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Research Article

Sudden Cardiac Death: Some Considerations On the Matter

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Abstract

Heart disease remains the leading cause of death worldwide. The Sudden cardiac death (or better the Unexpected Cardiac Death) can appear at any time and anywhere. Despite being the most wrenching event that any physician can find, (when it occurs in childhood the tragedy is supreme), the called "sudden cardiac death" is still quite unknown in its most intimate mechanisms of production. Delays in diagnosis, a big lack of previous diagnosis in many cases, as well as the immense discrepancies and controversies among the various authors, are the keys for such lack of knowledge.

Lately, the implementation of semiautomatic defibrillators on the streets, supermarkets and the education of non-specialists people, is a quite effective method for the recovery and survival of people who have suffered such a wrenching event. Problem? Not work if do not capture heartbeat.

Some deep and well documented studies are making possible the creation of some "risk score" for suffer an "unexpected cardiac death." This can be very beneficial to its prevention, through an advice and pertinent medical interventions. (Thompson and McCullough score. Rajat and Faye score: Appurtenances 1 and 2).

Unfortunately – at this time-, only around the 15% of people who have suffered this type of event is recovered. And of this 15% of people recovered, more than 85% will suffer some sort of neurological disorder for all life. Most sudden deaths occur outside the hospital, on the street. This considerably reduces the total recovery process. Also unfortunately, the discrepancies about the concept and management of such infamous situation are still very bountiful among medical professionals. The discrepancies about how it must be defined, what actions are necessary immediately to its apparition, and many other issues more, remain overwhelming. Here we will discuss some nuances about them.

By way of example, the meaning of "sudden" does not mean "unexpected" necessarily.

For **Framingham Heart Study**, sudden cardiac death is defined as: "death because of CHD occurring within 1 hour of symptom onset and not likely attributable to other causes".

We put 'called into question' this definition. We could say: "All the unexpected is sudden. Not all the sudden is unexpected".

We think this event should be called as: **Unexpected cardiac death.**

As for the overall management of such an event, there are also many differences and controversies among the different authors. This subject also will be discussed in the document.

Keywords: *Sudden Death; Sudden cardiac death; Cardiac arrhythmias; Cardiac structures*

Introduction

Death can be defined as an event resulting from the inability to sustain organic homeostasis. From the medical viewpoint is the global cessation of systemic functions; especially of brain bioelectric functions and thus neuronal. When all this happens in an unexpected and abrupt manner, we speak of Sudden cardiac death [1,2].

Sudden cardiac death (SCD) is being defined as an unexpected death due to cardiac causes occurring in a short time period, generally within 1 h of symptom onset, in a person with known or unknown cardiac disease. For many authors, most cases of sudden cardiac death are related to cardiac arrhythmias. For them, approximately half of all cardiac deaths can be classified as sudden cardiac deaths [1,2].

Sudden cardiac death represents the first expression of cardiac disease in many individuals who experience out-of-hospital, a cardiac arrest. Many patients at risk for sudden cardiac death may have prodromes of chest pain, fatigue, palpitations, and other nonspecific complaints, only (these circumstances are the most frequent). Many times, they are totally asymptomatic.

Factors relating to the development of Coronary Artery Disease (CAD) and, subsequently, Myocardial Infarction (MI) and Ischemic Cardiomyopathy include the following according most authors:

Family history of premature coronary artery disease
Smoking
Dyslipidemia
Hypertension
Diabetes
Obesity
Sedentary lifestyle

Alcohol and design drugs are not contemplated.

From higher to lower frequency for the sudden cardiac death between clinical cases which have been able to be diagnosed before occur the events, are:

1. Structural cardiac diseases.
2. Non- structural cardiac diseases.

Structural Cardiac Diseases

a. Coronary artery disease.

Previous cardiac arrest
Syncope
Prior myocardial infarction, especially within 6 months
Ejection fraction of less than 30%
History of frequent ventricular ectopy: More than 10 premature ventricular contractions (PVCs) per hour or nonsustained ventricular tachycardia (VT)

b. Dilated cardiomyopathy

Previous cardiac arrest
Syncope
Ejection fraction of less than 30%
Use of inotropic medications

Dilated cardiomyopathy (DCM), formerly called congestive cardiomyopathy, is a disorder characterized by a progressive hypertrophy and dilation of the heart, causing weakness to the point of reducing the ability to pump blood effectively.

c. Hypertrophic cardiomyopathy

Previous cardiac arrest
Syncope
Family history of sudden cardiac death
Symptoms of heart failure
Drop in systolic blood pressure (SBP) or ventricular ectopy upon stress testing
Palpitations
Most persons are asymptomatic

Hypertrophic cardiomyopathy is a disease in which a portion of the myocardium is hypertrophied or thickened with no apparent cause. Despite its reputation as a leading cause of sudden cardiac death in young athletes, the process is particularly important for causing sudden cardiac death in any age group and/ or cause debilitating cardiac symptoms.

d. Valvular disease

Valve replacement within past 6 months
Syncope
History of frequent ventricular ectopy
Symptoms associated with severe, uncorrected aortic stenosis or mitral stenosis

Length alterations on electrical cardiac intervals

Short and long QT-interval.
Short PR-interval.
Others.

