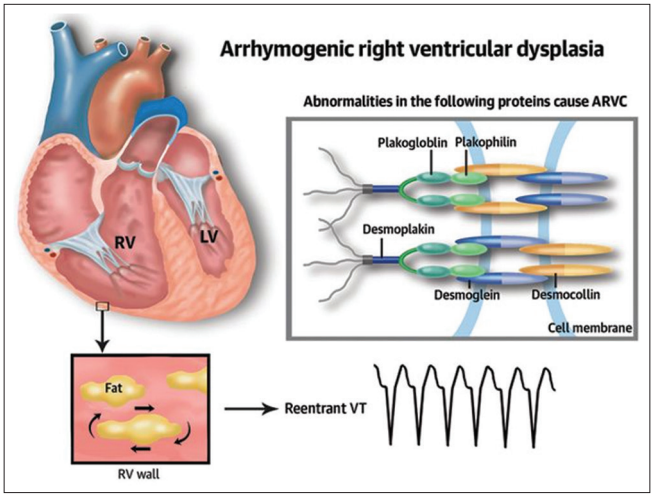



**Figure 4:** Arrhythmogenic right ventricular dysplasia

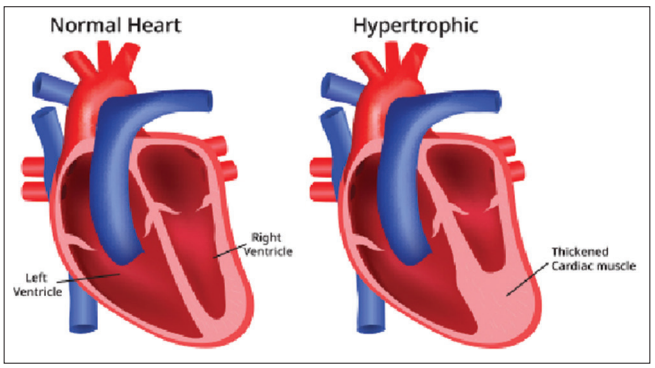



**Figure 5:** Hypertrophic obstructive cardiomyopathy

### Cardiac Tamponade

Cardiac tamponade is a clinical syndrome that results from an increased intrapericardial pressure and leads to impaired cardiocirculatory function.[14] Tamponade occurs when intrapericardial collections compress cardiac chambers to sufficiently constrain filling and impact stroke volume and hemodynamics. However, tamponade is not an "all-or-none" phenomenon and exists on a hemodynamic continuum mediated by several physiologic mechanisms and compensatory responses.

With a moderate increase in pericardial pressure, the right atrium begins to collapse, followed by early diastolic collapse of the RV with a resultant reduction in CO.[15]

Cardiac tamponade occurs when accumulation of fluid within the pericardial sac increases intrapericardial pressure and compresses the heart, impairing ventricular filling and reducing cardiac output, as illustrated in Figure 6.

### Brugada Syndrome

Brugada syndrome is an inherited disease characterized by an increased risk of sudden cardiac death owing to ventricular arrhythmias in the absence of structural heart disease.[16] Brugada syndrome is a genetically determined cardiac arrhythmia characterized by syncopal states, ST-segment rises (from point J) above the isoelectric line in the right precordial leads (V1–V3), which can disappear transiently. Furthermore, inverted T waves, complete or incomplete bundle branch block, and periodic prolongation of the P–R interval with a high risk of life-threatening ventricular tachyarrhythmias (episodes of polymorphic ventricular tachycardia and ventricular fibrillation) usually occur during sleep or at rest and can be recorded in these leads. Often, patients also have supraventricular arrhythmias, more often atrial fibrillation.[17]

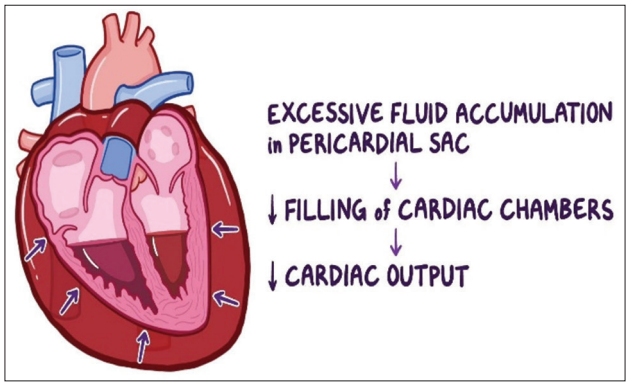



**Figure 6:** Cardiac tamponade

Brugada syndrome is an inherited cardiac channelopathy characterized by abnormal cardiac electrical activity and an increased risk of ventricular arrhythmias and sudden cardiac arrest, as illustrated in Figure 7.

