

# Sudden Cardiac Death: Can We Solve the Puzzle?

Ajita Suhrid Annachhatre<sup>1</sup>

<sup>1</sup>Department of Cardiothoracic and Vascular Surgery, Mahatma Gandhi Mission, Medical Centre and Research Institute Medical College and Hospitals, Aurangabad, Maharashtra, India

**Address for correspondence** Ajita Suhrid Annachhatre, DNB, FIACTA, FICCC, Department of Cardiothoracic and Vascular Surgery, Mahatma Gandhi Mission's Medical college, Medical centre and Research Institute and Hospitals, N-6, Cidco, Aurangabad 431003, Maharashtra, India (e-mail: sannachhatre@gmail.com).

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## Abstract

Sudden cardiac death is a leading cause of death worldwide. That is the reason it requires more focus on predicting the risk and identifying susceptible candidates to optimize risk and prevent the catastrophic events like sudden cardiac arrest (SCA) and sudden cardiac death. Role of cardiopulmonary resuscitation (CPR) is vital in such events. Several measures have been taken all over the world to make every person aware of SCA and cardiac compressions only CPR. Immediate intervention and advance cardiac life support requires for successful outcome. In this review we have studied the etiology, predictors, and treatment of sudden cardiac death.

## Keywords

- ▶ sudden cardiac death
- ▶ sudden cardiac arrest
- ▶ cardiovascular collapse

## Introduction

Sudden cardiac death is the leading cause of premature loss of life. It is major and serious national as well as international health care issue. At present international data figures show sudden cardiac death rate as 15 to 20% among all deaths.<sup>1</sup>

Sudden cardiac death can be defined as natural, unexpected, and irreversible death from cardiac and noncardiac causes which occurs within one hour of symptoms onset where witnessed or individual was observed to be alive within previous 24 hour.<sup>2</sup> Sudden cardiac death has cardiac and noncardiac etiological factors.

In India, autopsy rate shows incidence of sudden cardiac death in individuals older than 35 years is 39.7/100,000. Incidence of out of hospital sudden cardiac death are ~3,60,000 each year.<sup>3</sup> Sudden cardiac death rate in pediatric population ranges from 0.6 to 6.2 per 100,000 children in the United States.<sup>4</sup>

Differential diagnosis for sudden cardiac death (SCD) are cardiovascular collapse and sudden cardiac arrest. Cardiovascular collapse is sudden loss of effective blood flow due to cardiac and/or peripheral vascular factors that may reverse spontaneously or require intervention, for example, vasovagalsyncope, neurocardiogenic syncope. Sudden cardiac arrest (SCA) is defined as abrupt cessation of cardiac mechanical function which may be reversible by a prompt intervention but will lead to death in its absence. Sudden

cardiac death is Sudden, irreversible cessation of all biological functions.<sup>1</sup>

Incidence of sudden cardiac death is decreasing since last three decades, as predictors of risk factors and etiological factors are established. At present there is no gold standard treatment available to prevent sudden cardiac death.

Here we tried to review the incidence, epidemiology, etiology, and recent trends in management of sudden cardiac death.

## Epidemiology of Sudden Cardiac Death

Incidence of SCD or SCA is difficult to estimate because of diversity in protocols to manage this situation worldwide.<sup>5</sup> Many countries conduct national registries to document SCD, many trials have tried to estimate the SCD. It has been seen that the studies are more conducted in western developed countries of Europe and America. Very few trials are mentioned in literature regarding SCD from Asian countries like China, India and Japan. Other Asian countries like Pakistan, Nepal, and Sri Lanka have no data regarding SCD. Evaluation mainly depends on the verbal questionnaires collected from the bystander of victim, EMS personnel attending the out of hospital cardiac arrest (OHCA) victim, and death certification records. All these data lack uniformity of the management and treatment protocols.<sup>6</sup>

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Worldwide incidence of SCD is as follows:

