Review Article

Sudden Cardiac Death

Abstract

Background: Sudden cardiac death (SCD) remains a major open clinical and public health problem, with an estimated 300,000 deaths per year in the United States. The possibility of identifying potential SCD victims is limited by the large size of the large number of SCD victims and the apparent time-dependent risk of sudden death. The latter refers to the tendency of SCDs to detect other cardiovascular events during the most dangerous period of 6–18 months following a major cardiovascular event and the risk of subsequent collapse. The combination of time and lake size provides the basis for future research to find more vulnerable people. Pathologically, SCD can be seen as an interaction between some electrophysiological events that causes abnormalities in cardiac structure, temporal dysfunction, and malignant arrhythmias. Structural deformities represent an anatomical matrix of chronic risk and include the effects of electrophysiological anatomical abnormalities such as coronary artery disease, left ventricular hypertrophy, myopathic ventricles, and bypass leaflets in the myocardium.

Conclusion: Macroscopic cardiac features are common in about one-third of young SCD victims. However, in 79% of them, histological studies reveal hidden pathological features such as local myocarditis, heart disease and motor system disorders. A total of 16 (6%) victims had no evidence of systemic heart disease and the mechanism of SCD was not described.

***Keywords**: Arrhythmia, Cardiomyopathy, Conduction system, Myocarditis, Sudden death.*

Introduction

In addition to a genetic disorder called LQTS, there is a genetic mutation that determines a person's risk of fatal arrhythmia. In a prospective Paris study, a history of SCD in 1 parent was shown to increase the risk of fatal arrhythmia in children by 80%. A history of SCD in both parents increases the risk of SCD in children by 9 times. In a population-based study, the level of SCD in relatives of first-degree SCD sufferers was 50% higher than in control studies and, more importantly, from other risk factors such as diabetes, high blood pressure, or smoking. Free It is possible that several genes may contribute to the phenotypic expression of SCD. Participants may include myocardial ischemia, neurohomoral signaling, Ca2 + cardiomyocyte administration, and known or unknown genes associated with cardiac structure. Once genetic traits are identified through genomic testing, a major challenge will be to determine whether a particular gene carries a specific risk factor for SCD in addition to primary heart disease and whether it is involved in the pathogenesis of malignant arrhythmias (1).

Causes of Sudden Cardiac Death

There are various causes of sudden cardiac death in adolescents. Death often results from a heart attack. The heart can get out of control for a variety of reasons. This abnormal heart rate is known as ventricular fibrillation. Other direct causes of sudden cardiac death in adolescence include hypertrophic cardiomyopathy (HCM). In this often inherited condition, the wall of the heart muscle contracts. Stress can disrupt the cardiovascular system and cause a fast or abnormal heartbeat (arrhythmia) that can lead to sudden cardiac death. Hypertrophic cardiomyopathy, which is usually fatal, is the most common cause of sudden cardiac death in people under the age of 30. This is a common cause of sudden death in athletes. HCM is often not detected. Abnormal coronary arteries. Some people are born with abnormalities in the coronary arteries. The arteries can be compressed during exercise and do not provide sufficient blood flow to the heart. Long QT syndrome. This hereditary rhythm disorder causes a rapid and disturbed heartbeat, which can often lead to seizures. Young people with chronic long QT syndrome are at increased risk of sudden death (2).

Other causes of sudden cardiac death in adolescents include abnormalities in the structure of the heart, such as coronary disease and abnormal myocardial infarction. Other causes include myocardial infarction, which can be caused by microorganisms and other diseases. In addition to chronic QT syndrome, other abnormalities of the cardiovascular system, such as Brugada syndrome, can lead to sudden death. Commotio cordis, another rare cause of sudden heart attack that can happen to anyone, results in a severe stroke to the chest, like a hockey puck or other athletes. If the stroke occurs at the wrong time in the cardiovascular cycle, the stroke can cause inflammation of the cavity (figure 1, 2) (3).