### Wolff–Parkinson–White (WPW) Syndrome

It is a cardiac conduction disorder that presents with potentially life-threatening consequences. WPW syndrome is a congenital heart disease that is characterized by the presence of abnormal electrical connections between the atria and ventricles of the heart. The normal conduction of the AV node occurs more slowly than the accessory pathway conduction. Pre-excitation is a process in which the cardiac ventricles are activated earlier than the impulse from the AV node, which leads to a shorter PR interval and formation of a delta wave. The supraventricular tachycardia associated with WPW syndrome is called AV reentrant or reciprocating tachycardia. WPW syndrome is when there is an accessory pathway between the atrium and ventricles, which causes rapid heartbeat or tachycardia.[18]

Wolff–Parkinson–White (WPW) syndrome is a cardiac pre-excitation disorder caused by the presence of an accessory pathway that allows electrical impulses to bypass the normal atrioventricular conduction system, predisposing patients to tachyarrhythmias and, rarely, sudden cardiac arrest [Figure 8].

### CLQTS

It is the most common inherited arrhythmia. The QT interval, which marks the duration of ventricular depolarization and repolarization in the myocardium, can be prolonged due to

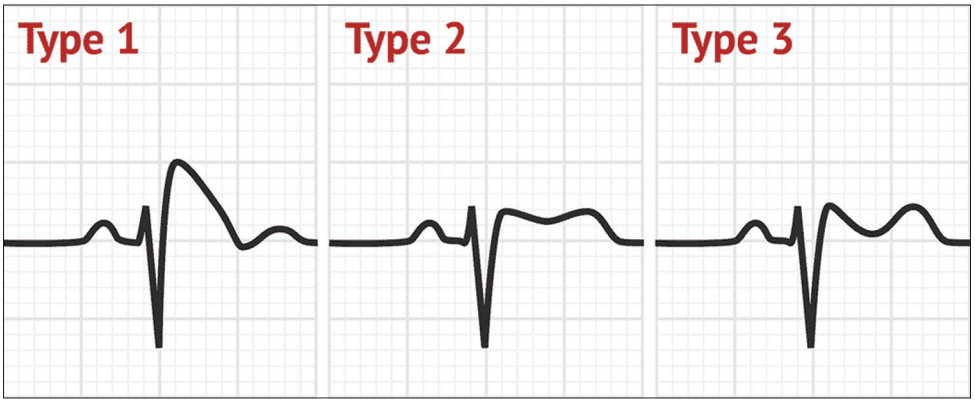



**Figure 7:** Brugada syndrome ECG patterns

mutations in genes coding for the ion channel proteins that govern the cardiac action potential. The lengthening of the QT interval can lead to a wide range of clinical symptoms, including seizures, torsades de pointes, and fatal arrhythmias.[19] CLQTS is a disorder of myocardial repolarization defined by a prolonged QT interval on electrocardiogram that can cause ventricular arrhythmias and lead to sudden cardiac death. The QT interval represents the time from the initiation of ventricular depolarization to the end of ventricular repolarization. In the majority of congenital LQTS patients, mutations in the genes that encode cardiac ion channels result in prolongation of the action potential; therefore, congenital LQTS is considered a cardiac channelopathy.

