**REUE | Review article**

# Automatic implantable defibrillators: an update for emergency physicians

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Implantable cardioverter defibrillators (ICDs) are first-line therapeutic devices for preventing sudden death due to ventricular arrythmia. The antitachycardia pacing of ICDs terminates a large proportion of arrhthymias painlessly. Energy consumption is low, avoiding shocks from the device. The implanted leads required by conventional ICDs have been associated with a high rate of complications related to the implantation process and to vascular injury, pneumothorax, perforations, and more. Long-term complications related to malfunctioning leads or infection at the implant site can oblige removal. Subcutaneous ICDs (S-ICDs), developed to minimize complications caused by endovascular leads, have been used in Spain since 2013 and in other parts of Europe since 2009. S-ICDs detect and treat arrhythmias with shocks that are always greater than those delivered by transvenous ICDs; moreover, S-ICDs cannot provide antitachycardia pacing. So far, S-ICDs have proven highly effective and safe, producing a very low rate of inappropriate shocks thanks to the various improvements in software and programing. They are larger than transvenous ICDs, however, and their batteries are shorter lived because they deliver much higher-energy shocks. S-ICDs also lack the ability to provide antibradycardia pacing. A new defibrillation system has been developed in recent years in an attempt to overcome the disadvantages of S-ICDs while retaining their advantages. The shock generators of the new extravascular implantable devices (EV-ICDs) are the same size as those of transvenous ICDs and theoretically have the same life span. They are implanted inside the left lateral chest wall, and a defibrillation and pacing lead is placed in the retrosternal space, covering the craniocaudal axis of the cardiac silhouette. This system offers low-energy cardiac defibrillation and is able to provide both antitachycardia and antibradycardia pacing. Because the potential complications of using these novel devices may generate emergency department visits, we must become familiar with them, given that treatment is not exclusively the remit of cardiologists and experts in arrhythmias but of all physicians who manage cardiovascular emergencies.

**Keywords:** Implantable cardioverter-defibrillator. Ventricular arrhythmias Sudden cardiac death.

## *El desfibrilador automático implantable: actualización para personal de urgencias y emergencias*

El desfibrilador automático implantable (DAI) es una herramienta terapéutica de primera línea para evitar la muerte súbita por arritmias ventriculares. La estimulación antitaquicardia de los desfibriladores, permite en un porcentaje elevado terminar las arritmias de forma indolora y con bajo consumo de energía, al evitar choques o descargas del dispositivo.
El implante de cables necesario en el DAI convencional se ha relacionado con una tasa elevada de complicaciones, tanto en el proceso del implante como por daño vascular, neumotórax, perforaciones, etc., así como a largo plazo, por disfunción de los cables o infecciones del sistema que obligan a la retirada. Para tratar de minimizar tales complicaciones relacionadas con los cables endovasculares, se ha desarrollado un desfibrilador subcutáneo (DAI-SC) que se implanta en Europa desde 2009 y en España desde 2013; permite la detección de las arritmias y su tratamiento mediante descargas de energía, siempre mayores que la necesaria con los desfibriladores transvenosos, y con imposibilidad de proporcionar estimulación antitaquicardia. En la actualidad, muestra una alta eficacia y seguridad, con una muy baja tasa de descargas inapropiadas, resultado de diferentes mejoras de programación y software de los dispositivos. Otras diferencias respecto a los desfibriladores transvenosos son el mayor tamaño y la menor longevidad de la batería, derivadas de la necesidad de aplicar choques de mucha mayor energía, y la ausencia de capacidad de estimulación crónica antibradicardia. Al objeto de solventar las desventajas del DAI-SC, pero conservar sus ventajas, en los últimos años se ha desarrollado un nuevo sistema de desfibrilación, el desfibrilador extravascular (DAI-EV), en el que se implanta un generador de tamaño igual al de un desfibrilador transvenoso y con igual longevidad teórica, en la pared torácica lateral izquierda, y un cable de desfibrilación y estimulación que se implanta en el espacio retroesternal cubriendo el eje craneocaudal de la silueta cardiaca. El sistema permite la desfibrilación cardiaca con baja energía, y trata de conseguir estimulación cardiaca que permita dar terapias antitaquicardia y estimulación antibradicardia. Las potenciales complicaciones de estos novedosos dispositivos pueden ser motivo de consulta en urgencias, por lo que resulta necesario estar familiarizados con ellos, ya que su tratamiento no es exclusivo de los arritmólogos o cardiólogos, sino de todos los médicos que se relacionan con las urgencias cardiovasculares.