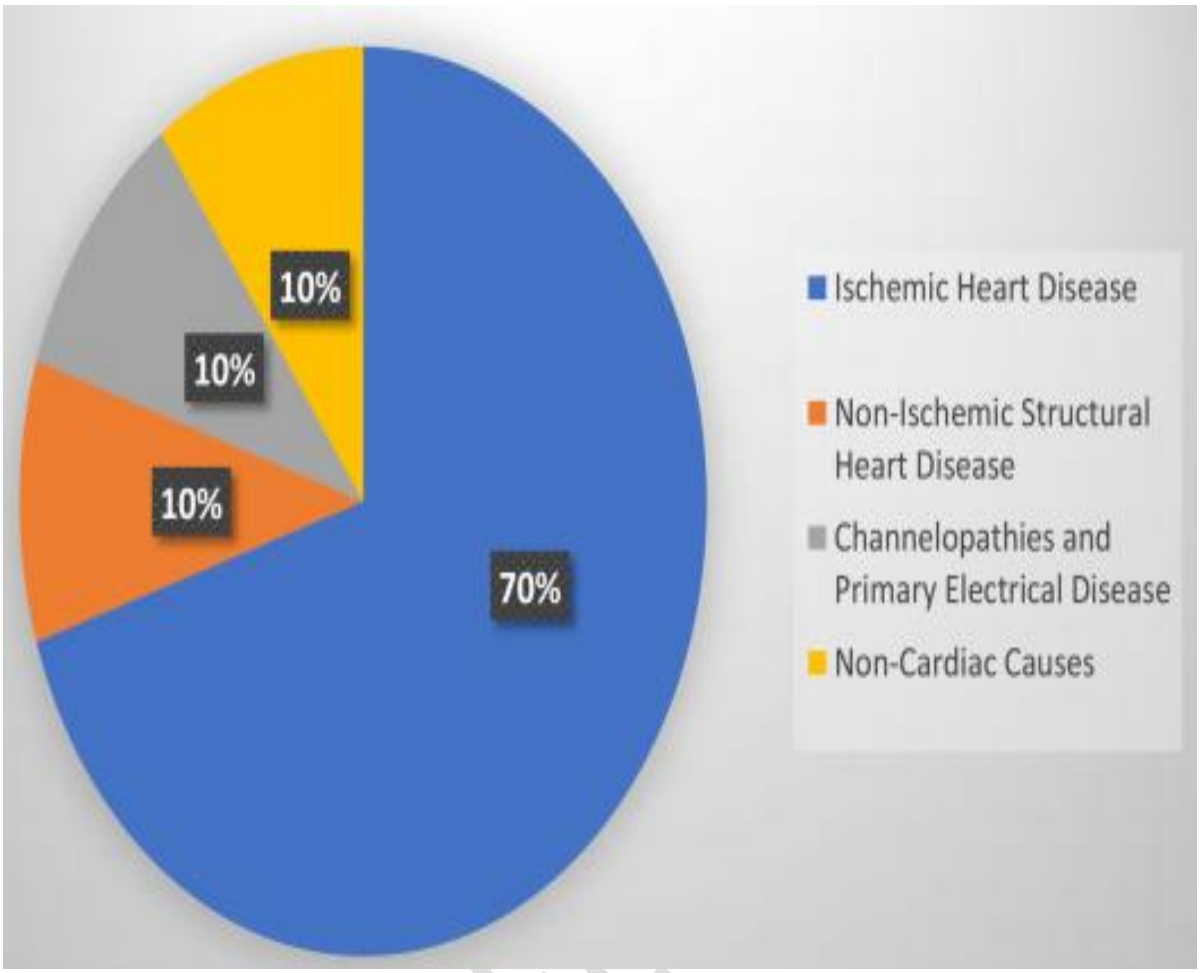

Figure 1 Causes of Sudden Cardiac Death (4).