Four genes (*CALM1, CALM2, CALM3,* and *TRDN*) were found to have strong or definitive evidence for causing LQTS but with atypical features. One gene (*CACNA1C*) showed a moderate level of evidence for causing LQTS.[20] LQT1 and LQT2 include loss-of-function mutations (*KCNQ1* and *KCNH2* genes, respectively) in the potassium channels, which cause decreased activity of the slow delayed rectifier current (IKs) and rapid delayed rectifier current (IKr) (phase 3 of the action potential), respectively. LQT3 includes gain-of-function mutations (*SCN5A* gene) in the sodium channel (phase 0 of an action potential) that cause persistent sodium influx that extends through the plateau phase. A loss of IKs or IKr function, or gain of INa function, in most cases, predisposes ventricular myocytes to early afterdepolarizations (EADs). When regions of the myocardium develop EADs synchronously, it can trigger lethal ventricular arrhythmias, including *torsades de pointes*.[21]

The characteristic electrocardiographic feature of congenital long QT syndrome is a prolonged corrected QT (QTc) interval, reflecting delayed ventricular repolarization, as shown in Figure 9.

Physiological changes that hinder effective CPR in pregnant women are as follows:

- Compression of the aorta and vena cava (aortocaval compression) by the gravid uterus can occur in the supine position, diminishing the amount of blood pumped by each cardiac contraction
- Pulmonary residual capacity (pulmonary volume that is available for gas exchange) is reduced by 20% at the end of pregnancy. At the same time, there is an increase in demand for oxygen of 20%
- For this reason, pregnant women develop hypoxia more rapidly than non-pregnant women
- The risk of pulmonary aspiration of gastric contents, which is complicated by difficult oxygenation as well as infectious complications, is also increased in pregnancy.

## TREATMENT

The advanced life support approach in cardiac arrest management begins with the identification of precursor symptoms. This is followed by early access to the emergency response system, early cardiopulmonary resuscitation or early defibrillation, and early reperfusion. The process then continues with complete revascularisation, hemodynamic support, and concludes with hypothermia for 24 hours.

The advanced life support approach to cardiac arrest management follows a structured algorithm that includes assessment of cardiac rhythm, high-quality CPR, defibrillation for shockable rhythms, appropriate pharmacological therapy, advanced airway management, and treatment of reversible causes [Figure 10].

### Basic Life Support (BLS)

Treatment for those who are certified to practice BLS includes treatment as above, with the addition of ventilation during active CPR. Current guidelines recommend 2 breaths for every 30 compressions (30:2).[22] Providers can also