- 111.9 per 100,000 person-years in Australia
- 98.1 per 100,000 person-years in North America
- 86.4 per 100,000 person-years in Europe
- 52.5 Per 100,000 person-years in Asia

This number in Asian countries is underestimated as there are no proper registries or availability of EMS service, uniform guidelines for death certifications by physicians, and autopsy protocols.<sup>7</sup> Autopsies are done in few conditions only. Autopsy rate is 10% in United States and 23.8% in Finland.<sup>8,9</sup> Academic autopsy is not a routine trend in India and other Asian countries. Autopsies are done in medicolegal cases only. Noncardiac causes of SCD require autopsies to confirm the diagnosis. These are the problems because of nonavailability of funds, resources, infrastructure like EMS service, and automated external defibrillators, social educational level and economical conditions, and CPR awareness and training in lay person. Many studies were done in United States, China, Netherland, and Ireland with standardized definition and protocols which showed China has lowest rate of SCD.<sup>7,10-12</sup> These studies mentioned incidence of SCD 40 to 100 per 100,000 per year in general population, and incidence of SCD is 1 to 3 per 100,000 in age less than 35 years.<sup>13-15</sup>

Regional variations with geographical differences in incidence of SCD are also noticed.<sup>6</sup>

### Demographic Incidences of SCD

#### Age

SCD constitutes 15 to 20% of all deaths. The incidence in general population is 40 to 100 per 100,000 in year with <1% occurring in age less than 35 years.<sup>15,16</sup>

#### Sex

Females have lower incidence of SCD and SCA than males.<sup>17</sup> Studies have shown that females who have high risk factors for SCD too have low incidence of SCD and high chances of survival.<sup>18,19,20</sup> Estrogen and small body size are the main differential factors for favorable CPR and post resuscitation care response.<sup>21</sup>

#### Race

Recent large cohort study had concluded that African-Americans had a significantly higher risk for SCD than Caucasians. African-American women are at high risk for SCD. African-Americans have ~65% of the increased risk of SCD mostly because of socioeconomic factors and cardiovascular disease incidences.<sup>22</sup>

Hispanic Americans have lower incidences of SCD than non-Hispanics. Asian population in United States have lower incidence of SCD. China and Japan have lower incidences of SCD than North America.<sup>10,23</sup>

### SCD in Sports and Athletes

The incidence of SCDs in sportsperson and athletes was reported to be 4.6 per 1 million population in a 5-year prospective study as compared with 50 to 100 per million in the general population.<sup>24</sup> The incidence risk factors of sudden death vary with the age, sex, race, ethnicity, training level of athlete, and the type of sport. Most sports-related SCD occurs in the middle-age group of 35 years or older or recreational athletes, and 80% of these deaths are due to atherosclerotic coronary heart disease (CHD).<sup>25</sup>

The incidence of SCD or SCA during training for the sports event is five-fold higher. SCA or SCD is higher in occasional runners (1 per 7,500–18,000) than with trained marathon runners (1 per 50,000–200,000).<sup>26</sup> Previously hypertrophic cardiomyopathy (HCM) has been considered as the commonest cause for SCD in athletes, but it has been proved that many other cardiac and noncardiac causes lead to SCD in athletes and not only HCM.

### Etiology of Sudden Cardiac Death

SCD occurs predominantly in cardiac disease patients. Approximately 52 to 55% SCD victims had no cardiovascular disease diagnosed before the event. There are many noncardiac causes which present as SCD. The causes can be summarized in ►Table 1.

#### Coronary Heart Diseases

SCD is commonest with structural heart disease in which 75 to 80% victims have CHD and 10 to 15% have nonischemic dilated cardiomyopathies.<sup>27</sup> SCD occurs in 10 to 15% in victims without any structural heart disease like sudden arrhythmic cardiac arrest.

Four main risk factors for SCD in CHD patients are as follows:

1. Syncope as first presentation episode of myocardial infarction because of transient ischemia.
2. New York Heart Association (NYHA) class III or IV, ischemic dilated cardiomyopathies.
3. Ventriculartachycardia/fibrillation occurring early after myocardial infarction probably scar related and ischemic cardiomyopathies (3 days–2 months).