**Palabras clave:** Desfibrilador automático implantable. Arritmias ventriculares. Muerte súbita cardiaca.

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## The problem of sudden cardiac death

Sudden cardiac death (SCD) is defined as natural and unexpected death of cardiac origin that occurs within the first hour after the onset of symptoms. It affects approximately 30,000 people per year in Spain, about 10 times more than the number of deaths caused by traffic accidents.[1]

Over the past 20 years, cardiovascular mortality has decreased in developed countries following preventive measures aimed at reducing ischemic heart disease (IHD) and heart failure (HF). Despite these encouraging results, cardiovascular diseases cause approximately 17 million deaths per year worldwide, with SCD accounting for about 50% of all cardiovascular deaths, and in up to 50% of cases it is the first sign of heart disease.[2]

The incidence rate of SCD increases with age, being very low during childhood and adolescence (1 per 100,000 persons/year).[3-5] In middle age (5th–6th decades), the incidence rate rises to approximately 50 per 100,000 persons/year,[6-8] and reaches at least 200 per 100,000 persons/year[9] by the 8th decade of life. At all ages, SCD is more common in men than in women, even after adjusting for coronary risk factors.[10-12] Across Europe, 20% of all deaths are attributed to SCD.[13,14]

The epidemiology of SCD is closely linked to coronary artery disease (CAD), which is responsible for 75–80% of all SCD cases.[15] While the prevalence of CAD has not declined, mortality from this condition has. Thus, although there is a downward trend in SCD incidence among patients with CAD,[15-17] the increasing prevalence of cardiovascular disease has resulted in a rise in the absolute number of SCD cases as a contributor to cardiovascular mortality.[18,19]

The cardiac conditions associated with SCD vary by age. Among younger populations, primary electrical disorders, cardiomyopathies, myocarditis, and congenital cardiac lesions predominate.[20-25] In adults (from the 4th decade onward), CAD—and specifically acute coronary syndrome—is the leading cause, accounting for 50% of all SCDs.[26,27] In the elderly, chronic structural heart diseases (coronary disease, valvular heart disease, and HF) are more common.[28]

Strategies for the prevention of SCD depend on recognizing the diseases that may lead to it. In recent years, significant advances have been made in identifying genetically determined disorders such as long QT syndrome, idiopathic ventricular fibrillation (VF), Brugada syndrome, short QT syndrome, and catecholaminergic polymorphic ventricular tachycardia (CPVT), as well as arrhythmogenic right ventricular dysplasia (ARVD) and hypertrophic cardiomyopathy.[1]

SCD is multifactorial, and any effort to combat it requires a comprehensive approach. It is therefore essential to understand the causes, identify patients at risk, find effective treatments, and raise awareness that sudden death is largely preventable. This process integrates the recognition of risk factors and potentially lethal conditions, preventive strategies, and the treatment of arrhythmias responsible for most SCD episodes—through both early external defibrillation and advanced primary and secondary prevention options, including implantable cardiac devices, arrhythmogenic substrate ablation, neurohumoral modulation, and heart transplantation.[28]

SCD is therefore potentially avoidable in a high proportion of cases, and recognizing patients at risk makes it possible to implement comprehensive preventive and therapeutic strategies to avoid or delay fatal outcomes. This recognition spans from classical clinical evaluation, population and individual risk profiling, and cardiac electrophysiological and functional assessment, to neurohumoral evaluation and modern genotyping techniques, which are increasingly accessible. A key element in this therapeutic strategy is the implantable cardioverter-defibrillator (ICD).[1]

## The implantable cardioverter-defibrillator (ICD): fundamentals and device types

The ICD represents one of the most important advances in the prevention of SCD in patients with ventricular arrhythmias and in improving survival among those at high risk of arrhythmic sudden death.

Between the 1950s and 1980s, the foundations were laid for the design and implantation of devices capable of detecting and treating so-called "malignant ventricular arrhythmias" (sustained VT and VF). The first implantable defibrillator was designed in the late 1960s by Michel Mirowski. In 1970, experimental models based on the design by Mirowski and Morton Mower were produced, and in 1980, Mirowski's team implanted the first automatic defibrillator in a young patient with recurrent VF.[29] In 1985, the U.S. Food and Drug Administration (FDA) approved the commercialization of ICDs. The earliest devices were rudimentary, delivering shocks based solely on mean heart rate. Over time, their functions improved, size decreased, and diagnostic and therapeutic capabilities expanded. In recent years, technological advances have significantly enhanced performance, reduced size and weight, and expanded clinical indications for both secondary and primary prevention of SCD.[30,31]

The first ICD implant in Spain was performed in 1985. It was a non-programmable, 250-gram abdominal device, with epicardial electrodes and patches placed via sternotomy.

Currently, ICDs are categorized into 3 types, depending on the number of chambers stimulated:

– Single-chamber: stimulates 1 chamber (right ventricle).

– Dual-chamber: stimulates 2 chambers (right atrium and right ventricle).

– Triple-chamber or cardiac resynchronization therapy (CRT) system, also called biventricular: stimulates 3 chambers (right atrium, right ventricle, and left ventricle).

