Implantable cardioverter defibrillators

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Who needs an implantable cardioverter defibrillator?

In the UK, sudden cardiac death (SCD) occurs in 70,000–100,000 patients annually, mainly caused by ventricular arrhythmias. Most of these patients have recognised heart disease with either a previous myocardial infarction (MI) or left ventricular dysfunction, but SCD is the first presentation of heart disease in 20% of patients.

The mainstay of treatment is neurohormonal modulation with beta-blockers and angiotensin-converting enzyme inhibition. While this reduces morbidity and mortality across the spectrum of cardiovascular disease, patients who have had an MI and heart failure with significant left ventricular systolic dysfunction continue to have a high rate of SCD.

The first implantable cardioverter defibrillator (ICD) to manage SCD was implanted in a human by Michel Mirowski in 1980 (Fig 1). Since then there has been an explosion in technology and randomised control trial data to support their use.

What are the components of an implantable cardioverter defibrillator?

An ICD comprises:

- a lithium silver vanadium oxide battery, which provides low voltage energy
- a transformer which multiplies this voltage
- an aluminium electrolytic capacitor which can store the high energy voltage for use, and
- sensing circuitry which can sense local electrograms and filter out noise like skeletal myopotentials.

ICDs have at least one lead in the right ventricle for pacing, sensing and defibrillation. There may also be a second lead or coil in the superior vena cava to improve the defibrillation threshold. Additional leads can be placed in the right atrium for dual chamber pacemaker indications or to help in the detection or discrimination of arrhythmias. In patients with symptomatic heart failure and dyssynchrony of ventricular contraction a further lead can be placed in the lateral tributaries of the coronary sinus for cardiac resynchronisation (Fig 2).

Fig 1. Michel Mirowski MD (1924–90).

The basic detection of ventricular arrhythmias involves measuring heart rate above which therapy will be delivered. Rate detection is very reliable, but unfortunately susceptible to delivering inappropriate therapy for sinus tachycardia or poorly controlled atrial fibrillation. Modern devices can be programmed to have multiple detection zones with detection enhancements to help prevent inappropriate shocks.

When ventricular arrhythmias are detected the device will try to terminate them by either antitachycardia pacing or delivering a shock. Most ICDs can deliver a maximum energy of 30–41 J via their capacitors as a biphasic waveform from the tip of the right ventricular lead to the generator or 'active can'. The shocking vector travels superiorly from the right ventricle to include most of the interventricular septum and left ventricle (Fig 3).

What are the indications for an implantable cardioverter defibrillator?

Randomised trials comparing ICDs and medical therapy have been conducted largely in patients with underlying ischaemic heart disease (IHD) who make up the majority of recipients. Other

Key Points

- **Sudden cardiac death occurs in 70,000–100,000 people annually in the UK, mostly in people with pre-existing heart disease but in 20% it is the first presentation**
- **Previous myocardial infarction (MI) and significant left ventricular dysfunction are associated with a high rate of sudden cardiac death**
- **Implantable defibrillators are recommended in the absence of a reversible cause for survivors of a cardiac arrest or in sustained ventricular arrhythmias if associated with syncope, haemodynamic instability or a left ventricular ejection fraction (LVEF) below 35%**
- **Implantable defibrillators are recommended as primary prophylaxis in patients who have had an MI and have an LVEF below 30% based on trial evidence (MADIT II) and the National Institute for Health and Clinical Excellence guidelines**

KEY WORDS: ischaemic heart disease, left ventricular dysfunction, sudden cardiac death, ventricular arrhythmias