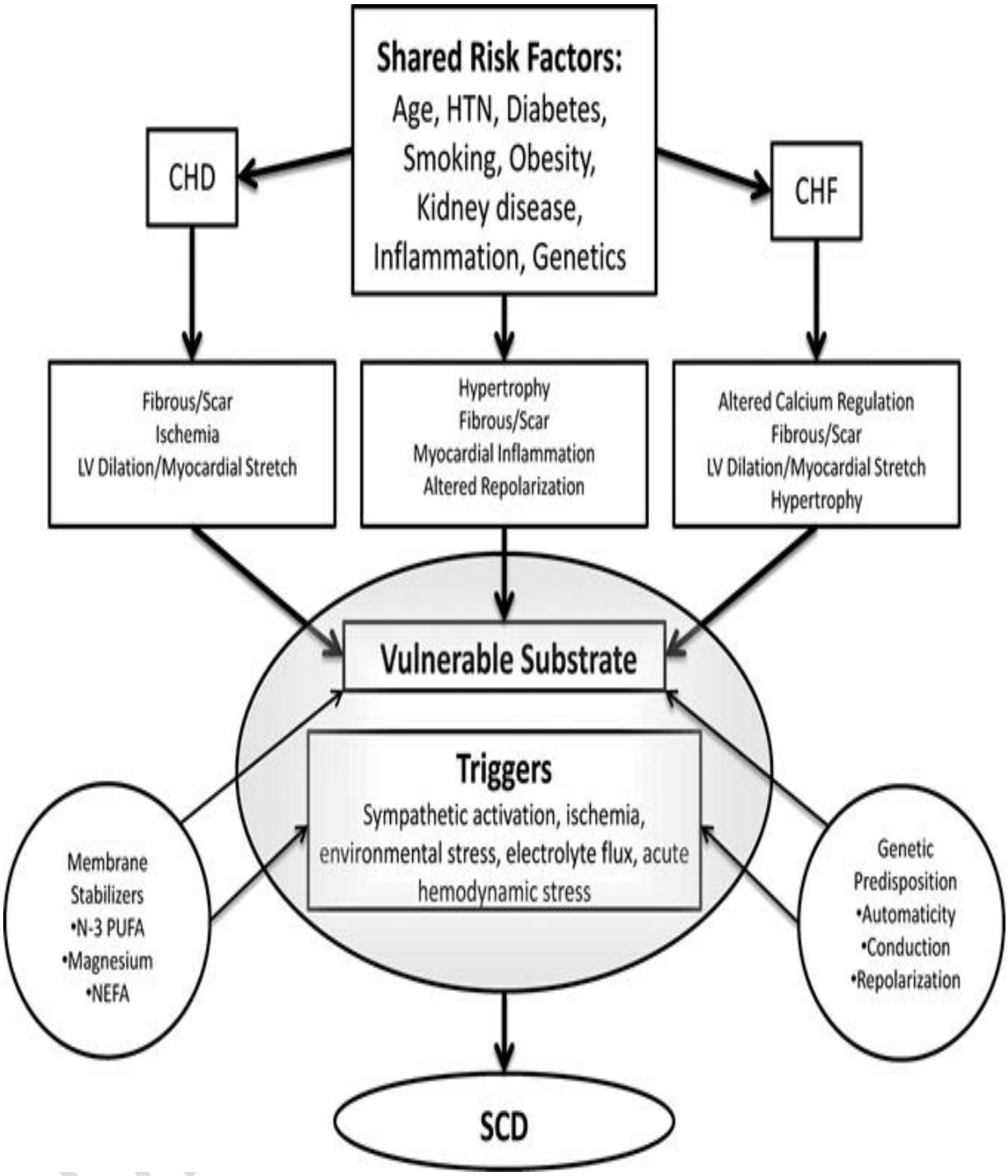

Figure 2 Risk Factors of Sudden Cardiac Death (4).

Signs and Symptoms of a Cardiac Arrest

A heart attack can show some signs and symptoms that can be considered "warning signs." If you or someone close to you experiences any of the following symptoms, call an emergency immediately. If you have a heart attack, you have: dizziness, shortness of breath, chest pain, heart rate, collapse or collapse, heart rate, shortness of breath, or complete loss. Shortness of breath, loss of consciousness, falls, feeling weak or tired, and the urge to nausea or vomit (Figure 3) (5).

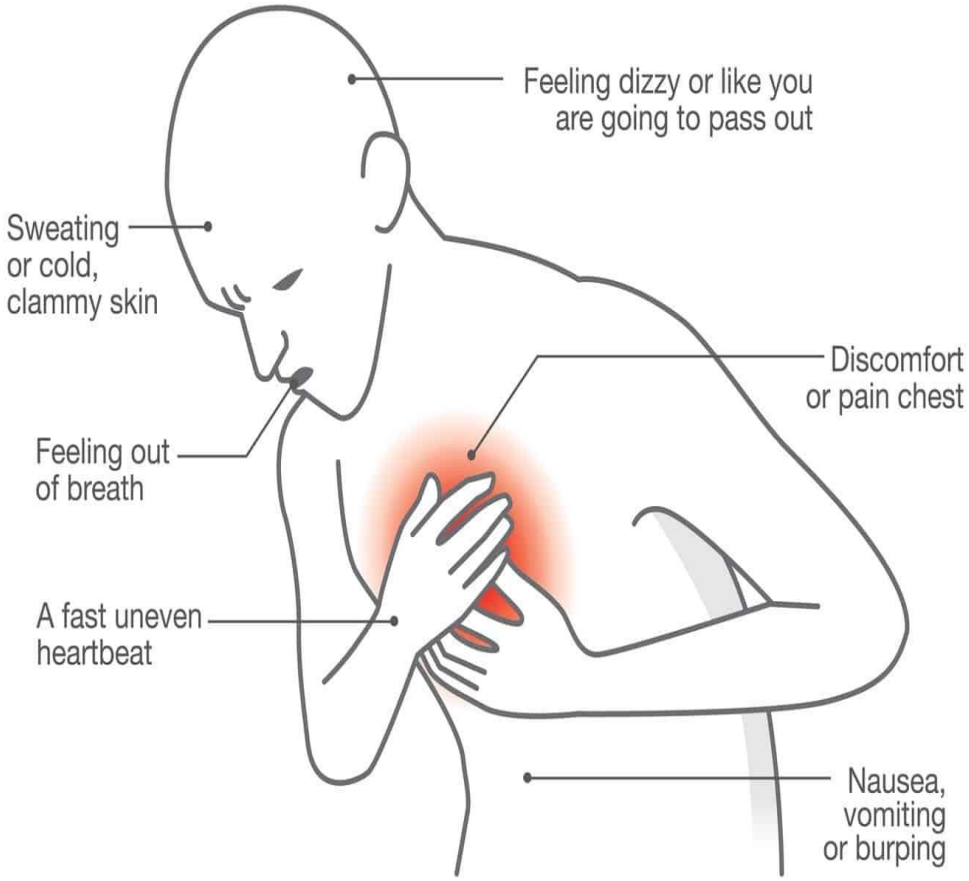

Figure 3 Signs and Symptoms of a Cardiac Arrest (5).