The ICD continuously monitors the heart's electrical activity, detects ventricular arrhythmias, and terminates them through shocks to restore normal rhythm. The transvenous ICD is the most widely used, but it carries risks associated with intracardiac leads. To address this, the subcutaneous ICD (S-ICD)[32] was developed as an alternative,

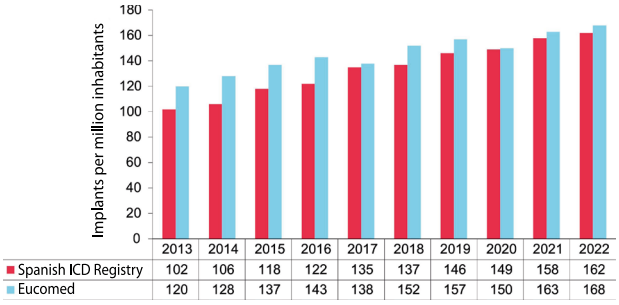
implanted via a subcutaneous route, avoiding intracardiac leads altogether.

## Epidemiology and trends in ICD implantation

The ICD is now one of the main therapeutic options for patients who have experienced malignant ventricular arrhythmias or recovered SCD. Its use for primary prevention in at-risk patients has also grown in recent years. Combined with technological improvements, the limited efficacy of antiarrhythmic drugs, and evidence from clinical trials, ICD indications have expanded exponentially. Consequently, the number of implanted patients—and related device complications—has increased.

Cardiovascular patients are frequent users of emergency services (EDs), particularly during acute events or exacerbations of chronic disease. Thus, ICD carriers are becoming increasingly common visitors to EDs, a trend expected to continue rising in the coming years.

According to the Spanish ICD Registry, published annually since 2005 by the Spanish Society of Cardiology (SEC), the 2022 report (Figure 1) documented 7,693 total implants (including first implants and replacements) — the highest number in the historical series, representing a 2.6% increase vs 2021 (7,499 units). Data from EUCOMED (European Confederation of Medical Suppliers Associations) showed 7,790 implants in 2022, also the highest figure recorded, with a 2.9% increase from 2021. The total implantation rate in 2022 was 168 implants per million inhabitants, up from 163 in 2021, 150 in 2020, and 157 in 2019. Despite this increase, the implantation rate in Spain remains well below the European average.[33]




| | 2013 | 2014 | 2015 | 2016 | 2017 | 2018 | 2019 | 2020 | 2021 | 2022 |
|---|---|---|---|---|---|---|---|---|---|---|
| Spanish ICD Registry | 102 | 106 | 118 | 122 | 135 | 137 | 146 | 149 | 158 | 162 |
| Eucomed | 120 | 128 | 137 | 143 | 138 | 152 | 157 | 150 | 163 | 168 |

Figure 1. Total number of implants recorded per million inhabitants and those estimated by the European Medical Technology Industry Association between 2013 and 2022. ICD: implantable cardioverter-defibrillator.
Adapted from Rev Esp Cardiol 2023; https://doi.org/10.1016/j.recesp.2023.06.018.

## Components of the ICD

An ICD consists of 2 main components: the pulse generator, and the lead(s) with stimulation/sensing and defibrillation electrodes (usually combined in one cable).

The generator contains the battery and all circuits needed for impulse delivery, signal processing, data storage, and energy discharge.

The battery life varies depending on the number of shocks and pacing frequency, typically lasting 5–8 years. Battery depletion is the most common reason for replacement. The battery, composed of lithium-silver and vanadium oxide, cannot deliver the required energy for defibrillation instantly, so the charge accumulates in a capacitor prior to discharge. The capacitor stores 30–45 joules, occupying a significant portion of the generator volume. Over the past decade, capacitor size has been reduced, and low-energy biphasic discharges have been introduced, enabling transvenous implantation similar to a conventional pacemaker, with the generator placed prepectorally on the anterior chest wall[34] (Figure 2) or, more recently, subcutaneously over the lower left ribs.[32] Modern ICDs weigh approximately 70 g with a volume of about 30 cc, a remarkable reduction from the 175 cc of early devices.[35]