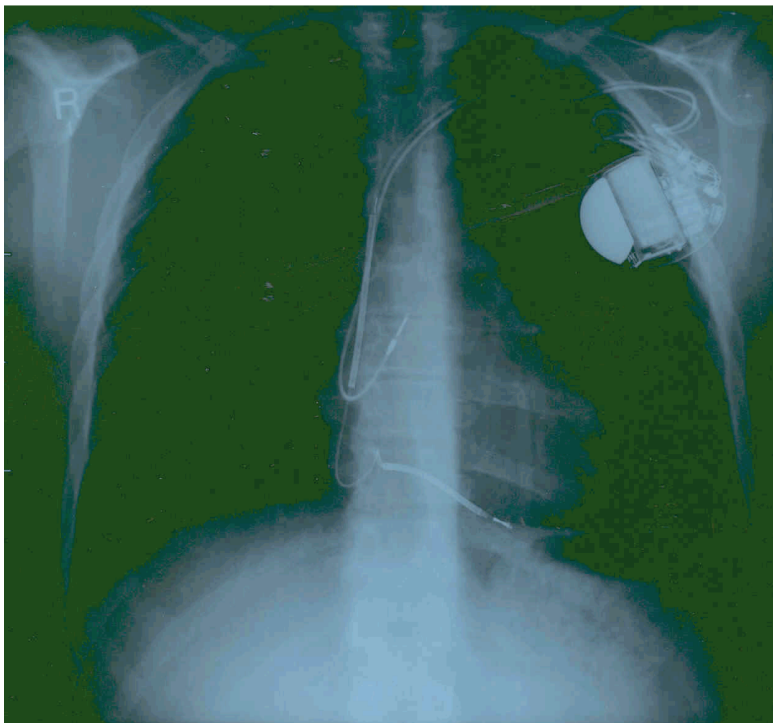

Fig 2. Dual chamber implantable cardioverter defibrillator.

important patient groups include those with cardiomyopathies and ion channel disorders. The National Institute for Clinical Excellence issued a revised technology appraisal for ICDs in January 2006.[1] These guidelines do not however apply to patients with non-ischaemic dilated cardiomyopathy.

Primary prevention is prophylaxis against a first life-threatening event. It is recommended for patients at least four weeks after an MI who either have a left ventricular ejection fraction (LVEF) of less than 35%, non-sustained ventricular tachycardia (VT) on Holter monitoring and inducible VT during electrophysiological testing, or LVEF of less than 30% and QRS duration of at least 120 ms. Primary prevention is also recommended in familial cardiac diseases with a high risk of SCD such as hypertrophic (HCM) and arrhythmogenic right ventricular cardiomyopathies (ARVC), long QT and Brugada syndromes, and in surgically corrected congenital heart disease.

In the absence of a reversible cause, secondary prevention is advised for survivors of a cardiac arrest due to ventricular arrhythmia, patients with sustained VT and syncope, haemodynamic instability or LVEF below 35%.[1]

What is the trial evidence for these guidelines?

Secondary prevention trials

The first trials to investigate the benefits of ICDs versus antiarrhythmic drugs were conducted in survivors of SCD. Important studies included the AVID,[2] CIDS[3] and CASH[4] trials (the full names of trials are given at the end of the text). These studies enrolled patients with and without IHD with LVEF below 40%, randomising them to ICD implantation or drug therapy with either amiodarone or beta-blockers. The results showed a significant mortality reduction with ICD treatment.

Primary prevention trials

Several trials investigated the benefits of ICDs for prevention of SCD in patients with no previous history of haemodynamically significant ventricular arrhythmias. Important studies included the MADIT,[5,6] MUSTT,[7] DINAMIT,[8] CABG Patch,[9] SCDHeFT[10] and DEFINITE[11] trials. The first four included only patients with IHD, but SCDHeFT and DEFINITE included non-ischaemic patients.

MADIT I and II, MUSTT, DINAMIT, CABG Patch. The MADIT II trial showed a 31% reduction in all-cause mortality in patients with a previous (more than 30 days) MI and LVEF less than 30%, given conventional drug therapy and an ICD. Unlike MADIT I and MUSTT, no documented ventricular arrhythmias or electrophysiology studies were required. Conversely, the DINAMIT and CABG Patch[10] trials both failed to demonstrate any benefit of ICDs. DINAMIT enrolled patients 6–40 days post-MI who also had indicators of autonomic dysfunction suggesting progressive heart failure. Consequently, this study either selected out a higher risk population or the benefits of ICD implantation may have been attenuated by conventional drug therapy. The lack of benefit from early ICD implantation has also been confirmed by retrospective analysis of the MADIT II population.