**Table 1** Etiology of sudden cardiac death

Cardiovascular causes	Nonatherosclerotic cardiovascular causes	Electrophysiological and conduction defects
<ul style="list-style-type: none"> <li>• Coronary Heart disease</li> <li>• Myocardial infarction</li> <li>• Anomalous coronary artery</li> <li>• Cardiomyopathies</li> <li>• Valvular heart disease</li> <li>• Heart failure</li> <li>• Myocarditis</li> </ul>	<ul style="list-style-type: none"> <li>• Coronary artery embolism</li> <li>• Systemic and autoimmune heart diseases.</li> <li>• Pulmonary artery embolism</li> <li>• Cardiac toxicity</li> </ul>	<ul style="list-style-type: none"> <li>• W-PW- Syndrome</li> <li>• Long QT syndrome</li> <li>• Brugada Syndrome</li> <li>• Catecholaminergic Polymorphic ventricular tachycardia</li> <li>• Early repolarization syndrome</li> </ul>

4. History of previous myocardial infarctions because multivessel coronary disease and scarring of myocardium.

Framingham cohort study was initial large study which reviewed SCD cases from 1950 to 1999 and concluded 20 to 30% SCD occurred in coronary artery disease patients. Autopsy reports of SCD patients have reported pathological entities like plaque fissure, plaque, hemorrhage, and thrombosis in 95% of patients who died immediately.

Patients with diagnosed hypertension, diabetes mellitus, hyperlipidemia, obesity, and smoking are at risk to develop atherosclerotic coronary artery diseases.<sup>28</sup>

SCD in such patient group can occur because of acute coronary syndrome.

## Management and Treatment

SCD occurred in known CHD patients or low-risk heart disease patients should undergo evaluation in the form of electrocardiogram, percutaneous coronary angiography, and rescue angioplasty in case of tight stenosis of the coronary arteries. These are helpful therapeutic interventions to treat culprit vessel and increases the outcome after SCD in myocardial infarction or acute coronary event. Advance cardiac life support (ACLS) as per the guidelines of American College of Cardiology (ACC). American Heart Association (AHA) is the main crux of the life-saving treatment while conducting the interventions. Incidence of out of hospital cardiac arrest in known CHD patients is 60% and survival at discharge is 10%.

Nonatherosclerotic coronary abnormalities are as follows:

1. Coronary artery embolism
2. Coronary artery spasm
3. Myocardial bridging
4. Coronary arteritis, that is, Kawasaki disease, polyarteritis nodosa.
5. Congenital coronary anomalies, that is, anomalous origin of left coronary artery accounts 17% incidence of SCD in young athletes.

## Cardiomyopathies

Three major cardiomyopathies cause SCD:

1. Nonischemic dilated cardiomyopathy (NIDCM)
2. Hypertrophic cardiomyopathy (HCM)
3. Arrhythmogenic right ventricular cardiomyopathy or dysplasia (ARVC/ARVD)

### Nonischemic Dilated Cardiomyopathy

Nonischemic cardiomyopathies are the second most frequent cause of SCD with incidence of 10 to 15%.<sup>7</sup> Prevalence is 1:2,500.

It is defined as left ventricular dilatation and left ventricular systolic dysfunction in the absence of abnormal loading conditions or coronary artery disease resulting in global systolic impairment.

Causes of NIDCM are genetic mutations (TTN, MYH7, TNNT2, and LMNA), myocarditis, cardiac toxicity (by alcohol, metals, chemotherapeutic agents), and systemic and autoimmune disorders.

