Sudden cardiac death

What is sudden cardiac death?

Sudden cardiac death (SCD) occurs when the heart completely stops beating and pumping blood. SCD is clinically defined as unexpected death from a cardiac cause occurring within a short time, usually within 1 hour of symptom onset.[1] Normally, a natural 'pacemaker' in the heart triggers electrical impulses that travel along defined pathways within the heart, allowing it to beat in a regular, controlled fashion at a rate of between 60 and 80 beats per minute (bpm). However, the electrical system in the heart can become irregular (known as an arrhythmia) and cause the heart to beat uncontrollably fast, at a rate of 120–200 bpm (ventricular tachycardia), or cause the ventricles of the heart to flutter or quiver erratically (ventricular fibrillation).[2,3] In SCD, ventricular tachycardia and ventricular fibrillation can cause the heart to lose its ability to effectively pump blood, thus stopping blood flow to the body and the brain. This leads to loss of consciousness and death if medical treatment is not administered immediately. Although less common, SCD can occur if the irregular electrical activity causes the heart to beat too slowly (bradycardia).[2,3]

What is the magnitude of the problem?

SCD due to coronary disease is the most important cause of death in Europe and the developed countries, accounting for half of all deaths from cardiovascular disease.[1,4] Studies have reported that the incidence of SCD ranges from 0.36 to 1.28 per 1000 people per year.[4] Based on this estimate, SCD would account for 262,000 to 934,000 deaths each year in a European population of 730 million. In the US, more than 300,000 deaths per year are due to SCD.[3] The incidence of SCD in less-developed countries is lower, running parallel with the rates of ischaemic heart disease.[5] However, these figures are unlikely to show the true magnitude of the problem because only witnessed events are recorded.

What are ventricular tachycardia and ventricular fibrillation?

Ventricular tachycardia (VT) and ventricular fibrillation (VF) are types of ventricular arrhythmias. VF is the most common cause of SCD, and is responsible for 75–80% of cases.[6]

VT is defined as 'three or more beats of ventricular origin in succession at a rate greater than 100 bpm.[7] VT usually involves a breakage in the electrical link between the atria (top chambers of the heart) and the ventricles (atrioventricular dissociation). This means that the sinus node (the natural pacemaker) causes the atria to contract in a normal manner, while the ventricles contract at an independent, abnormal rate that is equal to or faster than that of the atria. VT may be well tolerated or may prevent the heart from pumping enough blood to the body (impaired haemodynamic functioning)[7]. SCD often occurs when VT progresses to VF.[8] During VF, the electrical signals that control the contractions of the heart become erratic and cause the lower chambers of the heart (ventricles) to begin to quiver (fibrillate) instead of contract, preventing the heart from pumping blood to the rest of the body. The haemodynamic effects of VT and VF depend largely on the presence or absence of underlying structural heart disease (for example ischaemic heart disease).

What is bradycardia?

Ventricular bradycardia is responsible for a minority of cases of SCD (~20%), and is a general term that describes a number of conditions in which the heart beats at an unusually slow rate (less than 60 bpm).[6] It is usually the result of the slowing or blockage of the electrical signal between the atria and the ventricles (atrioventricular block). This results in a slowing of the ventricular beat, while the atria remains beating at a normal rate. Ventricular bradycardia reduces haemodynamic efficiency resulting in decreased blood circulation, which in turn may cause fainting, dizziness, shortness of breath and, in some cases, death.

Bradycardia can be caused by several different factors, including normal age-related degeneration of the heart's electrical conduction system,[9] coronary artery disease resulting in ischaemic damage to the heart tissues, and some congenital heart defects. It can also be

caused by certain medicines including those that treat arrhythmias and some anti-HIV drugs.[10,11]

Populations at risk of sudden cardiac death

Almost all people with underlying heart disease are at risk of SCD, but people with the following conditions will be discussed in this section:

- Coronary artery disease
- Inflammation of the heart muscle (cardiomyopathy)
- Heart attack (myocardial infarction)
- Heart failure

At least 80% of patients who experience SCD have coronary artery disease. The factors that predict the occurrence of SCD are generally the same as those for coronary artery disease: increasing age, male gender, family history of coronary artery disease, increased LDL cholesterol, hypertension, smoking, and diabetes mellitus.[6]

The second most important risk factor for developing SCD is inflammation of the heart muscle (cardiomyopathy). SCD occurs in around 2–6% of adults and children who have cardiomyopathy.[12] There are two types of cardiomyopathy – hypertrophic and dilated cardiomyopathy. Hypertrophic cardiomyopathy is an inherited disorder characterised by overgrowth of the heart muscle and scarring.[12] This overgrowth and scarring of the muscle contributes to a broad range of abnormalities that leads to arrhythmias and heart failure. SCD associated with hypertrophic cardiomyopathy often occurs in young adults, most of whom have not previously shown any cardiac symptoms.[13] Dilated cardiomyopathy is characterised by the enlargement of the left ventricle and impaired ability to pump blood. SCD accounts for around 30% of all deaths in patients with dilated cardiomyopathy and can affect those with either advanced or mild symptoms.[6]

Previous heart attack (myocardial infarction) resulting in tissue damage is a major indicator for SCD. The rates of SCD in patients who have had a previous heart attack have been

shown to be 4–6 times greater than the rates in the general population.[3] In patients whose heart attack resulted in a reduction in left ventricular ejection fraction to 30% or less (a normal value is at least 55%), the risk of SCD continues to increase.[14]