Mechanism of Sudden Cardiac Death

Calcium ions are important intracellular signaling atoms responsible for regulating many cellular processes in the cardiac muscle cell, including excitation–contraction, the gene register, enzyme activity, and cell death. The intracellular [Ca2+] concentration fluctuates significantly between systole and diastole, but the changes in cytosolic [Ca2+] are strictly controlled. Several signaling molecules, including calcium/calmodulin-dependent protein kinase II (CaMKII), PKA and PKC, are involved in the regulation of these Ca2+-bearing proteins. Ca2+-disturbed homeostasis and its management are associated with mechanical dysfunction in acute myocardial ischemia and arrhythmias and chronic cardiovascular diseases such as cardiac hypertrophy and heart failure (6).

During myocardial ischemia or metabolic inhibition, various metabolic parameters change significantly in cardiomyocytes. For example, ATP levels decrease, cells become more and more acidic, and high lactate levels and phosphate and magnesium levels increase. Decreased ATP levels and elevated phosphate levels can inhibit Na + / K + ATPase activity and cause intracellular accumulation of Na +. The slow increase in Na + current (late Ina) also contributes to the increase in intracellular [Na +] in myocardial ischemia and heart failure. An increase in intracellular [Na +] leads, at least in part, to an increase in [Ca2 +] cytosol. The actual Ca2 + or Ca2 + decrement enters the NCX function in reverse mode (Na + output and Ca2 + input). Both SR Ca2 + uptake activity (via SERCA) and its release (via RyR2) are

inhibited during myocardial ischemia. However, inhibition of RyR2 appears to exceed the decreased SERCA activity during ischemia, which is indicated by the low frequency of automatic Ca2 + release from RyR2 and the increased SR Ca2 + loading. When reactivated or reoxygenated, RyR2 is released into the blockade, producing an automatic wave of Ca2 + release. This contributes to the calcium / substitution interaction and may increase the risk of ventricular arrhythmias (Figures 4) (7).

