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EHRA EXPERT CONSENSUS

Management of patients receiving implantable cardiac defibrillator shocks

Recommendations for acute and long-term patient management

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Content

List of abbreviations

AF	Atrial fibrillation
ATP	Antitachycardia pacing
CRT	Cardiac resynchronization therapy
EHRA	European Heart Rhythm Association
ES	Electrical storm
HRS	Heart Rhythm Society
ICD	Implantable cardiac defibrillator
SCD	Sudden cardiac death
SSRI	Selective serotonin receptor inhibitor
VF	Ventricular fibrillation
VT	Ventricular tachycardia

Preamble

This expert consensus aims to provide guidance for the management of patients receiving one or multiple shocks from an ICD. The document expresses the view of a multidisciplinary group of experts in the fields of general adult cardiology, ICD treatment, invasive electrophysiology, and psychosomatic medicine. A variety of clinical settings, including emergency medicine, general cardiology, and interventional electrophysiology, are addressed as well as the different groups of clinicians involved in the care of these patients. To cover different levels of expertise in ICD treatment, it is intended to provide comprehensive information ranging from a basic explanation of how an ICD works to specialist advice for device programming.

Background

Almost 30 years after the first human implants, ICD therapy has become the treatment of choice in patients at risk of developing malignant ventricular arrhythmias.[1,2] Accordingly, the number of patients implanted with an ICD has increased substantially over the past decade,[3] mainly as a consequence of the expansion of ICD indications into the field of primary prevention of SCD in patients with decreased left ventricular function. In 2008, ~120 000 patients received an ICD worldwide.[3] As a result of the growing number of ICD patients, ICD-related problems are increasingly encountered and patients with one or multiple shocks are to day frequently seen at emergency departments, hospital wards, or ICD clinics. Therefore, personnel working in these environments should have specific knowledge concerning the management of ICD-related problems.

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Table 1 summarizes the most common causes of ICD shocks. Trials reported appropriate therapies in 17–64% of patients, whereas inappropriate shocks, most often caused by supraventricular tachycardia, occurred in 10–24%.[4] Among patients enrolled in primary prevention ICD trials, women experienced less appropriate ICD interventions than men.[5] Modern device algorithms facilitate effective termination of VT without shock delivery by ATP, discrimination between supraventricular tachycardia and VT, and prolonged detection windows. Therefore, we consider shocks delivered to terminate non-life-threatening or non-severely symptomatic arrhythmia as 'unnecessary'. Although patients generally report the number of perceived shocks with reasonably accuracy,[6] phantom shocks, a sensation of ICD therapies that cannot be confirmed by device interrogation, may occasionally occur.

Patients who received shock therapies should be subjected to a careful assessment of their clinical status and device function. ICD shocks are a powerful risk marker for subsequent clinical events. In patients with a primary preventive ICD indication, both appropriate and inappropriate shocks are associated with a marked increase in mortality, particularly due to death from progressive heart failure.[7,8] Possible explanations for this association include that arrhythmia on the ventricular and/or supraventricular level can be a harbinger of deteriorating heart failure, but it has also be proposed that defibrillator shocks cause cellular damage and exert negative inotropic effects, especially in patients receiving multiple therapies.[9–11]

Importantly, ICD shocks are often a very unpleasant experience and may cause acute and chronic psychological distress, anxiety,

and decreased quality of life.[12] Consequently, assessment and therapy to relieve distress and anxiety should be an integral part of treatment algorithms for post-shock ICD patients. In addition, early catheter ablation has recently been shown significant benefit in ICD recipients[13,14] and should be considered in shock patients.

The management of patients receiving ICD shocks is often complex and involves a variety of specialties and professions. Often, clinicians with no specialized training in the field feel uncomfortable taking responsibility for ICD patients. However, the increasing number of patients presenting after shock delivery makes it compulsory to ensure that appropriate treatment is delivered throughout the chain of care, in particular during the initial phase after shock delivery.