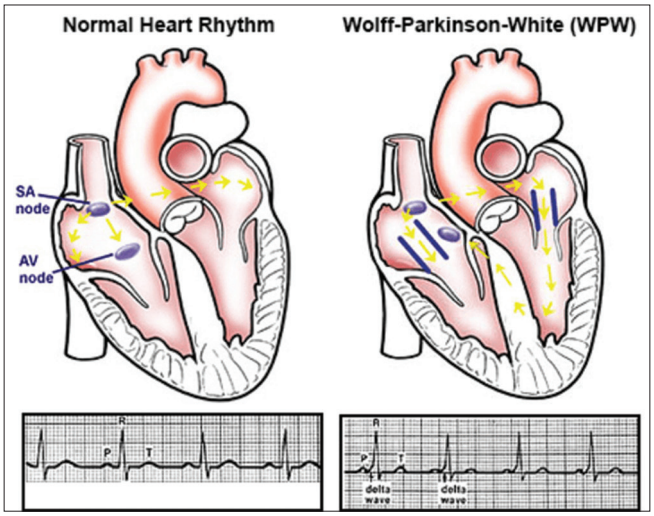



**Figure 8:** Wolff–Parkinson–White syndrome

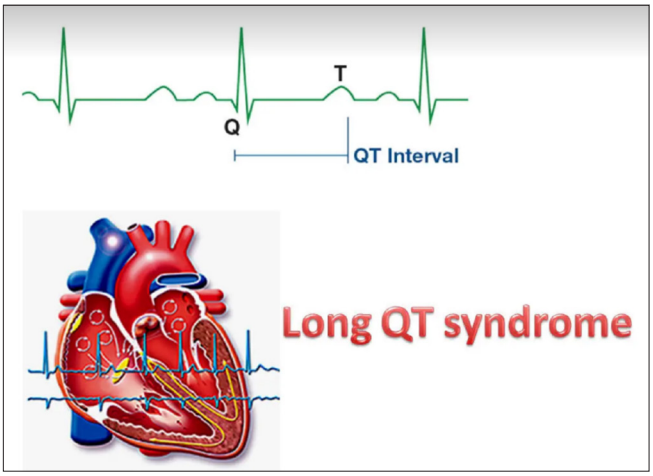



**Figure 9:** Congenital long QT syndrome

manipulate the airway to aid in airway patency, thus allowing for proper ventilation. These maneuvers include the head-tilt, chin-lift,[23] and the jaw thrust.[24] Oral airway adjuncts, including the oral pharyngeal airway and the nasopharyngeal airway, should also be utilized to benefit ventilation.

## Advanced Life Support (ALS)

Providers can use BLS treatment with the addition of medications and advanced airways, including supraglottic airway devices (King LT, Igel) and endotracheal intubation. Medications used in cardiac arrest include Epinephrine and Amiodarone. ALS providers have the additional benefit of cardiac rhythm interpretation, allowing for quicker defibrillation if indicated. Advanced cardiac life support can teach providers the algorithms used to resuscitate a patient in cardiac arrest.

### *Ischemic coronary disease – Medical management*

Short and long-acting nitrates, β-blockers, calcium channel antagonists, lipid-lowering medications, anticoagulants, and angiotensin-converting enzyme inhibitors (ACE inhibitors) are the primary pharmacological classes employed. Medication to avoid cardiovascular events and at least one antianginal/anti-ischemic medication are part of general conservative treatment.

#### *Surgical management*

Coronary artery bypass grafting and coronary revascularization.

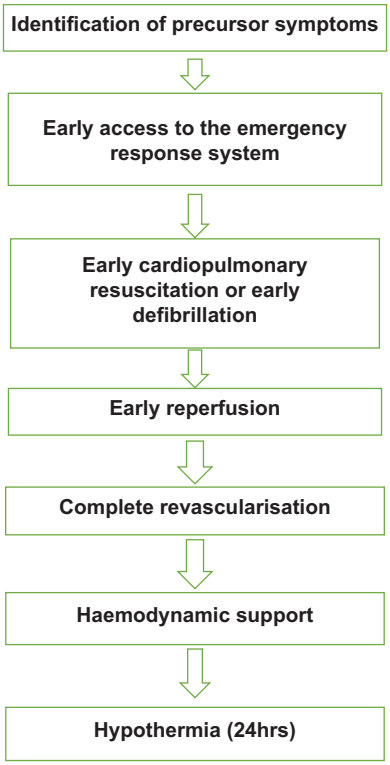



**Figure 10:** Advanced life support approach in cardiac arrest management

The commonly used pharmacological therapies for ischemic coronary disease and their therapeutic roles are summarized in Table 1.

### *LVH – Medical management*

Pharmacological management of left ventricular hypertrophy primarily focuses on treating the underlying cause, controlling blood pressure, reducing cardiac workload, and preventing cardiovascular complications, as summarized in Table 2.

These recommendations, which include the use of ACE inhibitors, β-adrenergic blockers, diuretics to relieve fluid overload, and digoxin to relieve persistent symptoms, should be followed in the treatment of heart failure patients with LVH and systolic dysfunction. In cases of advanced heart failure, spirolactone may be taken into consideration.

### *Congestive heart failure – Medical management*

The pharmacological management of congestive heart failure includes therapies aimed at reducing congestion, improving cardiac function, decreasing morbidity and mortality, and managing underlying cardiovascular risk factors, as summarized in Table 3.

Pharmacological heart failure treatment has evolved into a symptomatic and preventative management approach.