The vast majority of these abnormal cardiac rhythms is considered as "**non-structural diseases**".

For many authors, as many as 45% of people who have sudden cardiac death were seen by a physician within one month before death, although as many as 78 % of these complaints were not related to the cardiovascular system according them [1]:

A previous history of Left ventricular injury (ejection fraction lesser of 30%) is the strongest risk factor for sudden death. That is, many cardiac sudden deaths have not had a previous diagnosis with which to have been avoided.

We think it is worthy to be taken into account, the fact that in the vast majority of "tables" about risk factors for sudden cardiac death, do not appear neither **alcohol** nor drugs (doping). For example, dilated cardiomyopathy is closely linked to alcohol consumption.

However, these substances provide much risk as the above mentioned.

Pathophysiology

The most common electrophysiologic mechanisms leading to sudden cardiac death are tachyarrhythmias such as ventricular fibrillation (**VF**) or ventricular tachycardia (**VT**).

Both an Automatic external defibrillator (**AED**) as an Implantable cardioverter defibrillator (**ICD**) are a very useful tool for interrupting of tachyarrhythmias: using either and has been shown to be an effective treatment for Ventricular fibrillation and ventricular tachycardia [3-6]. This device type can be automatic and semiautomatic. Semi-automatic device is the most used by non-medical people when they witness a cardiac arrest. The biggest problem is that this type of device does not "shoot" if it does not capture any heartbeat.

The implantable defibrillator has become the leading therapeutic in the prevention and treatment of sudden cardiac death. Patients with tachyarrhythmias, especially ventricular tachycardia, carry the best overall prognosis among patients with Sudden Cardiac Arrest (SCA) [4-6].

There are multiple factors at the organ, tissue, cellular and subcellular level involved in the generation of ventricular tachycardia or ventricular fibrillation by different conditions [7,8]. An anatomical or a functional block in the course of impulse propagation may create a circuit with the wave front circling around it and resulting in ventricular tachycardia. Other mechanisms such as wave break and collisions are involved in generating a ventricular fibrillation from a ventricular tachycardia. While at the tissue level, the reentry and wave break mechanisms are the most important known for ventricular tachycardia and ventricular fibrillation, at the cellular level increased excitation or decreased repolarization reserve of cardiomyocytes may result in ectopic activity, contributing to ventricular tachycardia and ventricular fibrillation initiation.

At the subcellular level, altered intracellular Ca^{2+} currents, altered intracellular K^{+} currents (especially in ischemia), or mutations resulting in dysfunction of a sodium channel (Na^{+} channelopathy) can increase the likelihood of ventricular tachycardia and ventricular fibrillation [7]. (See image below)

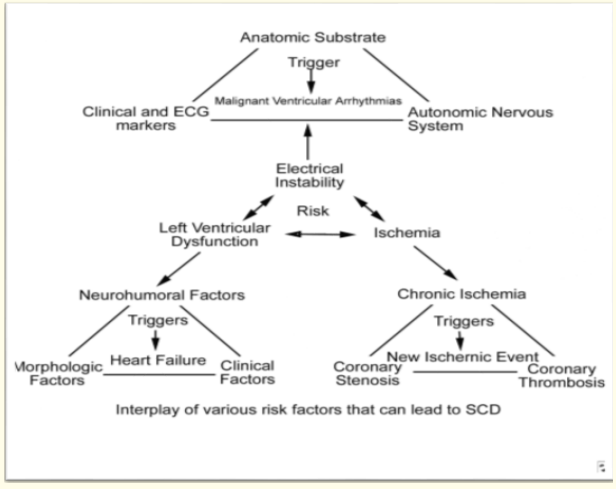

Figure 1: *The image was taken from Ali A Sovari, MD, with his permission.*

Approximately 30% of patients with documented sudden death events have sudden bradyarrhythmia or asystolia at the time of initial contact [7,8].

Oftentimes, it is difficult to determine with certainty the initiating event in a patient presenting with a bradyarrhythmia because asystole and Pulseless Electrical Activity (**PEA**) may result from a sustained ventricular tachycardia. Less frequently, an initial bradyarrhythmia producing myocardial ischemia may then provoke ventricular tachycardia or ventricular fibrillation.