Treatment implantable cardioverter-defibrillator (ICD) therapy. Large meta-analytical studies had shown the reduction in mortality with SCD in NIDCM with ejection fraction <35%.<sup>29,30</sup>

### Hypertrophic Cardiomyopathy

Is defined as increased left ventricular wall thickness >30 mm with the prevalence of 1:500.<sup>31</sup> It is an inherited condition and commonest cause of SCD in young population of less than 35 years and uncommon in older population causing death rate of 0.5 to 2% annually.<sup>32</sup>

Risk factors for SCD in HCM patients are (1) history of unexplained syncope, (2) SCD in family, (3) left ventricular wall thickness of >30 mm, (4) repetitive nonsustained VT, and (5) exercise-induced abnormal high blood pressures.

The primary prevention of SCD by ICD implantation in such patients is advised by ACC foundation and AHA guidelines. European guidelines too recommends implantation of ICD in HCM patients based on risk prediction model.<sup>32,33</sup>

### Arrhythmogenic Right Ventricular Cardiomyopathy or Dysplasia

Arrhythmogenic right ventricular cardiomyopathy or dysplasia (ARVC/ARVD) is genetic disease of heart muscle disorder in which there is fibrofatty replacement of right ventricular myocardial muscle.

Prevalence is 1:2,000 to 1:5,000.<sup>34</sup> Autosomal dominant trait in 60% patients involving nearly 15 genes mutation. ARVC has major and minor criteria in six categories: (1) Tissue characterization, (2) structural alteration, (3) depolarization abnormalities, (4) repolarization abnormality, (5) arrhythmia, (6) genetic diagnosis.

SCD occurs as first arrhythmic event in age group of 40 years in 50% of cases.

## Treatment

The prophylactic use of an ICD (in those who have  $\geq 1$  risk factors for SCD) is recommended by Current American College of Cardiology Foundation/American Heart Association/Heart Rhythm Society guidelines.<sup>35</sup> Valvular heart disease comprises 1 to 5% of total SCD.

Aortic stenosis patients are at more risk of SCD with 1 to 3% incidence. Post valve replacement surgery of aortic valve the incidence of SCD is 15 to 30% because of ventricular arrhythmias.<sup>36,37</sup>

Mitral valve prolapsed patients also have shown increased incidence of SCD in women.<sup>38</sup>

## Arrhythmias and Conduction Abnormalities

Sudden cardiac death victim autopsies negative for structural heart disease constitutes nearly 29 to 43%.<sup>13,14</sup> Autopsies

detail histologic examination diagnosed focal myocarditis, regional ARVC, and conduction system abnormalities.

Causes of SCD secondary to arrhythmias are as follows:

1. Electrophysiological abnormalities, that is, Wolf–Parkinsons–White syndrome.
2. Inherited disorders of electrophysiological abnormalities:
  - Congenital long-QT syndrome.
  - Brugada syndrome.
  - Catecholaminergic polymorphic ventricular tachycardia.
  - Early repolarization syndrome.

**Wolf–Parkinsons–White syndrome:** This syndrome causes sudden pre-excitation, tachyarrhythmias, and SCD. It is because of presence of atrioventricular accessory pathway. Incidence of SCD is low, that is, 0.15 to 0.39%. Atrioventricular re-entrant tachycardia is the commonest arrhythmia in this disease.

Diagnosis is on electrocardiogram (ECG)—short PR interval <0.12 second prolonged QRS complexes >0.12 seconds and characteristic slow rising and slurred QRS complexes, that is, “delta waves.”

**Long-QT syndrome (LQTS):** This is an inherited disorder. There is delayed myocardial repolarization results in prolongation in QTc interval (more than 0.45 s) on 12-lead ECG with predisposition to torsade de pointes and SCD.

Clinical features are of syncope, seizures, or palpitations during exercise or emotions. Sometime cardiac arrest can be first presentation.

There are three types of LQTS type 1, type 2, and type 3. Mutations in cardiac ion channels causing delayed rectifier current and the cardiac sodium channel present in 75% patients of LQTS but 25 to 30% patients have normal QTc interval at rest. These patients require catecholamine infusion to disclose the masked prolonged QTc interval. Schwartz et al, in their large study concluded the incidence of LQTS as 1:2,000 infants.