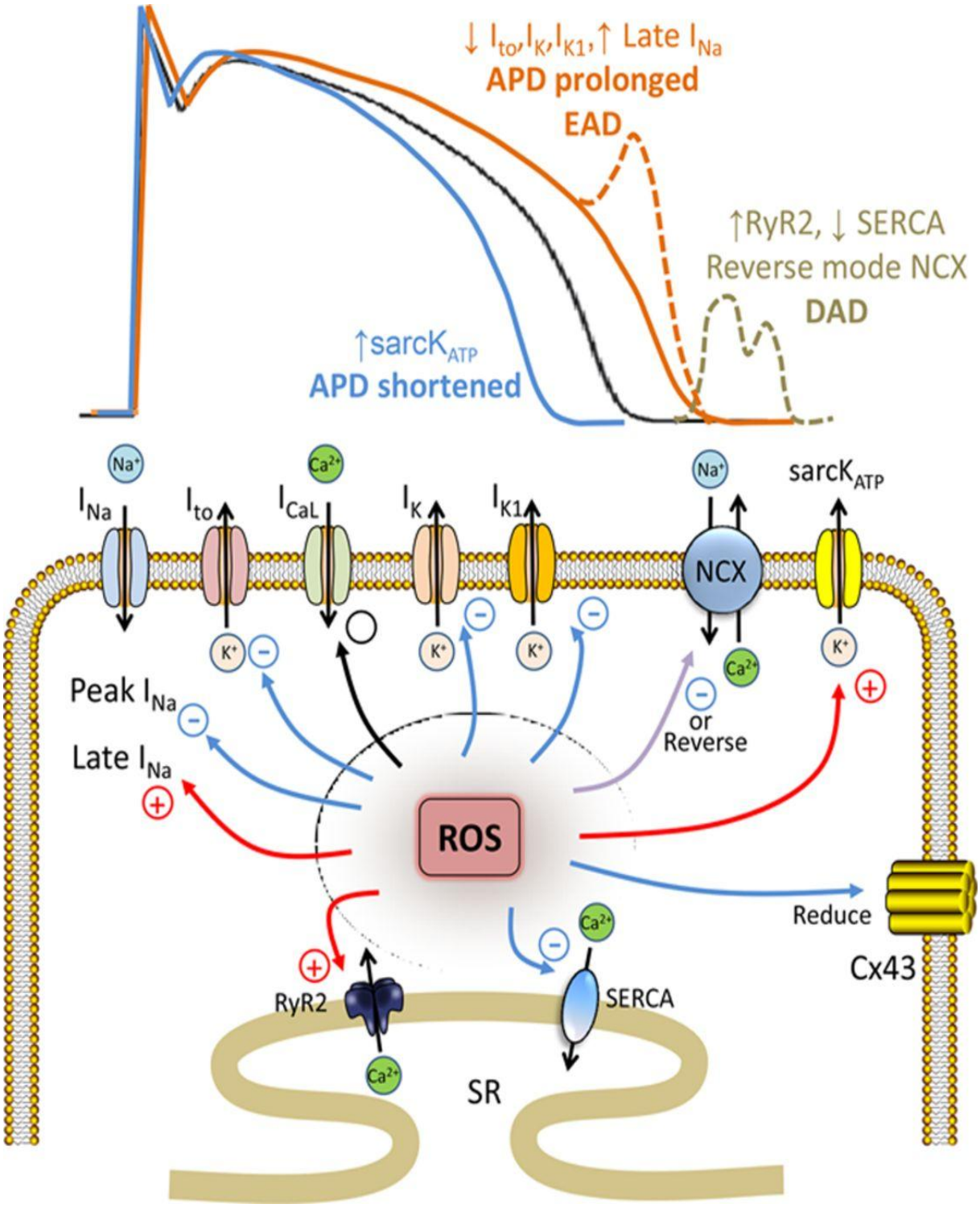

Figure 4 Mechanism of Sudden Cardiac Death (7).

Difference between Cardiac Arrest and Heart Attack

A cardiac arrest is the result of abnormal electrical activity in the heart, leading to an abnormal heartbeat (also called arrhythmia) or a sudden heartbeat. In this condition, the heart is unable to pump oxygen throughout the body, including the brain. If left untreated, it can lead to coma, decreased heart rate and even death. Cardiac arrest is usually without precaution. A heart attack (also called coronary artery disease) occurs when there is a blockage in the blood vessels that supply blood to the heart. If this obstruction is not removed immediately, it can damage the heart muscle or wall. The severity of the damage will depend on the size of the block. If not treated in time, the consequences can be serious and even death. Symptoms of a heart attack usually begin to appear in hours, days or weeks. Having a heart attack can lead to a heart attack (Figure 5) (8).

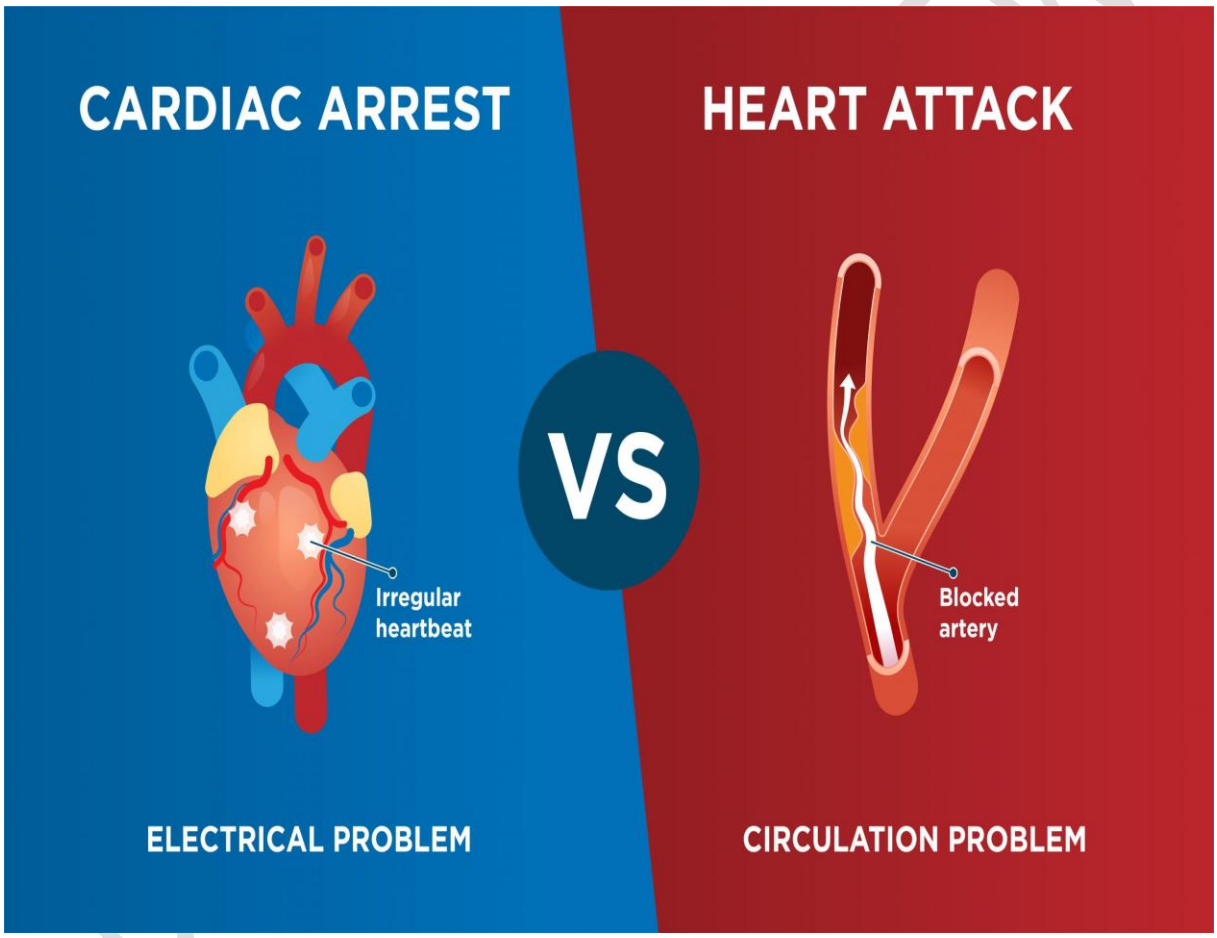

Figure 5 Difference between Cardiac Arrest and Heart Attack (8)