Most cases of sudden cardiac death occur in patients with structural abnormalities of the heart. Myocardial infarction (MI) and post-MI remodeling of the heart is the most common structural abnormality in patients with sudden cardiac death [8].

In patients who survive a myocardial infarction, the presence of Premature Ventricular Contractions (**PVCs**), particularly complex forms such as multiform PVCs, short coupling intervals (**R-on-T phenomenon**), or ventricular tachycardia (salvos of 3 or more ectopic beats), reflect an increased risk of sudden death. However, suppression of the PVCs with antiarrhythmic drugs can increase the mortality, owing to the proarrhythmic risk of currently available medications. "All antiarrhythmic drug, can be pro-arrhytmogenic" Hypertrophic cardiomyopathy and Dilated cardiomyopathy are also associated with an increased risk of sudden cardiac death.

Various Valvular Diseases such as aortic stenosis are also associated with increased risk of sudden cardiac death. Acute illnesses, such as Myocarditis, may provide both an initial and sustained risk of sudden cardiac death due to inflammation and fibrosis of the myocardium.

Also, less usually, sudden cardiac death happens in patients who may not have an apparent structural heart disease. These conditions are usually arrhythmia syndromes. These arrhythmic diseases can be inherited or acquired: Many drugs are liable to cause alterations in different of the ECG intervals, and therefore increase the risk of sudden cardiac death. Day after day are discovering more drugs with these features.

Even though many patients have anatomic and functional cardiac substrates that predispose them to develop ventricular arrhythmias, only a small percentage develop sudden cardiac death (lesser of 40%) [10,11]. Identifying the patients at risk for sudden cardiac death remains a challenge. The strongest known predictor of sudden cardiac death is significant left ventricular dysfunction of any cause. The interplay between the regional ischemia, Left ventricular dysfunction, and transient events (ischemia, acidosis, hypoxemia, wall tension, drugs, metabolic disturbances) have been proposed as being triggers of sudden death.

Epidemiology [9-13]

Epidemiology of sudden cardiac death may be the real "workhorse" as well as the leader of all discrepancies between different authors. Make a "statistic" by sudden cardiac death and concoct a number of incidences for each disease is very reckless. And very speculative, of course. Since in many cases, patients have not had a prior diagnosis and, therefore, any preventive measures haven't been practiced.

Knowing exactly the incidence and prevalence of this type of event is a very hard task. Especially if, as some authors argue, the causal trigger has been an abnormal heart rhythm since many cardiac arrhythmias were not diagnosed in due time and went unnoticed: they are many more the undiagnosed cases that with a certain diagnosis. Since once the person is in cardiac arrest and this one was not recovered this is diagnosed as a "non-structural cardiac death"; so it remains a diagnosis discard, since at autopsy, when it is done – Never appear any signs of structural damage ever. Namely, a 'diagnosis by eliminating'.

Frequency

Possibly, the people more likely to suffer sudden cardiac death are addicted persons to drugs (alcohol, cocaine, heroin, drug design) without any structural cardiac disease. With the added problem that, despite having been recovered from a previous cardiac arrest, they can suffer it again due to addiction and to continue with ingesting such drugs. In this disquisition point, the dead athletes suddenly form an important subgroup because drug intake to improve sports scores is very large in this subgroup of people. However, this matter has been very little studied. The average age of these patients is less than 30 years-old.

In several population-based studies, the incidence of out-of-hospital cardiac arrest has been noted as declining in the past 2 decades. A high incidence of sudden cardiac death occurs among certain subgroups of high-risk patients (congestive heart failure with ejection fraction < 30%, convalescent phase after myocardial infarction, patients who survived cardiac arrest). However, these populations are much smaller than pain the overall population, most sudden cardiac death occurs in lower risk patients. The time dependence of risk for sudden cardiac death has been noted in several studies, with an increased number of events in the first 6-24 months after surviving a major cardiovascular event.

Mortality/Morbidity [12,13]

With more than 300,000 deaths attributed to sudden cardiac death in the United States each year, a large portion (as many as 40%) is unwitnessed. For most people who experience sudden cardiac death, their survival depends on the presence of individuals who are competent in performing basic life support, the rapid arrival of personnel and apparatus for defibrillation and advanced life support, and transfer to a hospital. Even under ideal circumstances, only an estimated 20% of patients who have out-of-hospital cardiac arrest survive to hospital discharge. In a study of out-of-hospital cardiac arrest survival in New York City, only 1.4% of patients survived to hospital discharge [8,9]. Other studies in suburban and rural areas have indicated higher rates of survival (as high as 35%). Placement of automatic external defibrillators throughout communities and training people to use them has the potential to markedly improve outcomes from sudden cardiac death. Upon emergency department (**ED**) presentation, the most important determinants of survival include an unsupported systolic blood pressure (**SBP**) greater than 90 mm Hg, a time of loss of consciousness to return of spontaneous circulation (**ROSC**) of less than 25 minutes, and some degree of neurological responsiveness. A major adverse outcome of a sudden cardiac death event is anoxic encephalopathy, which occurs in 30-80% of cases.