How the implantable cardiac defibrillator works

An ICD system consists of a pulse generator, usually implanted below the left or the right clavicle, and one to three thin electrodes placed transvenously into different chambers of the heart (*Figure 1*). One electrode is always inserted in the right ventricle (in case this being the only lead, the system is called a 'single-chamber ICD'). In many patients, a dual-chamber ICD with a right atrial and a right ventricular lead is implanted. Selected patients with heart failure may receive an additional left ventricular electrode placed through the coronary sinus in a posterolateral epicardial vein [CRT ICD (CRT-D)].

The ICD has all sensing and pacing capabilities of a conventional bradycardia pacemaker. In addition, it is designed to detect potentially harmful ventricular arrhythmias and to terminate these by either ATP or shock treatment. The defibrillation lead contains a coil at the level of the right ventricle and, optionally, another one in the superior vena cava (called the proximal coil). For shock treatment, electrical current (energy ranging from <1 to 42 J) is delivered between the ventricular defibrillation coil, the device can, and the proximal defibrillation coil.

The detection windows for VT and VF as well as the sequence of treatment (ATP, low-energy shock, high-energy shock) can be individually programmed. Software algorithms enable the discrimination between atrial and ventricular arrhythmias. However, inappropriate shocks still occur (*Table 1*).

Current generation ICDs store information from various diagnostic features including intracardiac ECG registrations during arrhythmia and can transmit these data using remote monitoring technology. Furthermore, the device can generate audible alarms in the case of device malfunction, low battery capacity, and/or lead failure.

Patient management in the acute setting

Implantable cardiac defibrillator shocks may be related to a variety of conditions (*Table 1*); thus, clinical presentations range from mild discomfort to severe haemodynamic compromise. The shock experience itself, varying considerably between individuals from

| **Table 1 Causes of ICD shock delivery** |
| --- |
| Appropriate shocks |
| VF |
| Monomorphic VT |
| Polymorphic VT |
| Unnecessary shocks |
| Haemodynamically tolerated non sustained VT |
| Haemodynamically tolerated VT sensitive for ATP |
| Inappropriate shocks |
| Supraventricular tachycardia |
| Atrial fibrillation |
| Atrial flutter |
| Atrial tachycardia |
| AVNRT/AVRT |
| Sinus tachycardia |
| Signal misinterpretation |
| Frequent PVC |
| T-wave oversensing |
| Atrial far-field sensing |
| Diaphragmatic myopotentials |
| R-wave double-counting |
| Lead failure, insulation brake |
| Electromagnetic interference |
| Phantom shocks |