Results

An estimated 180,000 to 300,000 sudden cardiac deaths (SCD) occur annually in the United States. Sudden and unexpected cardiac death is the most common cause of death worldwide: about 17 million people die each year, and SCD accounts for about 25% of these cases. An acceptable definition of SCD is death that occurs within an hour after the onset of symptoms in tested cases, and within 24 hours, it finally appears alive in the dark. Most deaths go undiagnosed and VF is the last resort. Most patients are diagnosed with asystole or pulseless

electrical activity (PEA), and cardiac arrest is increasingly recognized as an etiology. Despite the decline in cardiovascular disease mortality over the past decades, 6 as a result of improved prevention strategies, the incidence of SCD as part of cardiovascular disease mortality has increased. This is because mortality in hospitals has fallen dramatically, highlighting the need for more effective prevention and risk management strategies. Treatment with class Ic agents or amiodarone has been shown to be ineffective in preventing SCD. An important advance in the prevention of SCD has been the development of an ICD. A second prophylactic trial, Antiarrhythmic versus Implantable Defibrillator (AVID), Canadian Implantable Defibrillator Study (CIDS), and Cardiac Arrest Study of Hamburg (CASH), showed significant improvements in survival rates with ICD implants versus drug therapy (9).

The Multicenter Automatic Defibrillator Implantation Trial (MADIT) and the Multicenter Ususstained Tachycardia Trial (MUSTT) study enrolled in patients referred for MI, compared primary prevention with ICD versus standard medical treatment for patients with reduced ejection fraction (EF) % and <40%, respectively), and documented or constructed ventricular tachycardia (VT) and showed a 58-59% decrease in mortality. Later MADIT II showed a 28% reduction in 2-year mortality-related risk in post-MI patients with EF of <30% without the requirement of a written or advertised VT. Defibrillators in the Non-Ischemic Cardiomyopathy Treatment Evaluation study compared the benefit of ICD against conventional treatment in patients with heart failure, EF ≤35% and premature ventricular congestion or chronic ventricular tachycardia (NSVT), showing a strong tendency to slow down death by ICD (10).

The Sudden Cardiac Death Scale (SCD-HEFT), which listed patients with ischemic and non-ischemic cardiomyopathy with New York Cardiac Association Class II or III and EF 35% or less, listed the benefits of ICD over ICD Show up with standard treatment. Interestingly, these major preventive trials lack other major risk predictions that indicate who will benefit from ICD, except for low EF. Larger studies use EF cut-offs below 30-40%. However, EF is very low in the middle population between these studies, and analysis of patient groups near the cut-off often does not show a clear benefit. In addition, in "high-risk" human studies such as MADIT II and SCD-HeFT, 68 years were not associated with a reduction in long-term mortality (11).

Using these studies as guidelines for CDI means targeting a small subset of patients only when the incidence is high and therefore the risk is high. The problem for clinicians is that many episodes of SCD occur in pre-event people who do not have known heart disease and are not considered a major risk factor by conventional criteria, or undiagnosed heart disease. It occurs as an early onset of. Therefore, most cases of sudden cardiac death occur in patients who are considered to have a low risk of developing the event. Although the incidence of patients in this group is low, they explain the increase in the number of cases. Finally, there are indications for primary prevention in rare cases such as hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), long QT syndrome (LQT), Brugada syndrome, and early repolarization. It remains unknown. Dangerous signs that go beyond the patient's symptoms (Figure 6) (12).