Race

Most studies demonstrate inconclusive data with regard to racial differences as they relate to the incidence of sudden death [12]. Some studies suggest that a greater proportion of coronary deaths were "sudden" in blacks compared to whites. In a report by Gillum., *et al*. [13] on sudden cardiac death from 1980-1985, the percentage of coronary artery disease deaths occurring out of the hospital and in EDs were found to be higher in blacks than in whites.

Sex

Men have a higher incidence of sudden cardiac death than women, with a ratio of 3:1. This ratio generally reflects the higher incidence of obstructive coronary artery disease in men. Recent evidence suggests that a major sex difference may exist in the mechanism of myocardial infarction. Basic and observational data point to the fact that men tend to have a coronary plaque rupture, while women tend to have plaque erosion. Whether this biological difference accounts for the male predominance of sudden cardiac death is unclear. Over the 32-year period, 89% of sudden deaths from all causes occurred in men. The mortality rate in men was eightfold higher than for women. The number of all-cause sudden deaths among white athletes was 62% and among black athletes and other minorities was 38%. However, the incidence of mortality was 3.2-fold higher among black athletes other minorities compared with white athletes (1 per 6,314 athlete-years vs. 1 per 20,096 athlete years;. Additionally, the rate of CV death was fivefold higher among black athletes and other minorities compared with white athletes (1 per 12,778 athlete-years vs. 1 per 60,746 athlete-years).

Age

The incidence of sudden cardiac death parallels the incidence of coronary artery disease, with the peak of sudden cardiac death occurring in people aged 45-75 years. The incidence of sudden cardiac death increases with age in men, women, whites, and nonwhites as the prevalence of coronary artery disease increases with age. However, the proportion of deaths that are sudden from coronary artery disease decreases with age. In the Framingham study, the proportion of coronary artery disease deaths that were sudden was 62% in men aged 45-54 years, but this percentage fell to 58% in men aged 55-64 years and to 42% in men aged 65-74 years. According to Kuller et al, 31% of deaths are sudden in people aged 20-29 years.

Physical Examination

Early diagnosis of these deadly diseases is paramount. However, on many occasions, this early diagnosis is not done.

A good physical examination may reveal evidence of underlying myocardial disease or may be entirely normal, depending on the underlying cause. Initial evaluation studies show that patients who survive to the Emergency Department present can be stratified by a cardiac arrest score, which has excellent diagnostic value [13-15].

A cardiac arrest score, developed by Thompson and McCullough (**Appendix 1**), can be used for patients with witnessed out-of-hospital cardiac arrest and is defined by the following criteria:

Clinical characteristic points:

ED SBP greater than 90 mm Hg = 1 point
ED SBP less than 90 mm Hg = 0 points
Time to ROSC less than 25 minutes = 1 point
Time to ROSC more than 25 minutes = 0 points
Neurologically responsive = 1 point
Comatose = 0 points

Maximum scores = 3 points (Thompson and McCullough score)

Patients with a score of 3 points can be expected to have an 89% chance of neurologic recovery and an 82% chance of survival to discharge (see the image below). Neurologic recovery is defined as discharged home and able to care for self [13]. McCullough indicates that even in the setting of ST elevation and early invasive management with primary angioplasty and intraaortic balloon pump insertion, patients with low cardiac scores are unlikely to survive. Severe anoxic encephalopathy in patients with scores of 0, 1, or 2 mitigates conservative management with empiric supportive and medical therapy. Given the very poor actuarial survival rates for these patients, invasive management with catheterization and Electrophysiology studies (**EPS**) is rarely justified.

Rajat Deo, Faye L. Norby, Ronit Katz., *et al.* have developed another 'risk score' very well structured; Prof. Douglas P et al. consider like the best risk score for SCD at the moment (**Appendix 2**). This author says "There were 345 adjudicated SCD events in the analysis that found 12 independent risk factors for SCD that included age, male sex, African American race, current smoking, systolic blood pressure, use of antihypertensive medication, diabetes, serum potassium, serum albumin, HDL, estimated GFR, and QTc interval. Strikingly, a reduced **EF** was found in only 1% of participants in an echocardiographic substudy and did not enhance risk prediction. This risk model outperformed the **2013 ACC/AHA CVD Pooled Cohort Risk Equations**. These findings provide the first generalizable risk score to help establish **SCD** risk in the general population. In the future, this risk score can be used to help prevent **SCD** in the high-risk subgroups of the general population, who had a 5% risk of **SCD** over 10 years. Clinicians need to follow future information from this study, which will translate into reducing **SCD** for their patients". Extraordinary reflection from Prof. Douglas P. Zipes.