Modified from Gehi *et al.*[109]
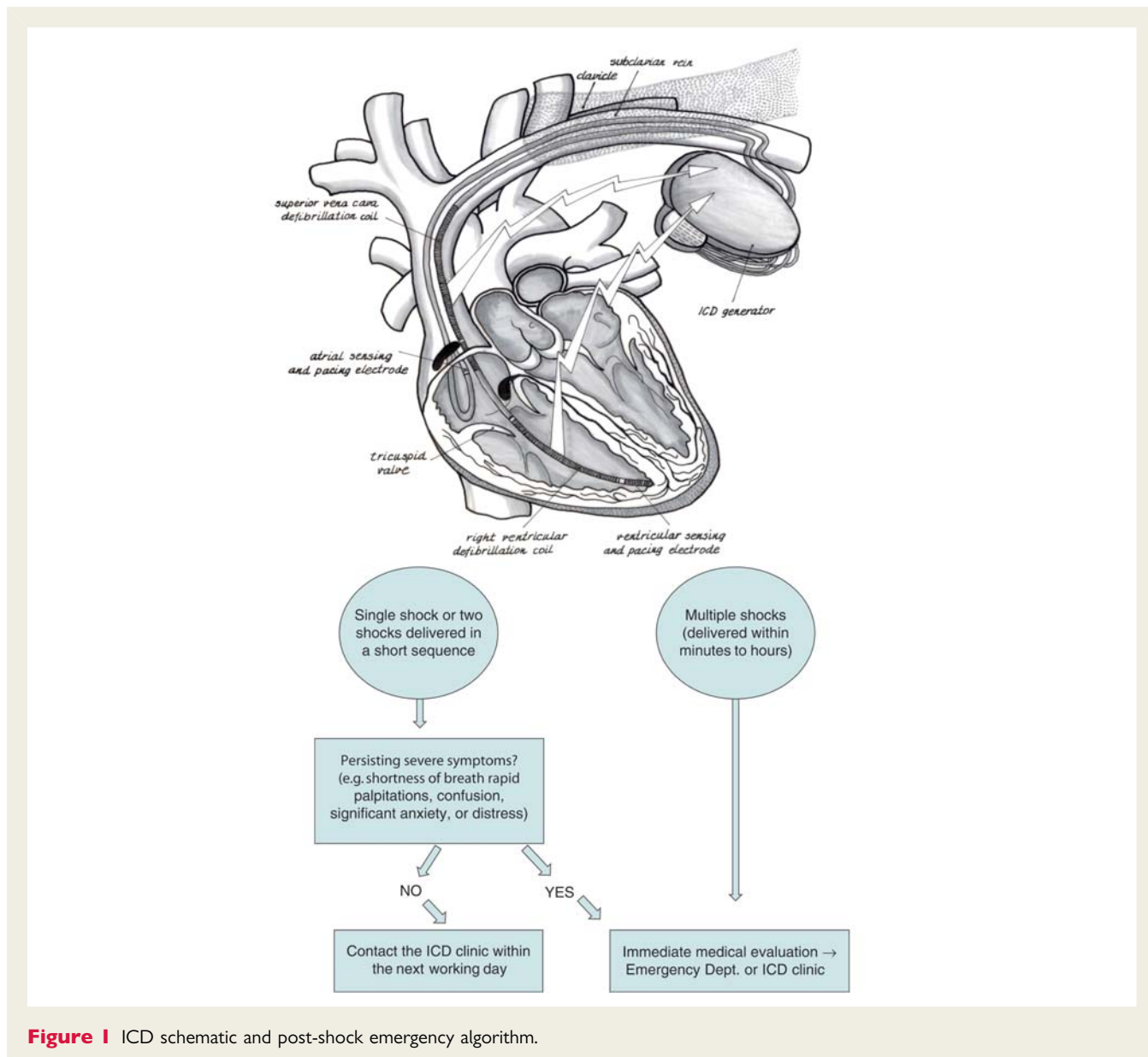
Figure 1 ICD schematic and post-shock emergency algorithm.

being 'hardly noticeable' to being 'hit with a bat' or 'stack with a knife', has been rated on average a '6' on a 0–10 pain scale.[6,15] The shock experience, the underlying cardiac conditions, or both are often associated with significant symptoms, prompting the patient to seek acute medical support by calling emergency services, going to a hospital emergency department or calling the responsible ICD centre.[6] Today, device-related information can also be checked using remote home monitoring systems.[16]

Owing to the possible underlying clinical instability and the potential severity of symptoms, we recommend that patients who experience one or more ICD shocks are evaluated by a clinical expert in due course. This should include a general clinical assessment of the patient and an interrogation of the device in order to establish the cause and appropriateness of therapy delivery. This section summarizes current recommendations regarding acute post-shock management of ICD patients.

(1) Out-of hospital setting

After experiencing a device shock, the patient or his kin need to establish appropriate contact to a healthcare provider (*Figure 1*). Detailed instructions should be given early after device implantation, preferably prior to hospital discharge.

- *Single shock or two shocks delivered within a short sequence (seconds to minutes).*
  - In the absence of persisting severe symptoms (e.g. chest pain, shortness of breath, rapid palpitations, confusion, significant anxiety, or distress), the ICD clinic (i.e. the healthcare provider responsible for device follow-up) should be contacted within the next working day to initiate device interrogation (in office or remote) and appropriate clinical assessment.
  - In the presence of persisting severe symptoms (e.g. chest pain, shortness of breath, rapid palpitations, confusion, significant anxiety, or distress), there is a need for immediate

medical evaluation through a hospital Emergency Department or the ICD clinic.