The defibrillator leads transmit electrical signals from the endocardial surface of the right ventricular (RV) apex (placement in the RV septum is also an option) to the generator for analysis, and deliver pacing and shock pulses back to the heart. The defibrillation vector for shocks is currently established between coils located on the distal electrode tip of the RV lead and a 2nd ring electrode several millimeters behind the tip (i.e., a true bipolar lead). These bipolar leads provide highly accurate sensing, with narrow and high-amplitude electrograms. Some ICD leads use integrated bipolar sensing, meaning they consist of a single-tip electrode and a distal shock coil electrode—often positioned in the superior vena cava or even in the generator casing per se. True bipolar leads improve sensing capability and reduce the risk of external interference, which could otherwise cause device malfunction (e.g., inappropriate shocks triggered by muscle activity). Modern ICD systems use a coil electrode running along the ventricular lead as the main defibrillation electrode. Therefore, a single transvenous lead can perform all three functions: pacing, sensing, and defibrillation. Additional pacing electrodes improve defibrillation efficacy and reduce the defibrillation threshold. Electrodes must record a ventricular electrogram ≥ 5 mV, as signals must be large enough to detect VT and VF. In indicated cases, these devices may include atrial sensing and pacing via a second lead. Later developments introduced a third lead implanted in a branch of the coronary sinus (close to the left ventricle [LV]), to achieve cardiac resynchronization therapy (CRT) through biventricular pacing.[34,35]

The subcutaneous ICD (S-ICD) allows defibrillation (without anti-tachycardia pacing capability) without transvenous lead insertion.[32] The pulse generator is implanted in a subcutaneous pocket at the left mid-axillary thoracic position. The subcutaneous lead, containing an 8-cm shock coil electrode, is tunneled from the generator in the left axilla along the left parasternal margin (Figure 3). Although the S-ICD can detect VT/VF and deliver therapeutic shocks, it cannot provide an-tachycardia pacing (ATP) or bradycardia pacing. These devices may be suitable for patients requiring an ICD who have valid reasons to avoid permanent transvenous leads — for example, those with existing intracardiac leads or catheters, high systemic infection risk, or relatively young patients likely to undergo multiple future device replacements.

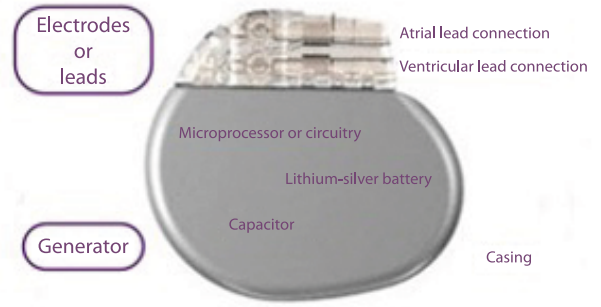



Figure 2. Components of the implantable cardioverter-defibrillator (ICD).
Original image. Cardiol 2023; https://doi.org/10.1016/j.recesp.2023.06.018.

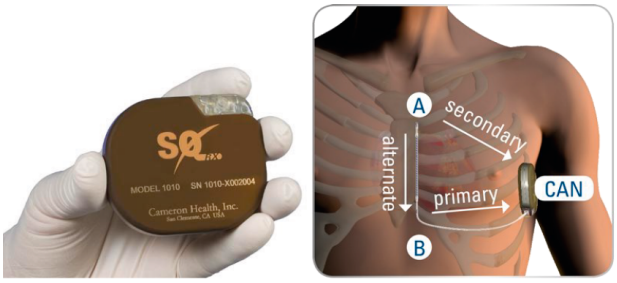



Figure 3. Subcutaneous implantable cardioverter-defibrillator (S-ICD).
Images courtesy of Boston Scientific.

Decision-making between device types should include discussion with the patient, explaining the lower complication risk of the S-ICD but also its larger size, shorter battery life, and inability to deliver pacing therapies, including ATP. The potential for inappropriate shocks must also be considered.

To overcome some of these limitations, the extravascular ICD was recently developed.[36] In a prospective, non-randomized clinical trial of 316 patients indicated for ICD implantation, defibrillation success rate was 98.7%, with 118 inappropriate shocks across 81 arrhythmic episodes. The extravascular ICD offers a lower risk of lead-related complications and pacing capability for anti-bradycardia and anti-tachycardia therapies. However, the high rate of inappropriate shocks noted by the authors, the need for specific surgical training, and the requirement for cardiac surgeon collaboration during early implants (as the lead traverses the substernal space, unfamiliar to many implanters) may limit its widespread adoption. This fact may limit the use of this device to centers with cardiac surgery availability, at least during the first years after its commercialization (Figure 4).

## Functions of the ICD

Since 1980, when the first human ICD implantation was performed,[30,31] the device has evolved far beyond its original defibrillation function. Over time, ICDs have incorporated synchronized electrical cardioversion, anti-bradycardia pacing (pacemaker function), anti-tachycardia pacing (ATP or overdrive pacing), and the ability to record and store arrhythmic episodes. Initially, arrhythmia detection relied solely on heart rate thresholds. Modern ICDs use additional programmable criteria to improve diagnostic specificity, reducing inappropriate therapy for non-ventricular tachycardias.