Diagnostic Considerations

Other conditions to be considered in patients with suspected sudden cardiac death include Arrhythmogenic Right Ventricular Dysplasia, Brugada syndrome, Breijo pattern and others cardiac electrical abnormalities. Important considerations include the following:

a. Recognize and initiate early management of patients with ischemic heart disease. The importance of this cannot be overestimated, because approximately 80% of sudden cardiac death cases can be attributed to ischemic heart disease.

b. Use appropriate medical therapy for cardiac arrest. Some study suggested that vasopressin is more effective in acute therapy for asystole than epinephrine [14].

It is very important to involve a specialist in cardiovascular disease in the care of patients who have had a cardiac arrest or have symptoms of ischemic heart disease, valvular disorders, or presentations with complex arrhythmias. It is also very important to educate patients about the consequences of noncompliance with medical therapy.

Differential Diagnoses

All cardiac dysfunction can produce a SCD. The most common and known alterations are as follows:

Angina Pectoris. Aortic Stenosis. Coronary Artery Atherosclerosis. Dilated Cardiomyopathy. Ebstein Anomaly. Hypertrophic Cardiomyopathy. Lown-Ganong-Levine Syndrome. Manhaim syndrome. Breijo Pattern. Myocardial Infarction. Tetralogy of Fallot. Torsade de Pointes. Ventricular Fibrillation. Ventricular Premature Complexes. Ventricular Tachycardia. Wolff-Parkinson-White Syndrome.

Workup

Laboratory Studies

Laboratory studies in the workup of sudden cardiac death include the following:

1.Cardiac enzymes (creatine kinase, myoglobin, troponin): Elevations in these enzyme levels may indicate ischemia and MI. The extent of myocardial damage usually can be correlated to the extent of elevation in the enzyme levels. Patients are at increased risk for arrhythmia in the peri-infarct period.

Electrolytes, calcium, and magnesium: Severe metabolic acidosis, hypokalemia, hyperkalemia, hypocalcemia, and hypomagnesemia are some of the conditions that can increase the risk of arrhythmia and sudden death.

Quantitative drug levels (quinidine, procainamide, tricyclic antidepressants, digoxin): Drug levels higher than the levels indicated in the therapeutic index may have a proarrhythmic effect. Subtherapeutic levels of these drugs in patients being treated for specific cardiac conditions also can lead to an increased risk for arrhythmia. All antiarrhythmic medications also have a proarrhythmic effect.

Toxicology screen: Looking for drugs, such as cocaine or alcohol, that can lead to vasospasm-induced ischemia is warranted if suspicion exists. Obtaining levels of drugs (antiarrhythmics) also may be warranted.

As already previously, we have written, more than, possibly, the people more likely to suffer sudden cardiac death are addicted persons to drugs (alcohol, cocaine, heroin, drug design), with the added problem that, despite having been recovered from a previous cardiac arrest, they can suffer it again due to addiction and to continue with ingesting such drugs. The average age of these patients is less than 30 years.

Thyroid-stimulating hormone: Hyperthyroidism can lead to tachycardia and tachyarrhythmias. Over a period of time, it also can lead to heart failure. Hypothyroidism can lead to QT prolongation by example. As well as, hyperthyroidism can lead to a short QT interval.

Brain Natriuretic peptide (BNP): BNP has predictive value, especially in post MI patients and in patients with heart failure. Although preliminary and not conclusive, emerging data support the notion that an elevated BNP level may provide prognostic information on the risk of sudden cardiac death, independent of clinical information and LVEF [15].

Another Diagnostic Procedures

Chest Radiograph: This may reveal whether someone is in congestive heart failure. It also can show signs suggesting Left Ventricular enlargement or Right Ventricular enlargement. Signs of pulmonary hypertension also may be evident on the chest radiograph.

Echocardiography: Two-dimensional echocardiography with Doppler is essential in the evaluation of sudden cardiac death. A number of studies have demonstrated that the use of 2-dimensional echocardiogram to evaluate left wall motion abnormalities after an acute MI (using the LV wall-motion score index) is useful in predicting the risk for major cardiac events, including sudden death. A decrease in the ejection fraction and worsening wall motion abnormalities upon exercise echocardiography in patients who have had an MI has been suggested to confer increased risk of cardiac death.