**Table 1:** Pharmacological management of ischemic coronary disease

| Category | Examples |
|---|---|
| 1. B-blockers | Atenolol, Bisoprolol, Metoprolol |
| 2. Nitrates | Nitroglycerin |
| 3. Calcium channel blockers | Diltiazem, Verapamil |
| 4. Potassium channel openers | Nicorandil |
| 5. Antiplatelet drugs | Aspirin |
| 6. HCN channel blockers | Ivabradine |

**Table 2:** Pharmacological management of left ventricular hypertrophy

| Category | Examples |
|---|---|
| 1. Statins | Atorvastatin, Simvastatin, Rosuvastatin. |
| 2. Calcium channel blockers | Amlodipine, Diltiazem, Verapamil. |
| 3. Diuretics | Furosemide, Spironolactone. |
| 4. B-blockers | Metoprolol, Carvedilol, Atenolol. |
| 5. Angiotensin-converting enzyme Inhibitors | Enalapril, Ramipril. |
| 6. Angiotensin receptor blockers | Losartan, Telmisartan, Valsartan. |

Diuretics, β-blockers, and ACE inhibitors are now commonly used in treatment. Digoxin is used to alleviate clinical symptoms, particularly in atrial fibrillation patients. If ACE inhibitors are not tolerated, hydralazine and isosorbide are advised. Only in cases when ACE inhibitors are intolerable (cough or angioedema) are angiotensin receptor blockers advised. It is crucial to combine lifestyle modification with early detection and preventative therapy.

### *Cardiac tamponade*

#### *Medical management: Pericardiocentesis*[25]

After pericardiocentesis or pericardiotomy, NSAIDs and colchicine can be considered to prevent recurrence and effusive–constrictive pericarditis.[26]

#### *Surgical management*

Surgical options include creating a pericardial window or removing the pericardium. Emergency department resuscitative thoracotomy and the opening of the pericardial sac is a therapy that can be used in traumatic arrests with suspected or confirmed cardiac tamponade. Volume resuscitation and pressor support may be helpful; however, these are temporizing measures that should be performed while preparing for definitive treatment with one of the above procedures.[27]

**Table 3:** Pharmacological management of congestive heart failure

| Category | Examples |
|---|---|
| 1. Diuretics | Furosemide, Bumetanide, Ethacrynic acid, Hydrochlorothiazide and Metolazone, Spironolactone. |
| 2. B-blockers | Carvedilol |
| 3. Vasodilators and nitrates | Combination of hydralazine and nitrate |
| 4. ACE Inhibitors | Ramipril |

**Table 4:** Pharmacological management of Wolff–Parkinson–White syndrome

| Category | Examples |
|---|---|
| Class 1A antiarrhythmic drugs | Procainamide, Quinidine, Disopyramide |
| Class 1C antiarrhythmic drugs | Encainide hydrochloride 52 and Flecainide acetate |
| Digitalis glycosides | Digitalis, Digoxin |
| Calcium channel blockers | Verapamil[30] |

**Table 5:** Pharmacological management of congenital long QT syndrome

| Category | Examples |
|---|---|
| B-blockers (non-selective) | Nadolol, Propranolol. |
| B-blockers (Selective) | Propranolol, Metoprolol, Atenolol. |
| Sodium channel blockers | Mexiletine, Flecainide, and Ranolazine. |
| Cystic fibrosis transmembrane conductance regulator corrector | Lumacaftor |

### *Brugada syndrome*

An implantable cardioverter-defibrillator (ICD) is the mainstay of treatment of Brugada syndrome patients. Quinidine is useful in Brugada syndrome patients with an ICD who experience multiple shocks and in those who have contraindications for ICD placement. Radiofrequency ablation of the anterior part of the right ventricular outflow tract is a new, emerging therapy with a promising prognosis for Brugada syndrome patients.[28]

### *Wolff–Parkinson–White syndrome*

Treatment must be individualized for each patient and should include individual risk assessment. Appropriate therapy for WPW syndrome is based on the likely prognosis and on the degree of symptoms the patient experiences.[29]

The pharmacological management of Wolff–Parkinson–White (WPW) syndrome depends on the type of arrhythmia and the patient's clinical status, with specific therapies used for the management of associated tachyarrhythmias, as summarized in Table 4.

### *CLQTS – Medical management*

The pharmacological approaches used in the management of congenital long QT syndrome are summarized in Table 5.

There isn't a causative treatment for LQTS at the moment. Lifestyle changes, pharmacological therapy with a focus on beta-blockers, device therapy, and surgical therapy are currently available therapeutic options. The most frequent cause of major cardiac events, such as an abrupt rise in sympathetic activity, which is primarily controlled by the left cardiac sympathetic nerves, is addressed by beta-blockers as the first-line treatment option.