## Diagnosis of tachycardias

The device detects ventricular electrical activity through the RV electrode. Initially, the only parameter available to determine whether a patient was in tachycardia was VF, which meant that shocks were delivered whenever the ventricular rate exceeded the cutoff frequency, thereby treating any type of tachycardia regardless of its origin.[34] With the improvement of ICDs, the cutoff frequency has become programmable, and more sophisticated discrimination criteria have been introduced:

1) Sudden onset criterion: distinguishes sinus tachycardia (gradual onset) from VT (abrupt onset).[37]

2) Rate stability criterion: differentiates AF (variable R-R intervals) from VT (regular R-R intervals).[37-39]

3) Morphology-based criteria: compare the detected signal with that of sinus rhythm—similar morphology suggests supraventricular origin, while dissimilar morphology suggests ventricular origin.[40,41] In dual-chamber ICDs, additional discrimination criteria are available, such as the presence or absence of atrioventricular (AV) dissociation or differences in atrial and ventricular activation timing.[41,42] These criteria can be programmed both in their activation and in the differentiation parameters, which is of great importance: poor programming may result in a ventricular-origin tachycardia not being properly detected as such.

4) In defibrillators with both atrial and ventricular electrodes, additional criteria can also be activated based on the presence or absence of atrioventricular dissociation or on the timing of atrial activation relative to ventricular activation.[41,42]

## Antibradycardia pacing

Early ICDs lacked pacemaker functionality, leading to dangerous device interference in patients requiring both an ICD and a pacemaker. Modern ICDs incorporate fully functional pacing systems, eliminating this issue. Initially limited to single-chamber (VVI) pacing, today ICDs are available as single-, dual-, or triple-chamber systems. Anti-bradycardia pacing is programmed similarly to standard pacemakers, with the added capability to adjust post-shock pacing parameters to prevent capture loss from transient threshold elevation.

## Anti-tachycardia pacing (ATP)

Initially, ICDs only delivered electric shock discharges. Currently, they can administer sequences of pacing stimuli—programmable in both rate and number of beats—faster than the detected tachycardia, in order to attempt to terminate it without the need for shocks; this is known as ATP. Anti-tachycardia pacing, or overdrive pacing, consists of delivering short bursts (for example, eight beats) of rapid ventricular stimulation to terminate VT. Although there are various algorithms, ATP is programmed to be delivered

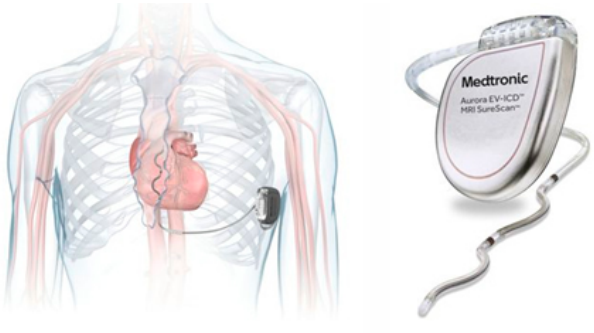



Figure 4. Extravascular implantable cardioverter-defibrillator (EV-ICD).
Images courtesy of Medtronic.

at a rate slightly faster (for example, with a cycle length 10–12% shorter) than that of the detected tachycardia. This therapeutic modality has proven to be very useful for treating many VTs, successfully restoring sinus rhythm in up to 90% of cases,[43-45] with the advantage that this therapy is painless and does not have the negative survival impact described in patients who experience repeated shocks.

### Cardioversion and defibrillation

Defibrillation is the fundamental function and the origin of the ICD's design, aimed at preventing sudden cardiac death due to VF. Since its inception, all efforts have focused on increasing its efficacy—that is, ensuring that the patient can always be defibrillated correctly and effectively. To this end, defibrillation systems have evolved (initially, patches were sutured directly onto the epicardium via thoracotomy, whereas the current defibrillation circuit consists of endocardial electrodes and the device's metallic casing), as have the direction of the delivered current and the waveform used (now biphasic, which is more effective than the older monophasic form).[46] With current ICDs, using a single RV lead, an active can, and biphasic shocks, successful defibrillation is achieved in the vast majority of patients (> 95%) with energies < 18 joules. This provides a safety margin of > 10 joules when shocks are programmed to the device's maximum deliverable energy. This ensures therapeutic efficacy, since in clinical practice the defibrillation threshold is not exact, and it is advisable to maintain a safety margin of several joules above the energy previously demonstrated to be effective.[47] Modern ICDs can also perform cardioversion for VT (by delivering shocks synchronized with the QRS complex) and even treat atrial arrhythmias (dual-chamber devices capable of atrial pacing or shock delivery), sometimes at the patient's own discretion (by holding a remote control device near the generator when, for example, an episode of AF does not subside).[48]

### Recording and storage of episodes

ICDs have memory that records the patient's events: they store all arrhythmic episodes and provide information on their date, duration, administered therapies, R-R intervals, and even electrograms (the equivalent of intracardiac ECG tracings during tachycardia). This makes it possible, for example, to determine whether an episode was due to a ventricular arrhythmia (as opposed to a supraventricular arrhythmia or interference) and helps optimize device programming to prevent inappropriate shocks for supraventricular rhythms.[49,50] Currently, remote monitoring capabilities via telephone or the Internet have been incorporated, allowing review of ICD parameters and events without requiring the patient to visit the clinic or hospital.