SCD and arrhythmic events developed during exercise or stress in type 1 with incidence 0.3% at rest or sudden noises in type 2 with incidence of 0.6% and at rest or during sleep in type 3 with incidence 0.56%.<sup>39</sup>

Treatment— $\beta$ -blockers are the mainstay of therapy. ICD is recommended in patients with prior history of cardiac arrest.

## Brugada Syndrome

Brugada syndrome (BrS) is another congenital hereditary disorder with genetic mutations in cardiac sodium channels. It typically affects middle-aged men with arrhythmic event developing in sleep at mean age of 40 years. So it was considered a mysterious cause of nocturnal death. It is also known as Pokkuri in Japanese, Bangungut in Philippines, and Lai Tai in Thai population.

Diagnosis of BrS or Brugada type 1 can be on ECG coved type of ST segment and J point elevation of >2 mm with negative T wave in the V1–V3 leads.

Recent multicenter study have concluded that incidence of SCD is 7.7 to 10.2% per year.<sup>40,41</sup> Treatment is ICD insertion to protect against SCD.

## Catecholaminergic Polymorphic VT

Familial arrhythmogenic condition presented with polymorphic ventricular tachyarrhythmias is induced by either physical or emotional stress. There is no structural heart disease with normal ECG at rest. Arrhythmic events can manifest as syncope, aborted SCA, and SCD in first or second decade of life. In untreated patients mortality can be as high as 30%.

Treatment— $\beta$ -blocker therapy, antiarrhythmic drug (e.g., flecainide), and ICDs.

## Early Repolarization Syndrome

This condition is characterized by J wave elevation > 0.1 mV, either notched or slurred, accompanied by an ST segment elevation.<sup>42</sup> It was considered as benign till 2,000, but recent expert consensus panel defined ERP as presence of J point elevation > 0.1 mV in > 2 contiguous inferior and lateral leads and diagnosed in a patient resuscitated from unexplained VF/polymorphic VT or autopsy-negative SCD victim with previous ECG demonstrating ERP. It is considered as a malignant condition.

Clinical features—Patients may diagnosed with arrhythmic attack, syncope or survival from SCA or incidental finding on ECG in asymptomatic patients.

Treatment: There is no current risk stratification strategy for asymptomatic patients with ER pattern in general population and screening of families which allows identification of higher risk individuals. Studies have shown efficacy of isoproterenol and ICDs to avoid malignant arrhythmias in ERS patients.<sup>43,44</sup>

## Clinical Characteristics of Sudden Cardiac Arrest

The course of symptoms can be divided in four phases as prodrome, onset, arrest, and death.

**Prodrome:** In this phase patients may experience angina, dyspnea, palpitations, easy fatigability, and other nonspecific complaints. These prodromal symptoms are predictive of cardiac event but not the SCD.

**Onset of the transition from symptoms to cardiac arrest** is defined as an acute change in cardiovascular status before 1 hour of cardiac arrest. In this phase many electrocardiographic changes occur, appearance of ventricular ectopics to ventricular tachycardias turning in to ventricular fibrillation, and malignant arrhythmia causing cardiac arrest. This cardiac arrest if immediately intervened with resuscitative measures can revive the patient and return of spontaneous circulation can be achieved. Delay of every minute in resuscitation measures can linearly decreases the successful outcome; a 5-minute delay in starting the therapeutic intervention decreases the chances of survival by 25 to 30% in out of hospital cardiac arrest (OHCA). Early defibrillation increases the chances of survival in shockable rhythm with prompt CPR.

Death: Progression to biological death is a function of mechanisms between cardiac arrest and the interval before starting the resuscitation measures. It is a crucial period as neuronal damage because of anoxic encephalopathy leads to poor outcomes.