CABG Patch implanted ICDs prophylactically in patients with left ventricular dysfunction at the time of coronary artery bypass surgery (CABG). The lack of survival benefit from ICDs in this study probably highlights the benefits of revascularisation in preventing SCD and the increased risks of device implantation during cardiac surgery.

SCDHeFT. SCDHeFT was the first study in heart failure patients with both ischaemic and non-ischaemic aetiology. It found a 23% reduction in all-cause mortality in patients with New York Heart Association (NYHA) class 2 or 3 heart failure and LVEF below 35% given conventional drug therapy and an ICD.

DEFINITE. The DEFINITE trial[11] randomised patients with non-ischaemic dilated cardiomyopathy with LVEF less than 36% and NYHA class 1–3 to best medical therapy with or without an ICD. All-cause mortality was not significantly reduced by ICDs but there was a significant reduction in arrhythmic

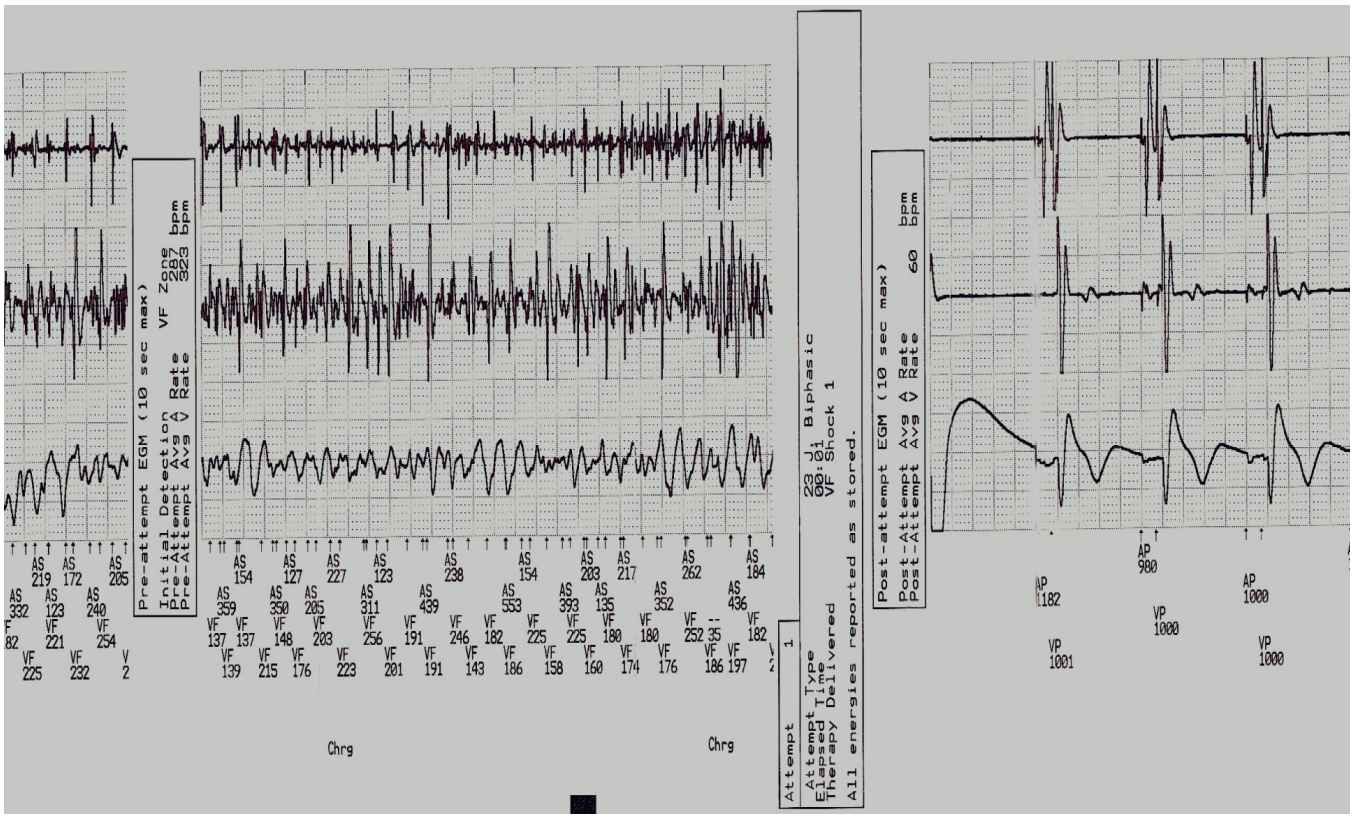

Fig 3. Detection and successful cardioversion for ventricular fibrillation.