Nuclear Imaging: Resting thallium or technetium-99m scintigraphy is helpful in assessing myocardial damage after MI. A larger defect has been associated with greater risk for future cardiac events. Exercise nuclear scintigraphy is very sensitive for detecting the presence, extent, and location of myocardial ischemia. Gibson., *et al.* found that pharmacological-stress nuclear (Dipyridamole or adenosine) scintigraphy was better than submaximal exercise ECG and coronary angiography in predicting cardiac death and other cardiac events. These

tests can be very helpful in patients with low functional capacity, such as chronic obstructive pulmonary disease, peripheral vascular disease, or orthopedic problems. The Multi-Center Post-Infarction Research Group provided evidence that resting ejection fraction was the most important noninvasive predictor of sudden cardiac death and other cardiac events in patients with MI.

This type of diagnostic testing has a very important problem: It is very expensive. Therefore, it is very rarely used.

Other Tests

Electrocardiogram: This study is indicated in all patients.

Evidence of MI, prolonged QT interval, short QT interval, epsilon wave, Brugada sign, short PR, a WPW syndrome, Breijo pattern or other conditions should be sought. Signal-averaged ECG **(SAECG**) has been variably reported to be useful in the analysis of patients with sudden cardiac death. What may be more useful is analysis of T-wave alternans in patients with ventricular tachycardia, ventricular fibrillation, and/or sudden cardiac death. Small changes in T-amplitude are not detected in 12-lead ECG. Microvolt T wave alternans (**MTWA**) amplifies the alternans and may be used in the workup to predict the risk of sudden cardiac death. **MTWA** appears to have high negative predictive value for the risk of sudden cardiac death in patient with low LVEF (< 30%). [24] In addition, a pooled- analysis of 5 prospective studies showed that SCA happens in approximately 3% of patients with an abnormal **MWTA** test and **LVEF** >30%, suggesting that MTWA may be considered as a tool to identify patients at risk for **SCA**. However, further studies are required to determine how MTWA alone or perhaps in combination with other electrophysiologic tools can be used to risk stratify the patients at risk for SCA.

Genetic testing: The value of genetic testing in conditions such as congenital long (or short) QT, Brugada syndrome, Breijo pattern, Hypertrophic Cardiomyopathy and many others electrical cardiac abnormalities is still being evaluated. Some studies have recommended the testing of siblings and close relatives of people with sudden cardiac death due to these conditions.

Procedures

Coronary angiography: Perform cardiac catheterization in patients who survive sudden cardiac death to assess the state of ventricular function and the severity and extent of **CAD**. The number of vessels with severe obstruction and the degree of **LV** dysfunction are important variables in predicting cardiac events. The ejection fraction is the best predictor of significant cardiac events and survival. Coronary angiography also can help identify coronary anomalies and other forms of congenital heart disease. Angiography is performed with the aim of identifying patients who may benefit from revascularization. Revascularization is indicated when the ischemic myocardium is present as the underlying substrate of ventricular tachycardia/ventricular fibrillation.

Electrophysiology studies (EPS): In targeted patients, **EPS** play diagnostic, prognostic, and therapeutic roles. **EPS** usually are performed after ischemic and structural heart disease has been diagnosed and addressed. These studies have been used to identify patients who have inducible versus noninducible sustained monomorphic ventricular tachycardia. The presence of inducible sustained ventricular tachycardia, at baseline or when the patient is on antiarrhythmic medications, confers a higher risk for sudden death. The significantly lower ventricular function also has been observed in patients with inducible sustained ventricular tachycardia. Inducible bundle-branch reentrant ventricular tachycardia can be seen in patients with Dilated Cardiomipopaty and in the postoperative period after valvular replacement. As many as 20% of patients with hypertrophic cardiomyopathy have inducible sustained monomorphic ventricular tachycardia. The identification of accessory pathways also is possible with these studies. EPS are performed with an eye toward the following:

Ablation of ventricular tachycardia foci, eg, bundle branch ventricular tachycardia, rvot ventricular tachycardia, and some cases of idiopathic LV tachycardia. ICD implantation, which is generally the case in survivors of sudden cardiac death (3-6).

General Management [13,16-20]

The main and unique finality in management a sudden cardiac death is that the patient can to recover from the same as soon as possible by means of the availability and suitable resources [17].

Chest compressions, cardioactive drugs and automatic (or semi-automatic) defibrillators are the first weapons that should be used. Especially when the patient is unknown, the event has happened in the street and there are not previous medical records.

The "**precordial coup**" before to chest compressions has been very reviled lately. However, many lives have been saved with this technique of "precordial coup" and should be considered yet. Although an "electro-mechanical dissociation" is present, this should not be relegated.

Epinephrine also has been questioned as a drug of first choice, trying to replace it with Vasopressine [14].