## Triggers of SCD

### Physical Activity

It has been proved that regular exercise have protective role in not developing SCD, but vigorous exercise may cause SCD at 3 to 13%. Male gender is at more risk to have SCD during vigorous physical activity or even during sport activities than females.<sup>45</sup>

### Diurnal/Seasonal Variations

SCD incidence is more at 6.00 a.m. to noon. It is highest on Monday and lowest during weekend. The adrenergic levels are underlying cause of triggering this diurnal variation.

SCDs are highest in winter months and lowest in summer month on both hemispheres. In recent study done in Japan concluded 23.8% of OHCA at lower temperature, probable mechanism can be coronary spasm leading to SCD.<sup>46</sup>

### Psychosocial Determinants

Anxiety, depression, and schizophrenia have been directly associated with high incidence of SCD in men than in women. This can be correlated with some antipsychotic drugs having adverse cardiac effects.<sup>47</sup>

### Air Pollution

Air pollution exposure to particulates like carbon monoxide or oxides of nitrogen can cause increase risk of OHCA in United States and Australia. Long-term air pollution increases risk of CHD and mortality.<sup>48,49</sup>

## Predictors and Risk Factors for SCD

It is mandatory to know the etiology of SCD as well as the risk factors. We need to establish the predictors of high risk conditions which lead to SCD. Till date we are successful in finding out many causes of SCD, but the negative autopsies of SCD again raise the question about the reason of SCD. Here comes the role of establishments of predictors. Studies should be conducted in uniform protocols. Prediction of SCD will decrease the incidence of SCD.

We have established *clinical markers* which are high risk for SCD; we need more *novel plasma biomarkers* as predictors of SCD where the obvious reasons are not present in SCDs.

*Plasma biomarkers* can be categorized as follows:

1. Inflammatory markers, for example, C-reactive protein for development of coronary artery disease.
2. Blood lipids—proven association between hypercholesterolemia and coronary artery disease.
3. Hemodynamic markers, for example, natriuretic peptides.
4. Free fatty acids—nonesterified free fatty acids are thought to be proarrhythmogenic.

Predictors help us to do the prevention. Prevention of SCD is at two levels, primary prevention, and secondary prevention. Primary prevention refers to the attempt to identify individual patient at specific risk for SCD and institute preventive strategies, for example, implantable cardiac defibrillator in high risk patients.

Secondary prevention refers to measures taken to prevent recurrent cardiac arrest or death in individuals who survived a previous cardiac arrest.

Third category of prevention consists of intervention to avoiding progression of arrest to death, for example, OHCA response team and bystander CPR.

### SCD and Coronary Heart Disease

We have established clinical factors to stratify the low and high risk for SCD in CHD. Hypertension and left ventricular hypertrophy, diabetes mellitus, hypercholesterolemia, and smoking are predictors for high risk.

*Family history suggesting SCD* screening of other family members for congenital diseases and arrhythmic diseases can prevent SCD in susceptible individuals.

*Myocardial infarction* creatine phosphokinase-MB (CPK-MB), troponin C are the predictors of high risk.

Post myocardial infarction patients are at high risk for SCD in 48 hour to 30 days, and 30 days to 6 months. VALIANT trial has concluded that the risk of SCD post MI changes according to time interval in patients having MI with heart failure, poor LV ejection fraction <40%, or both. In this study, valsartan or captopril, aspirin,  $\beta$ -blockers, and statins are used at time interval and studied.<sup>50</sup>

### SCD and Heart Failure

We have established plasma biomarkers like renin levels, natriuretic peptides BNP and end-terminal pro-BNP elevation indicating heart failure either in low or preserved ejection fraction.