#### *Surgical management*

- Left cardiac sympathetic denervation
- Implantable cardioverter-defibrillator

## PREVENTIVE MEASURES

Multiple strategies have evolved to predict and prevent SCD. One strategy receiving recent emphasis is primordial prevention of CAD. This approach stresses prevention of development of risk factors for cardiovascular disease and stroke, as represented in the term "*primordial prevention*." Optimization of blood pressure, weight, glucose, cholesterol, smoking, diet, and physical activity, through lifestyle

interventions, is the focus of this approach.[31] Use of the ICD has been demonstrated to reduce sudden death and improve total mortality in selected patient populations, including those with impaired ventricular function and those with ischemic or non-ischemic cardiomyopathy.[32]

## CONCLUSION

Cardiac arrest remains a major public health challenge due to its high mortality rate and sudden onset. It can result from a variety of cardiac and non-cardiac conditions, with IHD being the most common underlying cause. Early recognition of warning signs, prompt initiation of CPR, rapid defibrillation, and timely ALS are critical determinants of survival. A thorough understanding of the diverse etiologies and pathophysiological mechanisms enables more effective prevention and management strategies. In addition, risk factor modification, regular cardiovascular assessment in high-risk individuals, and the appropriate use of ICDs can significantly reduce the incidence of sudden cardiac death. Continued public education, improved emergency response systems, and adherence to evidence-based guidelines are essential to enhance patient outcomes and decrease the global burden of cardiac arrest.

## REFERENCES


1. Zipes DP, Wellens HJ. Sudden cardiac death. Circulation 1998;98:2334-51.
2. Field JM, Hazinski MF, Sayre MR, Chameides L, Schexnayder SM, Hemphill R, *et al*. Part 1: Executive summary: 2010 American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2010;122 Suppl 3:S640-56.
3. Pura A, Wilk M, Olszowska M. Prognostic value of inflammatory markers in acute coronary syndrome in a population with premature cardiovascular disease. J Rare Cardiovasc Dis 2019;4:37-41.
4. Raja S, Mustafa MA, Ghadir GK, Al-Tmimi HM, Alani ZK, Rusho MA, *et al*. Unlocking the potential of polymer 3D printed electronics: Challenges and solutions. Appl Chem Eng 2024;7:3877.
5. Centers for Disease Control and Prevention (CDC). State-specific mortality from sudden cardiac death--United States, 1999. MMWR Morb Mortal Wkly Rep 2002;51:123-6.
6. Chu J, Johnston TA, Geoghegan J, Royal College of Obstetricians and Gynaecologists. Maternal collapse in pregnancy and the puerperium: Green-top guideline no. 56. BJOG 2020;127:e14-52.
7. Semsarian C, Sweeting J, Ackerman M. Sudden cardiac death in athletes. Br J Sports Med 2015;49:1017-23.
8. Severino P, D'Amato A, Pucci M. Department of Clinical, Internal, Anesthesiology and Cardiovascular Sciences. Vol. 155. Rome, Italy: Sapienza University of Rome, Viale del Policlinico; 2024. p. 00161.
9. Bornstein AB, Rao SS, Marwaha K. Maharishi University of Management. Netherlands: Maharishi Vedic University Press; 2024.
10. Remme WJ. Congestive heart failure--pathophysiology and medical treatment. J Cardiovasc Pharmacol 1986;8:S36-52.
11. Fontaine G, Guiraudon G, Frank R, Vedel J, Grosgogeat Y, Cabrol C, *et al*. Stimulation studies and epicardial mapping in ventricular tachycardia: Study of mechanisms and selection for surgery. In: Kulbertus HE, editor. Reentrant Arrhythmias. Lancaster: MTP Publication; 1977. p. e334-50.
12. Eric L, Anderson LT, In: Nava A, Rossi L, Thiene G, editors. Arrhythmogenic Right Ventricular Cardiomyopathy/Dysplasia. Amsterdam: Elsevier; 1997.
13. Nishimura RA, Holmes DR. Clinica`l practice. Hypertrophic obstructive cardiomyopathy. N Engl J Med 2004;350:1320-7.
14. Ameli S, Shah PK. Cardiac tamponade. Pathophysiology, diagnosis, and management. Cardiol Clin 1991;9:665-74.
15. Goldstein JA. Cardiac tamponade in the interventional era: A paradigm shift in etiology and outcomes. Catheter Cardiovasc Interv 2020;95:387-8.
16. Sieira J, Dendramis G, Brugada P. The definition of the Brugada syndrome. European Heart Journal, 2017;38.
17. Maslova AY, Bazaeva KL, Abdullaeva ZA, Khazamova SO, Zeusheva KA, Grechkina TA, *et al*. Astrocytes and their phenomenal possibilities in the treatment of various neurodegenerative disorders: An overview. J Pharm Res Int 2021;33:60-8.
18. Prashanth PS, Jyotsna A. Department of Pharmacy Practice, Avanthi Institute of Pharmaceutical Sciences, Tagarapuvalasa, Vizayanagaram Dist, AP, India. 2020.
19. Schwartz PJ, Periti M, Malliani A. The long QT syndrome. Am Heart J 1975;89:378-90.
20. Adler A, Novelli V, Amin AS, Abiusi E, Care M, Nannenberg EA, *et al*. An international, multicentered, evidence-based reappraisal of genes reported to cause congenital long QT syndrome. Circulation 2020;141:418-28.
21. Bohnen MS, Peng G, Robey SH, Terrenoire C, Iyer V, Sampson KJ, *et al*. Molecular pathophysiology of congenital long QT syndrome. Physiol Rev 2017;97:89-134.
22. Kleinman ME, Brennan EE, Goldberger ZD, Swor RA, Terry M, Bobrow BJ, *et al*. Part 5: Adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2015;132 18 Suppl 2:S414-35.
23. Guildner CW. Resuscitation--opening the airway. A comparative study of techniques for opening an airway obstructed by the tongue. JACEP 1976;5:588-90.
24. Uzun L, Ugur MB, Altunkaya H, Ozer Y, Ozkocak I, Demirel CB. Effectiveness of the jaw-thrust maneuver