- **Multiple shocks (within minutes or hours)**

  Need for immediate medical evaluation, usually through a hospital Emergency Department or immediate contact with the ICD clinic (if possible).

(2) Assessment in the Emergency Department or ambulance setting

- **Cardiac arrest:**

  Deliver advanced cardiac life support according to guidelines, independent of the fact that the patient has an ICD implanted.

- **ECG:**

  Establish continuous ECG monitoring as soon as possible. Try to record a 12-lead ECG during tachycardia and normal rhythm, which provides crucial information for further assessment of the arrhythmia.

- **Clinical assessment:**

  Assessment of patient's history (underlying heart disease, primary/secondary prevention indication, history of previous shocks, drug therapy, etc.) haemodynamics (blood pressure, signs of low cardiac output) and ischaemia. Echocardiography or another imaging technique of cardiac function should be performed as soon as possible. Ask for patient symptoms preceding or following the shock (syncope, dizziness palpitations, chest pain, dyspnoea, etc.) and situational circumstances (rest, exercise, arm movement, potential exposure to electromagnetic interference, etc.).

- **Contact the ICD clinic:**

  A clear algorithm should be in place in the emergency department regarding how to establish contact with the ICD clinic.

- **Ongoing tachyarrhythmia (ventricular or atrial) with haemodynamic compromise:**

  Treat arrhythmia independently of the presence of the ICD. Consider external DC shock, iv administration of amiodarone and /or β-blockers (if haemodynamically tolerated). For external DC shock, avoid placement of paddles in the skin area over the ICD pocket. When possible, attempt an anterior–posterior electrode position.

- **Tachyarrhythmia (ventricular or atrial) without haemodynamic compromise:**

  Treat the arrhythmia with drugs. Consider consulting the ICD specialist for ICD reprogramming and/or delivery of ATP.

- **Repetitive ICD shocks in the absence of tachyarrhythmia or due to tachyarrhythmia (atrial or ventricular) that is haemodynamically well tolerated by the patient:**

  Place a **magnet** over the device to inhibit further shock delivery (*Figure 2*). For this purpose, the pacemaker manufacturers provide special magnets of sufficient size that should be readily available in ambulance cars, emergency departments, and cardiology wards. These magnets can be used in an emergency setting regardless of the device type or the manufacturer. Magnet placement temporarily disables tachyarrhythmia therapies by affecting

Figure 2 Placement of magnet. A pacemaker magnet is placed and fixated over the ICD device. During magnet placement, tachycardia treatments are inhibited while pacemaker functions are preserved. See also *Table 2*.

a reed switch in the device circuit. If constant inhibition of tachyarrhythmia therapies is desired, the magnet should be secured with tape. During magnet mode, it is mandatory to maintain continuous ECG monitoring in order to detect potentially life-threatening ventricular tachyarrhythmias. In contrast to pacemakers, magnet application over an ICD does not change the mode of pacing. A detailed review of the responses of currently available ICDs to a magnet is reported in *Table 2*. With very few exceptions, the ICD will resume the ability to deliver therapy for ventricular tachyarrhythmias as soon as the magnet is removed (*Table 2*).

- **Repetitive delivery of ICD shocks due to ineffective ICD therapies or immediate tachyarrhythmia re-initiation after shock:**

  See the section 'Electrical storm'. Institute appropriate drug therapy (sedatives, β-blockers, amiodarone). Consider deep sedation or general anaesthesia. If haemodynamically well tolerated, consider magnet placement to prevent further shock delivery. Consult ICD specialist concerning ICD reprogramming and further treatment.