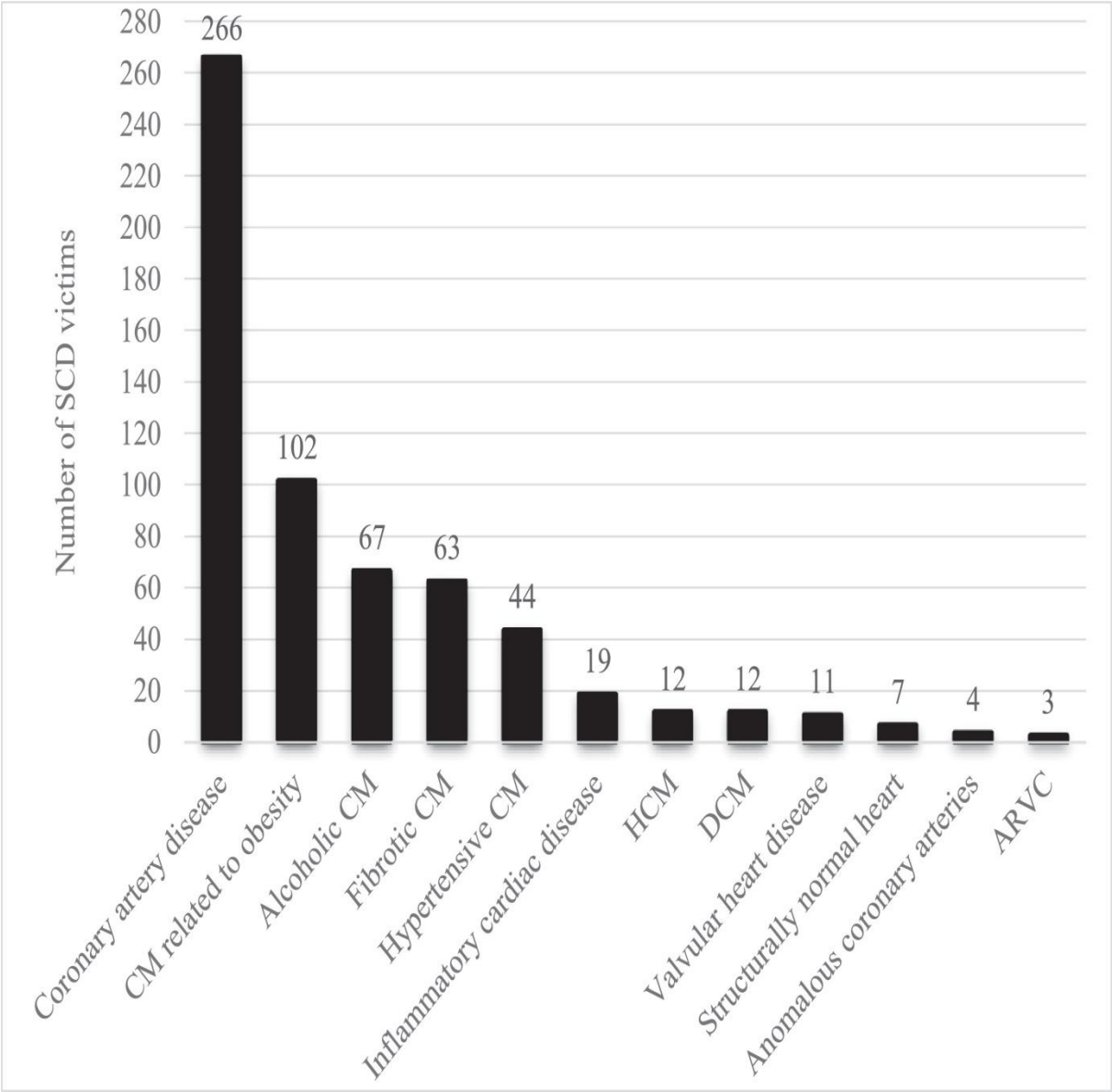

Figure 6 Number of Sudden Cardiac Death Victims (13).

Medication and Prevention of SCD from Arrhythmia

Chronic beta blockade improves survival in patients after myocardial infarction and reduces the incidence of sickle cell disease, especially in patients with left ventricular dysfunction. The use of other compounds such as encainide, flecainide, and D-sotalol that block certain cardiovascular ion channels, namely "classical" antiarrhythmic agents, is associated with side effects, side effects in patients with endless myocardial infarction. Is shown. The antiarrhythmic drug amiodarone blocks and blocks various cardiac ion channels and vehicles such as sodium channels, L channels, several potassium channels, and sodium-calcium exchangers at clinically appropriate concentrations. α- and β-adrenergic receptors also do not improve survival. Therefore, the prophylactic use of certain ion blockers does not reduce or increase mortality. In contrast, ICDs provide high-risk people with survival benefits over drugs. Treatment with ICDs is now the standard treatment for patients who have survived life-threatening arrhythmias (14).

Patients with heart failure often have regions of delayed myocardial function and shortness of breath, leading to cardiac dyssynchrony. Cardiac remodeling, including right atrial placement, right ventricular right, and left-ventricular lead, restores atrio-biventricular synchrony leading to improved left ventricular function and (without concomitant ICD treatment) can improve survival in patients. hearty. failure (partial ejection <35%) and cardiac dyssynchrony (figure 7) (15).