### Other functions

ICDs allow for an electrophysiological study to be performed without the need for catheter insertion: through the programmer, the heart can be paced or shocks delivered to induce arrhythmias and verify device efficacy. They also feature an alert function—a set of programmable alarms that emit audible tones in response to specific issues (such as excessive charge time, low battery, or pacing impedance outside the acceptable range) or to certain clinical situations (such as a tachycardia that required multiple therapies), prompting the patient to seek evaluation by an arrhythmia specialist. Finally, placing a magnet over the generator can deactivate ICD function when necessary (for example, in patients receiving inappropriate shocks due to uncontrolled rapid atrial fibrillation). The response to magnet application varies depending on the manufacturer, but in general, unlike pacemakers, the anti-bradycardia function remains unchanged.

## ICD Programming

Regardless of the reason for implantation, patients with an ICD—especially those with structural heart disease—may present with fast VT, slower VT, and VF.51 For this reason, up to 3 different detection zones with different rate thresholds can be programmed, along with distinct therapies in each zone. For example, in slow VT, ATP is programmed to minimize delivery of shocks (which are painful and impact quality of life in a conscious or even asymptomatic patient). If ATP fails, cardioversion shocks are programmed—initially at low energy and then at higher energy if necessary. To ensure treatment of sustained episodes, maximum-energy shocks are programmed in the VF zone from the outset. If a tachycardia crosses from one rate zone to another—spontaneously or because of a therapy—it will receive the therapies programmed for the new zone.

Programming also includes the time or number of beats a tachycardia must meet to be detected and treated, and—when shocks are planned—the time and number of beats after capacitor charge before shock delivery (redetection) to avoid treating episodes that have terminated spontaneously. The number of therapies per zone should be set (if the ICD exhausts therapies in a zone without success and the tachycardia persists, the device ends its intervention). In some arrhythmias, one may program detection only and no therapies (Holter function): the ICD will store episode data for analysis at the next follow-up. Additionally, based on patient characteristics, one programs the discrimination criteria mentioned earlier, electrogram storage vectors, alerts, and the device response to magnet application.

Finally, one programs the pacemaker function: if the patient is not pacemaker-dependent, VVI at a low rate is used to allow intrinsic rhythm whenever possible and avoid the detrimental effect that ventricular dyssynchrony (caused by RV apical pacing) may have on ventricular function[52]—though dual- and triple-chamber ICDs exist for AV-sequential pacing and ventricular resynchronization, respectively. All of this underscores the importance of ICD programming, which should be performed by specialists experienced in arrhythmia management and, above all, should be individualized to the patient's characteristics and to the arrhythmias they have had (secondary prevention) or those induced during implantation in patients with a primary-prevention indication.

## Indications for ICD implantation

The ICD is one of the main therapeutic options for patients who survive a cardiac arrest due to a ventricular arrhythmia or those considered at high risk of SCD. Technological development and evidence from successive clinical trials have led to an exponential expansion of indications.

Current indications are based on multicenter studies comparing ICDs with optimal conventional therapy or antiarrhythmic drug therapy, in both primary and secondary prevention of SCD. These have been compiled and updated in clinical practice guidelines such as those of the American Heart Association (AHA)[53,54] and the latest European Society of Cardiology (ESC) guidelines published in 2022.[28]

Although ICD recommendation is uniform for patients resuscitated from SCD due to VF or VT, and for patients with poorly tolerated VT plus structural heart disease, the indication in patients with well-tolerated VT and prior MI is controversial, especially when there is no severe ventricular dysfunction.

Situations in which ICD therapy is NOT indicated[54] include:

1. Ventricular tachyarrhythmias due to a reversible disorder in the absence of structural heart disease (e.g., electrolyte imbalance, drugs, trauma).
2. Life expectancy ≤ 1 year (Level of evidence I C).
3. Incessant VT/VF until the arrhythmic process is controlled (e.g., by catheter ablation).
4. Severe psychiatric illness likely to worsen with device implantation.
5. NYHA FC IV HF refractory to optimal medical therapy, ineligible for transplant or CRT.
6. Syncope without inducible ventricular tachyarrhythmias and no structural heart disease.
7. VF/VT amenable to surgical or catheter ablation in which SCD risk is minimized after successful ablation (e.g., atrial arrhythmias in WPW, outflow-tract VT [RV or LV], idiopathic or fascicular VT without structural disease).
8. Active infections or other acute medical problems—implantation should be deferred until resolution.