One recent study tried to established ECG markers for heart failure. ECG indices included turbulence slope (TS), reflecting autonomic dysfunction; T-wave alternans (TWA), reflecting ventricular repolarization instability; and T-peak-to-end restitution ( $\Delta\alpha Tpe$ ) and T-wave morphology restitution (TMR), both reflecting changes in dispersion of repolarization due to heart rate changes. These ECG markers capturing complementary proarrhythmic and pump failure mechanisms into risk models based only on standard clinical variables substantially improves prediction of SCD and PFD in CHF patients.<sup>51</sup>

### SCD and Congenital Heart Disease Patients

SCD is leading cause of death in adult with repaired lesions of CHD. In one study of 936 adults or repaired CHD, authors concluded the subaorticventricular systolic dysfunction is a dominant multivariate predictor of SCA and SCD. Prediction of subaortic ventricular systolic dysfunction can stratify the treatment regimens to prevent the SCA and SCD.<sup>52</sup>

### Atrial Fibrillation and SCD

Recent large cohort study mentioned the incidence rate of SCD in atrial fibrillation patients is 2.9 per 1,000 per year compared

with non-AF controls 1.3 per 1,000 per year.<sup>53</sup> The probable reason for SCD is coexisting heart failure in AF patient rather antiarrhythmic agents or cardiovascular disease.

### Renal Disease and Obstructive Sleep Apnea

Chronic renal failure has high prediction for SCD with annual rate of 5.5% in patients on dialysis.<sup>54</sup>

Obstructive sleep apnea (OSA) and seizure disorders also associated with SCD but need more exploration to establish predictors of SCD for these conditions.<sup>55,56</sup>

### Treatment for Sudden Cardiac Arrest

Sudden cardiac death is major burden of public health. Only 10% survival for OHCA for all ages of victims. Early recognition, activation of emergency services and early start of chest compression comprises basic life support treatment and have impact on survival in OHCA victims. AHA guidelines emphasizes chest compressions only bystander CPR, Indian resuscitation guidelines also gives importance on compression only life support COLS for lay people.<sup>57</sup>

Early defibrillation by EMS services is promptly available in developed western countries but in Asian countries like India, Pakistan, Nepal not available easily. SCD secondary to shockable arrhythmia will have good outcome with AED use and followed by CPR. Awareness of bystander CPR and AED use should be promoted to deal in OHCA.

In hospital cardiac arrest management should targeted goals like (1) identify and treat the cause of cardiac arrest, (2) restore and maintain hemodynamic stability, (3) management of complications of hypoperfusion, (4) coronary interventions in case of SCD secondary to coronary artery disease is useful, (5) targeted temperature management if feasible with the infrastructure.

ACLS algorithm and ventricular tachycardia and ventricular fibrillation treatment algorithm by AHA/ACC 2015 should consider for further management. European and Australian guidelines are also available for management of SCD.

### Post Resuscitation Care

SCA as presentation of myocardial infarction and ventricular fibrillation is responsive to treatment and may require ventilator support for short duration. Hemodynamic stability can achieve with antiarrhythmics and inotropic drug supports.

Recurrent VT/VF are less responsive to treatment and can result in hemodynamic instability resulting in bradyarrhythmias, PEA (pulseless electrical activity), and asystole.

OHCA victims can have anoxic encephalopathy which has very poor prognosis and strong predictor of in hospital death.

Outcome of in hospital cardiac arrest patients with coexisting diseases like end-stage renal diseases, terminal phases of cancer patients, septicemia have poor prognosis, survival  $\leq 10\%$ .

Long-term management after survival of cardiac arrest has prevention of recurrent cardiac arrest as the goal. Diagnosis of the cause and appropriate drug therapy with

periodic screening of risk predictors is important. Implantable cardioverter defibrillator have important prognostic role in preventing fatal arrhythmias in susceptible individuals.

### Conclusion

Incidence of SCD is at declining curve and the successful management in such cases showing improvements. We are trying to establish the etiology, predictors with risk factors, diagnosis, and management of SCD. Awareness in cardiac compressions cases only, CPR patients have been increased in developing countries too giving a green signal to the road blocks of SCD. Though worldwide uniform data collection and protocols to manage SCD are yet not available, in coming years it will be followed as the concern of SCD important for every individual. We can definitely be optimistic and positive in solving this puzzle.

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#### Conflict of Interest

None.

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