*Review Article*

# Sudden Cardiac Death (SCD) -A Raising Concern of Modern Cardiology

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## Introduction

Sudden cardiac death is a challenging problem to cardiologists all over the world. As it involves a widely varied array of clinical and pathological conditions, understanding the mechanism leading to sudden cardiac death poses a problem.

## Definition

Sudden cardiac death (SCD) generally refers to an unexpected death from a cardiovascular cause in a person with or without preexisting heart disease. The specificity of this definition varies depending on whether the event was witnessed; however, most studies include cases that are associated with a witnessed collapse, death occurring within 1 hour of an acute change in clinical status, or an unexpected death that occurred within the previous 24 hours[1-3]. The World Health Organization defines sudden death as death occurring within 24 hours of an abrupt change in previous clinical status[4].

## Epidemiology

The global incidence of sudden death is not known, but there are studies from different parts of the world addressing this issue. In the Western world (Europe and the USA), sudden cardiac death accounts for 20% of all mortality[5]. and about 50% of all deaths attributable to cardiovascular disease in the USA and other developed countries[6]. The incidence of SCD in the United States ranges between 180 000 and 450 000 cases annually[4]. These estimates vary owing to differences in SCD definitions and surveillance methods for case ascertainment[4,5]. In recent prospective studies using multiple sources in the United States,[6,7] Netherlands,[8] Ireland,[9] and China,[10] SCD rates range from 50 to 100 per 100 000 in the general population[3].

**Abnormalities associated with SCD**

SCD is a complex phenomenon and is believed to be the interaction between a transient event and underlying substrate. This process induces electric instability and lethal ventricular arrhythmias followed by hemodynamic collapse. CHD is the most common substrate underlying SCD in the Western world, being responsible for ≈75% of SCDs[8,11-13]. Cardiomyopathies (dilated, hypertrophic, and arrhythmogenic right ventricular cardiomyopathy) and primary electric disorders related to channelopathies account for most of the remainder[11]. In≈5% of SCDs or cardiac arrests, a significant cardiac abnormality is not found after extensive evaluation or at autopsy[14-17].

**CHD predisposes to SCD in 3 general settings:**

1. Acute myocardial infarction,
2. Ischemia without infarction, and
3. Structural alterations such as scar formation or ventricular dilatation secondary to prior infarction or chronic ischemia.

In those who die suddenly of CHD, 19% to 27% have pathological evidence for myocardial necrosis, and only 38% of cardiac arrest survivors will develop enzymatic evidence of myocardial infarction. In autopsy studies, stable plaques and chronic changes alone are found in≈ 50% of SCD patients with CHD.

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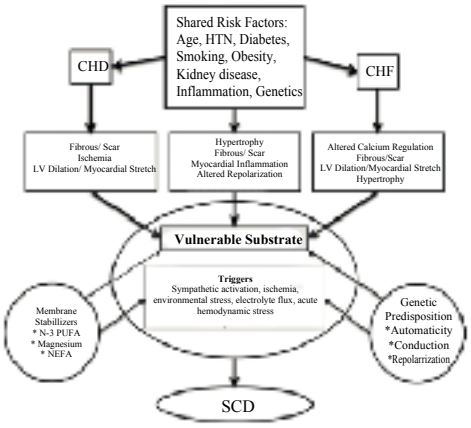
Suggesting that plaque rupture and acute mycoardial infarction (MI) is present in some, but not the majority, of S




Critical pathways leading to electric instabillity and sudden cardiac death. HTN indicates hypertension; CHD, coronary heart disease; CHF, congestive heart failure; LV, left ventricular; PUFA, polyunsaturated fatty acids; NEFA, nonesterified fatty acids; SCD sudden cardiac death.

# Risk Factors

SCD risk in the population is not only a function of the underlying substrate and its vulnerability to arrhythmias but also the frequency of exposure to acute precipitants or triggers[18].

### Diurnal/Seasonal Variation

Several studies have demonstrated a circadian pattern to the occurrence of SCD and out-of-hospital cardiac arrest. The peak incidence occurs in the morning hours from 6 AM to noon with a smaller peak in the late afternoon for out-off hospital VF arrests. This morning peak in SCD is blunted by ß blockers, supporting the concept that excessive activation of the sympathetic nervous system in the morning hours may be responsible[19-23].

### Physical Activity

Physical activity has both beneficial and adverse effects on SCD risk. Despite the long-term benefits of exercise, it is also well known that SCD occurs with a higher-than-average frequency during or shortly after vigorous exertion. Case-control and case-crossover studies performed among men have demonstrated that vigorous exertion can trigger cardiac arrest and SCD[24-26].

### Psychosocial Determinants

Lower socioeconomic status, depression, anxiety, social isolation, and psychological stress have all been linked to an increase in cardiovascular mortality in diverse populations[27,28].

### Genetic Predisposition to SCD

Over the past decade, investigations focused on the genetic bases of rare, inherited arrhythmic diseases (IADs) have provided insight into understanding the heritability of vulnerability to ventricular arrhythmias[15].

### Clinical features of the patient with cardiac arrest & SCD

Clinical cardiac arrest and SCD can be described in the framework of four phases of the event used to establish temporal definitions-prodrome, onset of the terminal event, the cardiac arrest, and Progression to biological death or survival.

### Prodromal Symptoms

Patients at risk for SCD can have prodromes such as chest pain, dyspnoea, weakness or fatigue, palpitations, syncope, and a number of nonspecific complaints[29].

### Onset of the Terminal Event

The period of 1 hour or less between acute changes in cardiovascular status and the cardiac arrest itself is defined as the "onset of the terminal event." Ambulatory recordings fortuitously obtained during the onset of an unexpected cardiac arrest have indicated dynamic changes in cardiac electrical activity during the minutes or hours before the event[29,30].