Major indications fall into 2 groups[53] (Table 1):

1. Secondary prevention of SCD in patients with prior sustained VT, VF, or resuscitated SCD due to VT/VF.
2. Primary prevention of SCD in patients at high risk of potentially lethal VT/VF.

### Secondary prevention

A meta-analysis of the earliest ICD trials[55-57] comparing ICDs with medical therapy for secondary prevention showed a 28% reduction in mortality, driven by fewer arrhythmic deaths in the ICD group.[58] Thus, ICD use for secondary prevention, in the absence of reversible causes, is widely accepted.

A key issue is all-cause mortality (not only arrhythmic or sudden death), since suppressing VT/VF may not improve overall mortality. Patient selection should consider both the known SCD risk from VT/VF for a given condition and the overall mortality risk from underlying comorbidities.

In schematic form, secondary-prevention ICD implantation is recommended[54] for:

1. Patients with a prior episode of resuscitated VF/VT or hemodynamically unstable sustained VT with no reversible cause, including a wide range of underlying heart diseases, idiopathic VT/VF, and congenital long QT syndrome—but not VT/VF limited to the first 48 hours after acute MI.
2. Patients with spontaneous sustained VT in the presence of heart disease (valvular, ischemic, hypertrophic, dilated, infiltrative cardiomyopathies, and channelopathies).

### Primary prevention

Some patients are at high risk of malignant ventricular arrhythmias and may warrant an ICD. Multiple randomized trials[59-62] show that patients with severe LV dysfunction (LVEF ≤ 35%) benefit most. Recently, two large registries (> 5,000 patients) confirmed mortality reduction.[63,64] However, the DANISH trial suggests that mortality benefit may be less evident in non-ischemic HF.[65]

In patients with prior MI and LV dysfunction, MADIT studies[59,60] demonstrated a marked mortality reduction with ICD vs conventional therapy—especially weeks after MI and with LVEF < 35% (≈ 54% 2-year mortality reduction). In MUSTT[61] (prior MI, NSVT on Holter, LVEF < 40%), ICD recipients had lower sudden and overall mortality.

Six years later, MADIT II (post-MI, LVEF ≤ 30%)[60] showed mortality reduction for patients with LVEF < 25% receiving an ICD vs optimal medical therapy, though DINAMIT did not confirm early post-MI benefit.[66] Together with a MADIT II subanalysis indicating greater benefit with later post-MI inclusion,[67] these data highlight the lack of primary-prevention benefit in the early weeks after MI.

In dilated cardiomyopathy, ICDs have not consistently improved survival vs medical therapy alone;[67-69] thus, ICDs are indicated for primary prevention in patients with structural heart disease, severe LV dysfunction, and HF.[62,70] If bundle-branch block with QRS > 120 ms is present, combining an ICD with CRT reduces overall mortality and all-cause hospitalizations vs medical therapy alone or CRT without defibrillation.

For hypertrophic cardiomyopathy, Brugada syndrome, and other channelopathies (notably long- and short-QT

syndromes), primary-prevention indications are controversial; follow the cited guidelines.[28,54]

In brief, primary-prevention ICD is recommended for potentially lethal VT/VF SCD risk in patients who, despite optimal medical therapy [beta-blockers, ACE inhibitors or ARBs, mineralocorticoid receptor antagonists, and SGLT2 inhibitors], remain at high risk:[28,54]

1. Prior MI (≥ 40 days before) with LVEF ≤ 30%.

2. Cardiomyopathy with NYHA FC II–III and LVEF ≤ 35%. Non-ischemic cardiomyopathy generally requires ≥ 3 months of optimal medical therapy with persistent LVEF ≤ 35%. As noted, DANISH[65] questions prophylactic ICD benefit in non-ischemic cases; ischemic patients should be reassessed ≥ 3 months after revascularization (surgery or stent).

## Cardiac resynchronization therapy (CRT)

CRT is indicated in congestive HF with QRS widening (> 120 ms), most often due to left bundle branch block (LBBB). CRT has been shown to improve symptoms, reduce hospitalizations, and lower mortality.[71,72]

### *Pathophysiology*

LBBB causes a temporal mismatch (asynchrony) in the contraction of both ventricles (interventricular asynchrony): the contraction of the left ventricle (LV) is delayed relative to that of the right ventricle (RV). In addition, intraventricular asynchrony occurs within the LV—the lateral wall contracts later than the interventricular septum. Likewise, atrioventricular asynchrony develops, as the atrium contracts partly during ventricular contraction.[73] The hemodynamic consequences of these contractility abnormalities are considerable, including a reduction in left ventricular ejection fraction (LVEF) and cardiac output, along with an increase in the degree of mitral regurgitation. These alterations are associated with clinical deterioration and a worsened medium- to long-term prognosis.