- **Laboratory assessments:**

  Electrolytes (potassium, magnesium) should always be checked and imbalances should be corrected. Tests for myocardial ischaemia, heart failure (BNP), renal function, respiratory insufficiency, pH, drug intoxication (e.g. digoxin, barbiturates, etc.),

Table 2 Response to magnet of currently available ICDs from various manufacturers

Manufacturer	Response to magnet		
	Tachyarrhythmia therapies	Bradycardia therapies	Confirmation
Boston (Guidant/CPI)	Inhibition of detection and therapy delivery for VT/VF, lasting as long as the magnet is positioned over the ICD; if the 'change tachy mode with magnet' function is enabled (currently not a default programming), magnet application for more than 30 s results in deactivation of the device (the ICD remains inactive when the magnet is removed)	As programmed	R-wave synchronous beeping tones are emitted by the device if it is active, whereas a continuous tone is emitted if the device is inactive (these functions are currently the default programming)
Medtronic	Transient inhibition of detection and therapy delivery for VT/VF, lasting as long as the magnet is positioned over the ICD	As programmed	No confirmation
St Jude Medical	Transient inhibition of detection and therapy delivery for VT/VF, lasting as long as the magnet is positioned over the ICD	As programmed	No confirmation
Sorin	Transient inhibition of detection and therapy delivery for VT/VF, lasting as long as the magnet is positioned over the ICD	Pacing at magnet rate in the programmed pacing mode (VVI, DDD, DDI)	No confirmation
Biotronik	Transient inhibition of detection and therapy delivery for VT/VF, lasting as long as the magnet is positioned over the ICD	As programmed	No confirmation

Modified from Pinski.[110]

or drug addiction (e.g. cocaine) should be performed if clinically indicated.

- **Skin contact with the patient during ICD discharge:**

  This involves no immediate danger; however, the use of gloves (single or double) decreases conductivity and attenuates potential discomfort.[17]

- **Audible device alert:**

  Ask if the patient recently perceived audible alerts from the device possibly indicating battery exhaustion, abnormalities in lead impedance, or other device-related or clinical problems.

(3) Device-related recommendations in the acute setting

- **Programmer:**

  Ensure ECG monitoring and device interrogation with real-time telemetry. If unknown, the ICD device type can be identified on the patient identification card in order to select the specific device programmer.

- **Device interrogation:**

  - Analyse ICD detections, delivered therapy reports, and stored electrograms in order to assess the appropriateness of therapies.
  - Re-evaluate tachyarrhythmia settings (detection zones, programmed therapies)
  - Pay particular attention to warnings for low battery voltage, device life time, charging time of the capacitors, and shock impedance (normal value: 20–100 Ω).

- Check shock impedance and its change compared with previous assessments.
- Assess pacing-lead impedance (normal value: 200–1000 Ω). A significant increase may indicate micro-/macro-lead dislodgment, lead fracture, or altered connection to pulse generator or adapters, fibrosis at lead-myocardial interface, change in myocardial substrate (infarct in the area surrounding lead tip), or adverse pharmacological effects; a significant decrease in pacing impedance suggests lead or adaptor insulation failure.
- Assess capture thresholds and sensing of R and P-waves in comparison to previous assessments.
- If oversensing due to lead or adaptor failure or myopotentials is suspected, consider provocative manoeuvres (adduction, abduction of the arm, Valsalva manoeuvre, manipulation of the pocket, etc.) during real-time telemetry of near- and far-field electrograms with marker channels, after inactivation of therapy delivery.
- If lead dislodgement, altered connection of lead(s) or adaptor(s) to the device, insulation defect due to subclavian crush syndrome, or Twiddler's syndrome is suspected a chest radiography should be performed.

(4) Assessment of acute stress

Receiving one or multiple shocks can cause acute stress reactions. These can potentially lower the threshold for repeated arrhythmias by causing an imbalance in sympathetic and parasympathetic tone and, in a subgroup of patients, lead to anxiety disorders, post-traumatic stress, depression, and other forms of psychological maladaptation. Psychological reactions to the event should therefore be assessed in the acute setting:

- Ask patients for his/her perception and interpretation of the shock. Misconceptions sometimes occur and can impair psychological adjustment.
- Ask for feelings of anxiety, helplessness, or panic.
- Ask for sources of support in the family, friendship, etc., as well as in the medical system (support from general practitioner, ongoing psychotherapy, etc.).