Heart failure is a major cause of serious morbidity and death in the population and is one of the leading causes of hospitalisation in people aged over 65 years.[15] Heart failure may occur as the end result of damage caused by a number of disease processes including coronary artery disease, hypertension, valvular defects, alcohol misuse and viral infection.[15] The disease is slow and progressive and is characterised by a loss of muscle from the heart (myocardium). This means that the heart is unable to pump as well as it should, decreasing blood circulation. As a result of impaired blood circulation, both the heart and the body suffer from reduced oxygen and nutrient supplies, which ultimately results in damage to the tissues. This damage to the heart can affect the electrical signalling pathways, potentially causing ventricular tachycardia and bradycardia – major causes of SCD.[1,6] In patients with advanced heart failure, bradycardia may account for up to 62% of SCD.[6] In people diagnosed with heart failure, the incidence of SCD is 6–9 times the rate of the general population.[3]

Can SCD be prevented?

Primary prevention

The risk of SCD can be reduced by making lifestyle modifications that include eating a healthy diet and exercising regularly. Patients with known cardiac disease and documented arrhythmias can be treated with pharmacological therapies, including antiarrhythmic drugs such as ACE inhibitors, beta-blockers, calcium channel blockers and statins.[3,16] Patients can also be treated with an implantable cardioverter-defibrillator (ICD), which is a small device that monitors and then corrects arrhythmias. Both of these treatments are recommended in guidelines for the prevention of primary attacks.[10]

Secondary prevention

Patients who have already experienced sudden cardiac arrest (SCA) can be revived using cardiopulmonary resuscitation (CPR) and an electric shock to the heart (defibrillation) to

restore the heart beat. Once revived, patients require antiarrhythmic drugs and an ICD to prevent secondary attacks.[16]

Pharmacological treatment

Pharmacological agents aim to correct ischaemia, prevent plaque rupture, stabilise the electric conduction system, improve pump function and prevent arrhythmias.[1] Prevention of arrhythmias is the most important goal in the treatment of SCD, however, the identification of an effective antiarrhythmic drug regimen is only possible in a minority of patients with documented life-threatening ventricular arrhythmias.[10]

With the exception of β-blockers, currently available antiarrhythmia drugs have not conclusively been shown to be effective in the primary management of patients with life-threatening ventricular arrhythmias or in the prevention of SCD.[10] In fact, many marketed cardiac drugs prolong the period between ventricular relaxation and contraction (repolarisation period) and have the potential to cause life-threatening ventricular tachyarrhythmias. For example, the effectiveness of amiodarone in improving SCD mortality is controversial and some studies suggest that it may be associated with thyroid dysfunction, leading to worsening arrhythmias and, in extreme cases, death.[17-20]

In patients with left ventricular dysfunction (both before and after a heart attack), antiarrhythmic drugs do not always reduce mortality. This is due to the many different kinds of arrhythmia that can develop from this disorder. Patients are treated with antiarrhythmics to reduce symptoms until they can receive an ICD.[10]

Implantable cardioverter defibrillator (ICD)

An implantable cardioverter defibrillator (ICD) is a device that helps stop dangerously fast heart rhythms in the ventricles and restore a normal heartbeat. For many patients that die suddenly, ventricular fibrillation is the primary reason. For these patients, treatment with an ICD prevents more secondary attacks than antiarrhythmic drug therapy alone.[21] This superiority is still evident after 11 years and increases over time.[22] Implantable cardioverter-

defibrillators reduce the risk of SCD by 63% over conventional drug therapy and this has resulted in ICDs being adopted as the primary treatment in patients who have survived SCD, and those who survive life-threatening disturbances in blood circulation due to VT.[11]

Prophylactic (preventative) use of ICD has been shown to reduce the rates of sudden deaths by 80% and reduced the overall rate of mortality by 54% after 2 years, compared with conventional antiarrhythmia drugs in patients with a left ventricular ejection fraction of less than 35%. This striking reduction in overall mortality and SCD has been shown in several clinical trials.[20,23] A significant benefit has also been shown in patients that have had a previous heart attack resulting in left ventricular damage. The implantation of an ICD resulted in a reduced risk of SCD compared to conventional therapy, which actually showed an increased risk of SCD.[24]

How does an ICD work?

The device is typically inserted into the body just under the patient's collarbone. Thin, insulated leads that are connected to the device are run down veins into the heart. Depending on the model, one lead is attached inside the right ventricle (single chamber ICD), a second can be attached inside the right atrium (dual chamber ICD) and a third can be attached to the outside of the left ventricle (biventricular ICD).

The computer within the ICD, which can be remotely programmed by the physician, monitors the rhythm of the heart, identifies abnormal rhythms and determines the appropriate therapy. The device delivers electric shocks that increase in intensity as the heart rhythm worsens: very low levels of energy are used for antitachycardia pacing, then low levels of energy or 'mild shocks' are used for cardioversion, and finally, if necessary, a high energy shock is used for defibrillation.

When the device is pacing, the patient is very unlikely to feel anything. However, when cardioversion occurs, the patient is likely to feel a thump in the chest which does not linger.

In the case of defibrillation, if the patient is conscious, the electric shock feels like a kick in the chest, which only lasts for a moment.

ICD are cost-effective for SCD prevention

As well as increasing the number of years that patients at risk of SCD are likely to survive, ICDs have also been shown to be cost effective when compared with medical treatment. Major clinical trials have shown that, although there is initially a greater expense with ICD (due to the cost of the device and the surgery), medical therapy quickly becomes more expensive on a monthly basis. This is especially evident in high-risk patient groups where quality of life is a particular issue.[25-27]

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