deaths with the greatest benefit seen in NYHA class 3 patients. Risk stratification of these patients is difficult since electrophysiology studies are not helpful at predicting SCD. However, patients presenting with syncope who had ICDs implanted go on to have appropriate ICD therapies for ventricular arrhythmias.[12] Consequently, patients with dilated cardiomyopathy who have a syncopal episode should be offered ICD implantation.

Survivors of sudden cardiac death

Patients with HCM or ARVC who survive SCD or have haemodynamically unstable VT should all have an ICD implanted for secondary prevention.[13]

Hypertrophic cardiomyopathy

In HCM, the risk stratification of patients to guide ICD implantation for primary prevention is more difficult. Currently, the major risk factors for SCD include syncope, family history of SCD, non-sustained VT on Holter monitoring, interventricular septal thickness greater than 30 mm and an abnormal blood pressure response during exercise testing.

Arrhythmogenic right ventricular cardiomyopathy

In ARVC, risk stratification for primary prevention is difficult due to a lack of data. However, patients with a family history of SCD who present with syncope should be deemed at high risk and undergo electrophysiology studies. There is some evidence to suggest that inducible ventricular arrhythmias are associated with appropriate ICD therapies.[14]

Brugada syndrome

Both the Brugada and long QT syndromes (LQTS) arise because of inherited ion channel abnormalities that predispose to ventricular arrhythmias and SCD.

The Brugada syndrome is characterised by coved ST elevation in leads V1 and V2 (type 1) that may occur spontaneously or be unmasked by sodium channel blockers such as flecainide and ajmaline. Patients presenting with aborted SCD, syncope, seizures or nocturnal agonal respiration should undergo ICD implantation,[15] although no prospective, randomised trials have evaluated ICD therapy in these patients.

Asymptomatic patients with a family history of SCD should be risk stratified with a VT stimulation test and undergo ICD implantation if this is positive. However, the supporting data are equivocal. Asymptomatic patients without a family history should be risk stratified only if the type 1 changes are spontaneous. All other patients should be followed up closely.[15]

Long QT syndrome

There are two well-known inherited forms of LQTS, but a modern gene based classification has now replaced these eponymous syndromes with at least six

Explanation of trials with implantable cardioverter defibrillators

AVID	Antiarrhythmics Versus Implantable Defibrillators
CABG Patch	Coronary Artery Bypass Graft Patch
CASH	Cardiac Arrest Study Hamburg
CIDS	Canadian Implantable Defibrillator Study
DEFINITE	DEFibrillators In Non-Ischemic cardiomyopathy Treatment Evaluation
DINAMIT	Defibrillator IN Acute Myocardial Infarction Trial
MADIT	Multicenter Automatic Defibrillator Implantation Trial I and II
MUSTT	Multicenter UnSustained Tachycardia Trial
SCDHeFT	Sudden Cardiac Death in Heart Failure Trial

chromosome loci (LQTS 1–6) coding for six genes identified. Each genetic syndrome can be characterised by distinct clinical features. Patients surviving SCD or presenting with recurrent syncope are considered at high risk and should undergo ICD implantation in addition to beta-blockers.

Most patients do not die from LQTS but are at risk (13% risk of a fatal event over a lifetime if untreated), but predicting risk is extremely difficult. All patients will require careful counselling. A recent study has suggested high-risk patients include:

- LQTS 1 if QTc is greater than 500 ms in men and women
- all men with LQTS 2
- women with a QTc above 500 ms
- all patients with LQTS 3.[16]

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