In general, **Advanced Cardiac Life Support (ACLS)** guidelines [17] should be followed in all cases of sudden cardiac arrest. Bystander **cardiopulmonary resuscitation (CPR)**: Immediate chest compression and defibrillation are reportedly the most important interventions to improve the outcome in the Sudden Cardiac Arrest [14].

The number of chest compressions and rescue breaths is still a very controversial matter. Every four years or so, this number changes according to the different "*Councils of cardiac resuscitation*." Until about two years ago, this number should be 100: 2. Currently, the number has returned to 30: 2. Even there are trends that advocate for not administer rescue breaths, only continuous chest compressions (**Cardio Cerebral Resuscitation: CCR) [19].**

Such controversies are intolerable. Research indicates that bystander use of automated external defibrillators increases neurologically intact survival to discharge (14.3% without bystander defibrillation; 49.6% with defibrillation) [20].

Pharmacologic Therapy

Ventricular arrhythmia: Epinephrine or vasopressin [14]; amiodarone and lidocaine can be used as antiarrhythmic drugs if defibrillation does not control the arrhythmia. Pulseless electrical activity **(PEA)**: Epinephrine; atropine used in case of bradycardia. Asystole: One study suggested that vasopressin is more effective in acute therapy for asystole than epinephrine [14]. Medical stabilization: Empiric beta blockers are reasonable in some circumstances [21].

But, we must always keep in mind that any antiarrhythmic drug is also pro-arrhythmogenic by nature.

Therapeutic hypothermia: This intervention limits neurologic injury associated with brain ischemia during a cardiac arrest and reperfusion injury associated with resuscitation [16]. This should be a mandatory application whenever the brain damage is potentially irreversible.

Surgery

Often are the ultimate solution. Temporary cardiac pacing. Radiofrequency ablation. Cardioverter defibrillator therapy. Coronary artery bypass grafting (CABG). Excision of ventricular tachycardia foci. Excision of left ventricular aneurysm. Aortic valve replacement. Orthotopic heart transplantation.

Prognosis

Generally, the prognosis is not good. Irreversible brain disorders reach more than 80% of cardiac arrest survivors.

Prognosis of morbidity and mortality for people who have had sudden cardiac arrest can be made using the cardiac arrest score developed by McCullough and Thompson or Rajat Deo, score **(Appendices 1 and 2).**

The detection of the underlying cause of sudden cardiac death and available treatment options plays an important role in the natural history and prognosis of sudden cardiac death.

Sudden cardiac death/Sudden cardiac arrest has been a frequently encountered problem for emergency physicians, internists, and cardiologists. Ischemic cardiomyopathy in all adult cases and hypertrophic cardiomyopathy in pediatric and adolescent cases are at the top of the list of causes of sudden cardiac arrest.

The clinical course, once the patient is resuscitated, largely is predicted by the ED presentation of hemodynamic stability, early neurologic recovery, and the duration of the resuscitation. Patients who survive the initial phases require a systematic evaluation of left ventricular performance, myocardial perfusion, and electrophysiologic instability.

Survivors of sudden cardiac arrest have a recurrence rate on the order of 20-25% per year, making **ICD** implantation important in the majority of these patients [3-6,20,21]. ICD implantation saves lives. Risk stratification will continue to be an area of active research.

Preventive measures, at their roots, are measures of coronary artery disease prevention. Efforts to inform and train the public about external defibrillator use likely will have a great public health impact on improving survival rates of Sudden cardiac arrest. However, more basic and clinical research is required to understand the mechanism of ventricular fibrillation/ventricular tachycardia and to be able to identify the patients at risk who benefit from ICD therapy.

Some Thoughts About Sudden Cardiac Death

A consensus on care and overall management of this problem is imperative. Currently there is none at all:

What is it? How does it occur? What is the true incidence? What are the most effective therapeutic measures?

We always have to 'start from by the beginning'. Therefore, the definition of such heartrending event is the first thing we must agree on [2]. As we already have written previously, all the unexpected is sudden, but not all the sudden is unexpected. Therefore, the definition should be **"Unexpected Cardiac Death" (UCD)** and not "Sudden Cardiac Death". **(SCD)**: A death can be expected during a long time period (by example, a terminal cancer), however, the passage from life to death is always sudden and very subtle.

On the other hand, it is always necessary to emphasize that the "Unexpected cardiac death" in the vast majority of the time, is caused by multifactorial causes. And not just a factor is the only trigger for it.