If relevant distress is encountered, basic interventions should be offered in order to stabilize the patient. These can include:

- Provision of reassurance by empathetic listening, correction of misbeliefs about the shock and/or arrhythmia, or help with calling friends or relatives for emotional support. If patients feel traumatized by the shock, physical and emotional security is their most urgent need and can help prevent the development of severe post-traumatic stress disorders.
- Benzodiazepines may be used for reducing acute anxiety, in particular if distress is considered relevant for triggering repeated arrhythmias.[18]
- If needed, consultation of a mental health expert (Psychosomatic Medicine, Psychology, or Psychiatry) for assistance in the judgement of the acute distress and the need for acute treatment.

Electrical storm

Electrical storm is defined as the occurrence of three or more distinct episodes of VT and/or VF within a 24-h period, either resulting in a device intervention or monitored as a sustained VT ($\geq$ 30 s).[18–20] Some authors have set an arbitrary 5 min interval between VT/VF episodes to define ES.[20] Treatment recommendations for repetitive ICD shocks in the absence of tachyarrhythmia, e.g. due to device malfunction, are provided in the previous section.

Incidence, triggers, and substrates

Electrical storm has been reported in 10–40% of patients in secondary prevention,[19,21–23] whereas the incidence of ES is lower in primary prevention.[24] Electrical storm results from a complex interplay between arrhythmogenic substrates and acute perturbations in autonomic tone and cellular milieu.[25] No independent predictors have been reproducibly identified.[21,22] Potential triggering factors include modification of or non-compliance to drug therapy, worsening of heart failure, early postoperative period, emotional stress and anger, alcohol excess, electrolyte abnormalities, and myocardial ischaemia. However, most cases of ES occur without any apparent cause.[19,22,23,26] It is predominantly caused (more than 80%) by episodes of monomorphic VT.[19–23] Nevertheless, it can also occur due to polymorphic VT/VF, especially in the context of myocardial ischaemia. Knowledge of the underlying type of arrhythmia is important for the selection of management strategies.

Management

Reversible cause?

Whenever a reversible cause can be identified such as drug side effects, electrolyte disturbances, or myocardial ischaemia, measures should be undertaken for its rapid correction.

Drugs

- A reduction in sympathetic tone by β-blockers and sedation are essential as increased sympathetic activity plays a key role in the genesis of ES.[27] When imminent haemodynamic instability is a threat, intravenous administration of short acting agents, such as esmolol, may be considered.
- Class III antiarrhythmic agents such as intravenous amiodarone[28] or sotalol[29] can be effective. Amiodarone combined with a β-blocker appears to be more effective than sotalol or β-blocker alone.[25] Class I antiarrhythmic drugs should generally be avoided. However, procainamide, flecainide, and quinidine have been used successfully in some ICD patients. Lidocaine could have some beneficial effect, especially if ES is associated with acute ischaemia. The combination of antiarrhythmic agents with different mechanisms of action (e.g. amiodarone and lidocaine) may prove effective in some cases. Some genetic disorders may require specific treatment. For example, in the Brugada syndrome, quinidine or isoproterenol may terminate incessant arrhythmias. More detailed information on the treatment of ventricular arrhythmia is provided in the specific guidelines.[2]

Importantly, the use of most antiarrhythmic drugs (especially amiodarone and procainamide) may cause an increased cycle length of the arrhythmia. As a result, the rate of the arrhythmia may drop below the detection cut-off, thus requiring adjustments of the device settings.

Polymorphic ventricular tachycardia

In polymorphic VTs such as torsades de pointes, intravenous administration of magnesium sulphate, potassium, and overdrive pacing (e.g. at 90 bpm or more) may be effective in suppressing the re-initiation of polymorphic VT. In ES patients with inherited or drug-induced long QT syndrome, isoproterenol may prevent recurrent episodes of arrhythmia. β-Blockers and revascularization are indicated in polymorphic VT triggered by myocardial ischaemia.