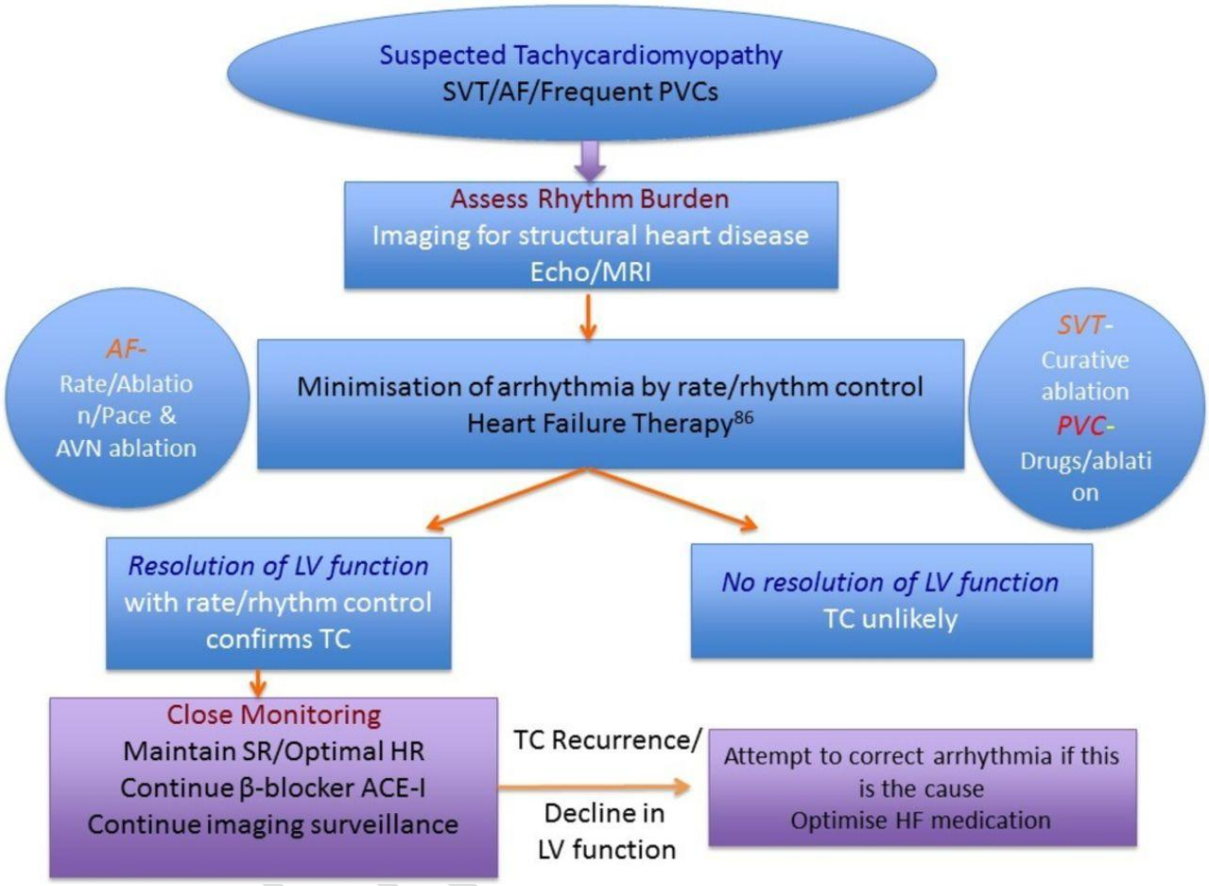

Figure 7 Prevention of SCD from Arrhythmia (15).

Discussion

Sudden cardiac death refers to an unexpected natural death caused by a heart attack over a short period of time, usually ≤ 1 hour after the onset of symptoms, in someone other than a previous condition that may appear fatal. Such a rapid death is often referred to as cardiac arrhythmia, but with the advent of the monitoring capabilities of body-implanted cardioverter-defibrillators (ICDs), it is now well known that clinical isolation can be misleading and often impossible. , since 40% of sudden death can be undetectable. Only an ECG or ventricular electrogram recorded on a device installed at the time of death can provide accurate information about the arrhythmia. Prodromal symptoms are often vague, and even those taken to indicate ischemia (chest pain), tachyarrhythmia (palpitations), or symptoms of congestive heart failure (dyspnea) can only be taken as suggestions. For these reasons, total mortality, rather than the classification of cardiac and arrhythmic deaths, should be used as the main objective of many outcome studies (16).

Conclusion

SCD most often occurs in the presence of relatives at home and long after the onset of typical discomfort symptoms. Although the widely welcomed use of defibrillation in the public domain has been supported by many studies, these results raise the question of whether educational interventions and targeted education programs designed for at-risk patients and their families should be given high priority. Sudden cardiac death (SCD) is one of the most common causes of death in industrialized countries. Annual events per 100,000 inhabitants & estimated 100 cases. The emergence appears to be multifactorial and has only been partially studied. Recent studies have discussed chronobiological factors as potential triggering mechanisms. Daily, weekly and seasonal fluctuations have been recorded in the SCD edition. Certain risk factors, such as nicotine, caffeine or the use of certain drugs, and other triggers such as psychological or physical stress and arousal have been identified, although the underlying role of coronary heart disease and other causes of SCD, such as long-term QT syndrome. Each event seems to represent a sudden and unexpected situation.

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