True etiology is usually unknown at this time, at least totally. There are not just one, but several causes in the same individual: it can be the predominant, but not the only. As an example, a person may have a 'Hypertrophic Cardiomyopathy' and besides also can have a lethal abnormal heart rhythm from birth and being a habitual consumer of drugs, alcohol and snuff.; all them can be 'asleep' for several years and unleash death by another unknown hostile element. That is to say, it is totally inappropriate to mention that the most common cause unexpected cardiac death is only the Hypertrophic Cardiomyopathy, or dilated, or produced by a WPW syndrome or a Brugada or a Breijo pattern in isolated form, because usually they are mixed: Really, we speak of death- in general- when the cardiac rhythm has disappeared and just an asystolia can be seen on the ECG. That is, the final cause of death is always a complete abolition of the cardiac rhythm.

In the field of the **Epidemiology**, to speak about prevalence and incidence is truly very biased. Noteworthy that in many cases, sufferers go unnoticed because can be very lightweight the 'warning signs (or symptoms)' and sometimes the patient does not comment with doctors, and in sometimes are the doctors who do not reach an accurate diagnosis. Unfortunately, the latter is all too common: many symptoms, signs as well as test interpretations are too often underestimated.

The athletes theme, is being very controversial since many of them have not been studied under their relationship with doping during their active stage; this study of narcotic drugs in athletes is absolutely imperative. It is evident that the heart is much more vulnerable to the unexpected death when he has a previous alteration. But we must also bear in mind that a healthy heart may suffer from sudden death when the intake of drugs to improve sports performance is abusive. There are very few clinical studies about it.

For Harmon K., *et al.* in their clinical essay entitled '*Sudden cardiac death risk may be underestimated in NCAA athletes*', the sudden cardiac death accounted for more than half of the medical-related fatalities in a cohort of collegiate student-athletes in the US, according to study results. According these authors, 68 % of those deaths were attributed to nonmedical or traumatic causes, 29% were due to medical causes and 2% were the result of unknown causes. Among the medical cases, CV-related sudden death has been the most frequently

reported causes, accounting for 56% of the fatalities. CV-related sudden deaths also accounted for 75% of medical-related sudden deaths during exertion. So, we can say that the accurate assessment of the incidence of sudden cardiac death is very necessary to shape appropriate health policy decisions and develop effective strategies for prevention. However, such accurate and rigorous assessment conspicuous by its absence, currently.

Regarding to the immediate treatment of cardiac arrest, discrepancies are awe inspiring. All guidelines are only speculations. All recommendations from AHA are very weak and with a very-low-quality evidence. Nearly 100% of all hospitals in the world follow the guidelines set forth by ILCOR. After, they elaborate their own rules and rarely the indications given by ILCOR are practiced. The lawsuits for not following these rules are too frequent. This is a real ballast for those physicians who have not followed the same and however have saved the life of the patient.

In a manifest cardiac arrest: how many thoracic compressions/minutes are necessaries? How many rescue breaths?

That question is another great discussed matter. How many times of rescue breath must be performed? Different specialized Councils on this matter are not in agreement yet. 100 over 2?, 30 over: 2? How many really are necessaries for a better result? It is truly disappointing, even disheartening, to read how there is no any evidence, nor 'controlled' clinical trials at the time of developing an effective guideline in the management of cardiac resuscitation during a cardiac arrest.

The ILCOR manifests its very several limitations on issues of effectiveness, certainty, reliability and positive results on the latest consensus on the cardiac resuscitation during a cardiac arrest [17].

What is a 'Cardiac arrest'? A rhythm of ventricular fibrillation? . Obviously they are not the same thing. In like manner that a 'respiratory arrest' a 'cardiac arrest' are not the same.

As the colophon to these thoughts, just say that, to override all discrepancies and disputes expressed in this document, many more studies (controlled, prospective, with the largest number of assessed people possible, and above all, based on an indisputable evidence) are necessaries in an imperative manner. This must be the way to follow. Given that so cruel event can occur at any time and anywhere, the early diagnosis of its causes is a priority. Even when the patients refer very weak and nonspecific symptoms. We think that some simple diagnostic tests (ECG and Echocardiography, mainly) in individuals with a known risk factor for SCD should always be performed. They are not expensive and yet these procedures can greatly help in early detection. In this way, preventive measures could avoid the terrible event. However, discrepancies in this topic are supreme: the sensitivity and specificity of these diagnostic methods take precedence over the potential benefits for many authors.

Studies on the effects of alcohol and drugs (design, cocaían, heroin, etc.) are very sparse. The effects of alcohol on the heart should be taken into account as much as has been done with smoking (in many countries, there is a very aggressive campaign against snuff and very permissive with alcohol: both are very harmful to the heart, not only the snuff). Humbly we think that these nuances can help a lot in the control and management of unexpected cardiac death. Many more lives could be recovered from an unexpected cardiac death.

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22. Appendices

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