Catheter ablation

Catheter ablation has developed into a successful treatment strategy for ES with acceptable safety.[30] Although it is predominantly used for recurrent or incessant monomorphic VTs, it can also be considered in polymorphic VT/VF triggered by ectopic foci[31,32] (see also section below).

Anaesthesia and other measures

If the above strategies fail, general anaesthesia or thoracic epidural anaesthesia can be considered.[33,34] Mechanical circulatory support can be an alternative as a bridge to cardiac transplant or temporarily for stabilization and subsequent catheter ablation.[35]

General measures

The general assessment of an ICD patient after shock(s) always includes a thorough evaluation of underlying heart disease, in particular the assessment of myocardial ischaemia and heart failure. Potential triggering factors should be identified, such as electrolyte derangements, medications prolonging the QT interval, and/or potential intercurrent illnesses, e.g. infection that may provoke increased arrhythmic activity. The possibility of drug non-compliance or recent medication changes should be considered. For example, electrolyte abnormalities secondary to the use of diuretics or to angiotensin converting enzyme inhibitors are not uncommon. This is particularly relevant to heart failure patients, a substantial proportion of which have concomitant renal failure.

Ischaemia

As ischaemic heart disease is the principal diagnosis in the majority of ICD patients, it is essential to recognize the possibility of myocardial ischaemia as a trigger of ventricular arrhythmias.[36–38] Since patients may have atypical symptoms and the 12-lead ECG may not be reliable (especially immediately after an ICD shock or in patients who are paced), acute coronary syndromes need to be excluded by means of cardiac enzyme measurements.

However, it should be noted that troponin level elevations are commonly observed after episodes of sustained VT, in cardiac arrest survivors and after ICD shocks. In many cases, such biomarker increase rather reflects global myocardial oxygen deprivation during arrhythmia than a local obstruction of coronary flow. Therefore, the diagnosis of 'myocardial infarction' with a concomitant ventricular arrhythmia should be made only after a careful diagnostic work-up. Coronary angiography (in comparison with previous findings, if available) may be necessary in selected patients. If an acute coronary event is ruled out, exercise stress testing in combination with appropriate cardiac imaging should be performed.

If ischaemia is considered to play a role, revascularization may be necessary and has been demonstrated to reduce arrhythmia recurrences.[38] The CABG-Patch trial showed a low incidence of ventricular arrhythmias requiring a defibrillator shock, in patients with decreased systolic function and a substrate for VT, subjected to CABG. This may, in part, depend on a protective effect of revascularization with suppression of ischaemia-mediated VT.[39] However, in other studies, post-MI patients with previous VT/VF remained at high risk for VT/VF even after revascularization.[40,41] Ventricular tachycardia substrates originating from border zones between scar tissue and normal myocardium may be refractory to pharmacological treatment and are often the source of frequent ICD shocks or ES. In these cases, catheter ablation may be indicated[42] (see the section below).

Heart failure

The majority of ICD patients suffer from chronic heart failure and deteriorating heart failure may trigger supraventricular and ventricular tachyarrhythmia. In primary prevention patients, both appropriate and inappropriate ICD shocks are associated with an increased risk for death, in particular death from heart failure.[7,8,43] Therefore, device shocks should raise attention as to

the possibility of disease progression and a careful assessment of the patient's heart failure status and optimization of therapy, in particular with regard to β-blockers, should be performed.[36,43]

In ICD patients without CRT, it is important to avoid unnecessary RV pacing,[44,45] whereas the percentage of biventricular stimulation should be as high as possible in those with CRT-D. If poor rate control during AF causes insufficient CRT delivery, AV-junctional ablation should be considered.[46] Although not yet supported by clinical outcome trials, optimization of programmable pacemaker parameters should be suggested in patients with refractory symptoms of heart failure after CRT implantation. Heart failure management may also be improved by using the diagnostic information from device-based diagnostic features such as monitoring of intrathoracic impedance, heart rate, heart rate variability, and physical activity.[47]