### *CRT devices*

CRT is achieved through simultaneous stimulation of both ventricles (biventricular pacing) by placing one electrode at the apex of the right ventricle (RV) and another in the left ventricle (LV), the latter positioned via cannulation of a tributary branch of the coronary sinus. Atrial sensing, essential to achieve physiological pacing, is performed through an electrode placed in the right atrial appendage (RAA). The system is completed with the pulse generator, which is implanted subcutaneously in the upper right chest area. Therefore, these devices are called triple-chamber systems, as electrodes are implanted in the right atrium (RA), right ventricle (RV), and coronary sinus (CS). In patients with atrial fibrillation (AF), only RV and CS leads are implanted. Finally, CRT can be combined with intracardiac defibrillation therapy (ICD) by incorporating coils into the RV lead for shock delivery.

### *Consequences of biventricular pacing*

When biventricular pacing is activated, the first observable effect is a narrowing of the QRS complex compared with the patient's baseline QRS and with the QRS produced by RV apical pacing. This finding is accompanied by an acute improvement in all hemodynamic parameters previously altered by ventricular asynchrony. This improvement is maintained over time and translates into better functional class, increased distance in the six-minute walk test, increased LVEF, a reduction in the number of hospital admissions, and a decrease in overall mortality.[74-76]

Furthermore, patients with depressed LVEF of any cause and HF are at high risk of sudden cardiac death due to ventricular arrhythmias.[77] For this reason, the combination of CRT and ICD therapy has been shown to improve survival in these patients.[78,79]

**Table 1.** Indications for implantable cardioverter-defibrillator implantation

| |
|---|
| **Secondary prevention** |
| Patients with a previous episode of resuscitated VF/VT or hemodynamically unstable sustained VT in the absence of reversible causes (Class IA). |
| Patients with recurrent sustained VT (not within 48 hours following myocardial infarction) with optimal medical therapy (OMT), normal LVEF, and reasonable life expectancy with good functional status > 1 year (Class IB in ESC; Class IIaC in AHA). |
| **Primary prevention** |
| Patients with a previous myocardial infarction more than 40 days old, with LVEF ≤ 35% in NYHA FC II–III after ≥ 3 months of OMT (Class IA). |
| Patients with a previous myocardial infarction > 40 days old, with LVEF ≤ 30% in NYHA FC I after ≥ 3 months of OMT (Class IA in ACC/AHA; Class IIaB in ESC). |
| Patients with coronary artery disease, LVEF ≤ 40%, and asymptomatic NSVT if VT can be induced during an electrophysiological study (Class IB in ACC/AHA; Class IIaB in ESC). |
| Patients with non-ischemic dilated cardiomyopathy with LVEF ≤ 35%, in NYHA FC II or III (Class IB). |
| Patients with non-ischemic dilated cardiomyopathy with LVEF ≤ 35%, in NYHA FC I (Class IIB, level of evidence C in ACC/AHA). |
| **Cardiac resynchronization therapy** |
| In heart failure with QRS widening (> 120 milliseconds) and left bundle branch block (LBBB), it is recommended to assess whether the patient could benefit from cardiac resynchronization therapy with an ICD (Class IC). |

VF: ventricular fibrillation; VT: ventricular tachycardia; OMT: optimal medical therapy; LVEF: left ventricular ejection fraction; NYHA: New York Heart Association; SMVT: sustained monomorphic ventricular tachycardia; NSVT: nonsustained ventricular tachycardia; ICD: implantable cardioverter-defibrillator.

### *Indications for CRT*

In patients with HF who are eligible for ICD implantation and who present with intraventricular conduction delay (≥ 120 milliseconds), CRT with a biventricular pacemaker is indicated. In these patients, implantation of a device with combined ICD (defibrillator) and biventricular pacing (CRT-D) functions should be performed.

In summary, CRT-D is indicated in patients with underlying disorders considered to carry a high risk of potentially fatal VT/VF, such as:

a. Patients with congenital long QT syndrome who have recurrent symptoms and/or torsades de pointes despite beta-blocker therapy, or other high-risk patients.

b. High-risk patients with hypertrophic cardiomyopathy or arrhythmogenic right ventricular cardiomyopathy.

c. High-risk patients with Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, and other channelopathies.

## Conclusions

The ICD is one of the main therapeutic options for patients who survive cardiac arrest due to ventricular arrhythmia or those considered to be at high risk of SCD. The technological development of ICDs—with the introduction of new devices as alternatives to conventional ICDs (such as subcutaneous and extravascular ICDs)—along with the growing evidence from successive clinical trials, has led to an exponential expansion in their indications.

As emergency physicians, we must be familiar with these innovative devices, as their potential complications may present in the emergency department. The management of these patients is not exclusive to electrophysiologists or cardiologists, but to all physicians involved in cardiovascular emergency care.

## ARTICLE INFORMATION

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