### Cardiac Arrest

Cardiac arrest is characterized by abrupt loss of consciousness caused by lack of adequate cerebral blood flow. It is an event that uniformly leads to death in the absence of an active intervention, although spontaneous reversions occur rarely. The most common cardiac mechanism is VF, followed by asystole, pulseless electrical activity and sustained VT. Other mechanisms include rupture of the ventricle, cardiac tamponade, acute mechanical obstruction to flow, and acute disruption of a major blood vessel[29].

### Progression to Biological Death

The time course for progression from cardiac arrest to biological death is related to the mechanism of the cardiac arrest, the nature of the underlying disease process, and the delay between onset and resuscitative efforts. The onset of irreversible brain damage usually begins within 4 to 6 minutes after loss of cerebral circulation related to unattended cardiac arrest, and biological death follows quickly[31-33].

### Mechanism of SCD

### Relationship between Structure and Function in SCD

The vast majority of patients who have experienced SCD have cardiac structural abnormalities. In the adult population, these consist predominantly of CHD, cardiomyopathies, valvular heart disease, and abnormalities of the conduction system[11-17].

**Tachyarrhythmias versus Bradyarrhythmias in SCD**

Ventricular fibrillation is the first recorded rhythm in approximately 75% of patients who have cardiac arrest[9]. Sustained ventricular tachycardia is only rarely (less than 2%) documented as the initial rhythm, but it is unknown how often it precedes and precipitates ventricular fibrillation. Electromechanical dissociation and asystole are found in approximately 30% of patients experiencing cardiac arrest, and this finding is usually related to the time interval from collapse to first monitoring of the rhythm, suggesting that it is often a later manifestation of cardiac arrest[9].

**Electrophysiologic Effects of Ischemia**

Within the first 3 days of myocardial infarction, SCD may occur as a result of ventricular fibrillation initiated by early, frequent premature ventricular complexes (PVCs). Such PVCs have been shown in experimental models to be predominantly caused by impulse formation consistent with abnormal automaticity[11-14].

**Mechanoelectrical Feedback**

Left ventricular dysfunction has been identified as the strongest independent predictor of SCD[10]. Despite the clinical recognition that acute heart failure can precipitate ventricular tachyarrhythmias, the mechanism by which this occurs is incompletely understood. Besides mechanisms related to acute and chronic ischemia, it has been shown that acute changes in the mechanical state of the heart related to altered preload and contractility can have direct electrophysiologic effects that may precipitate arrhythmias; this relationship is referred to as mechanoelectrical feedback[12].

**Reduction of SCD**

Incidence of SCD can be reduced by taking following measures-

**Evaluation of a Patient and Risk stratification-by**

**History**

A prior history of cardiac arrest is the most significant risk factor for recurrent cardiac arrest. Structural heart diseases which are considered as substrates for SCD, unexplained syncope.

**Family history**

Sibling having congenital long QT syncope hypertrophy in familial idiopathic cardiomyopathy, Drug history - that may prolong QT, In children- syncope, pre-syncope, dyspnoea, chest pain on exertion, palpitation-premonitoring features in children who die suddenly. Family with a history of syncope, arrhythmia, Marfan syndrome, HCM sudden or unexplained death.

**Investigations**

Coronary perfusion e.g. Coronary Angiogram (CAG), exercise Tolerance Test (ETT), 24 hours Holter monitoring, ST-T changes in ECG, Pump function e.g. LVEF, NYHA classification, Arrhythmias e.g. Holter, Signal averaged ECG, QT interval dispersion, T wave abnormality, Neurohumoral e.g. heart rate variability, baroreflex sensitivity, Psychosocial e.g. depression.

# Management

**1. Primary prevention:**

- **Pharmacological management**-B blocker and amiadarone-can reduce frequency of sudden cardiac death after MI. Statin, ACEi, aspirin, clopidogrel-Prevention of Plaque rupture, ACEi, Beta-blocker-Stabilizing Autonomic function. ACEi and B blocker- Improving pump function, B blocker & Anti-Arrhythmic drugs-Preventing arrhythmia.
- **Intervention:** PCI-PTCA with or without stenting-to reduce coronary heart disease, Automated Implantable Cardiac Defibrillator (AICD & CRT): Treatment of choice in patient of: Documented VT (non MI), VT (hemodynamically poor tolerated), History of unexplained syncope in impaired LV function, EP study & radiofrequency catheter ablation: to prevent sustained ventricular Arrhythmia.
- **iii. Cardiac surgery**-CABG and Arrhythmic surgery-cardiac reconstructive surgery.

**2. Secondary prevention**

If patients successfully resuscitated from an episode of cardiac arrest than secondary prevention is needed. As most causes of SCD occurs in the population of CAD, secondary prevention therapy is directed to the patient with CAD specially to survivors of AMI. Following measures should be taken- Anti-ischaemic drugs, Anti arrhythmic drugs, AICD & CRT, Revascularization-PTCA or CABG and Arrhythmic surgery such as cardiac reconstructive surgery.

**3. Resuscitation**

Out of Hospital- Promptness and restoring circulation is essential, it is measured by Basic Life support (BLS) followed by advanced cardiac life support (ACLS) and cardiac Defibrillator (At hospital).

## Conclusion

In conclusion, it is evident that as SCD is a challenge even in this era of modern cardiology with hi-tech support system. High degree of suspicion, proper & detail history taking, meticulous physical examination & necessary investigations are advised to triage the patient for the prevention of Sudden Cardiac Death (SCD) by cardiologists & at the same time of need to grow attention & awareness for in-time referral of risky patients for other physicians & also health education for all.

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