in opening the airway: A flexible fiberoptic endoscopic study. ORL J Otorhinolaryngol Relat Spec 2005;67:39-44.
25. Imazio M, Gaita F. Diagnosis and treatment of pericarditis. Heart 2015;101:1159-68.
26. Adler Y, Charron P, Imazio M, Badano L, Barón-Esquivias G, Bogaert J, *et al*. 2015 ESC guidelines for the diagnosis and management of pericardial diseases: The task force for the diagnosis and management of pericardial diseases of the European society of cardiology (ESC)Endorsed by: The European association for cardio-thoracic surgery (EACTS). Eur Heart J 2015;36:2921-64.
27. Ancion A, Robinet S, Lancellotti P. Cardiac tamponade. Rev Med Liege 2018;73:277-82.
28. Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: A distinct clinical and electrocardiographic syndrome. A multicenter report. J Am Coll Cardiol 1992;20:1391-6.
29. Christopher R, Ellis MD, FACC, FHRS. Assistant Professor of Medicine, Cardiac Electrophysiology, Director of Clinical Arrhythmia Research, Vanderbilt Heart and Vascular Institute. 2024.
30. Arai A, Kron J. Current management of the Wolff-Parkinson-White syndrome. West J Med 1990;152:383-91.
31. Lloyd-Jones DM, Hong Y, Labarthe D, Mozaffarian D, Appel LJ, Van Horn L, *et al*. Defining and setting national goals for cardiovascular health promotion and disease reduction: The American heart association's strategic impact goal through 2020 and beyond. Circulation 2010;121:586-613.
32. Epstein AE, DiMarco JP, Ellenbogen KA, Estes NA, Freedman RA, Gettes LS, *et al*. American college of cardiology/American heart association task force on practice guidelines J Am Coll Cardiol 2008;51:e1-62.

**Source of Support:** Nil. **Conflicts of Interest:** None declared.