Driving

Once a patient has received one or more device shocks, it is important to provide adequate advise about the pertaining driving regulations. A newly published EHRA consensus statement provides guidance in this area.[48] Notably, there may also be national guidelines that apply. Usually, a delivered ICD shock implicates that the patient is excluded from driving a car for a period and/or may be permanently excluded for driving certain vehicles according to the national laws. Owing to legal aspects, it is mandatory to make a note in the patient's record about the information provided.

Treatment termination

In an end-of-life situation, ICD therapies can be deactivated equivalent to a 'do not resuscitate order' taking individual circumstances with regard to the quality-of-life-impact by the device, the expected survival and the futility of therapy into consideration. The decision may differentiate between deactivation of shock therapies and ATP algorithms. In a joint consensus document, EHRA together with the HRS provide helpful direction in the field.[49] A European perspective to this statement will follow shortly. Further advice can be found in the literature.[50–52] It is prudent that patients and kin are informed about the possibility of discontinuing ICD therapies early on in the course of treatment to elaborate a competent view on this complex subject.

Drug therapy

Chronic treatment with various drugs, 'antiarrhythmics' and others, can prevent ICD shocks by suppressing ventricular tachyarrhythmia or slowing rapid VTs to make them amenable to successful ATP. However, it remains to be proven whether the prevention of ICD shocks by drug therapy also improves the prognosis of ICD patients.

β-Blockers

β-Blockers prolong life in patients with heart failure and survivors of myocardial infarction. Furthermore, they convey some protection against sudden death[53] and their proarrhythmic potential is low. Therefore, β-blockers are the first-line choice to reduce episodes of atrial and ventricular arrhythmias or to slow the

ventricular rate during supraventricular arrhythmias in ICD patients. Despite this, the effectiveness of β-blockade to reduce SCD in ICD patients has been disputed[54,55] and many patients will still experience shocks necessitating the prescription of additional antiarrhythmic agents.

Amiodarone

According to a recent meta-analysis in patients without ICD, amiodarone decreases the risk of SCD and cardiovascular death by 29 and 18%, respectively.[56] However, amiodarone has no effect on all-cause mortality and is associated with a two- and a five-fold increased risk of pulmonary and thyroid toxicity.

Observational studies and randomized clinical trials showed that amiodarone is effective in reducing the number of ICD shocks by a variable extent.[57,58] A combination with a β-blocking agent proved more effective than β-blocker alone or compared with sotalol.[58] However, these positive effects should be balanced against the increased risk of drug-related adverse effects. In addition, the possibility of an increase in the defibrillation threshold under amiodarone has to be kept in mind. However, data from modern biphasic endovascular devices do not suggest a clinical relevance of this potential adverse effect.[59]

Sotalol

In a randomized multicentre trial,[29] sotalol significantly reduced the combined endpoint of all-cause mortality or first appropriate ICD shock by 44%. These findings were corroborated in a smaller prospective study in patients with ICD for secondary prevention.[59] In the OPTIC study, sotalol tended to reduce shocks compared with β-blockers but was significantly inferior to amiodarone. Thus, sotalol is a valid option for ICD shock prevention and can be considered on an individual patient basis, in particular if amiodarone is contraindicated.

In selected cases, also the use of class IA or IC drugs may be justified and special recommendations may apply for patients with genetic disorders such as the Brugada syndrome. Detailed advice concerning the long-term drug treatment of ventricular arrhythmia has been published elsewhere.[2]

Upstream therapy

Apart from specific antiarrhythmics, other drugs can contribute to lower the risk for ventricular tachyarrhythmia and ICD shocks. Angiotensin converting enzyme inhibitors given in appropriate doses[60] and statins[61] can reduce the atrial and the ventricular arrhythmia burden as shown in retrospective trial analyses. The role of statins has also been assessed in ICD patient cohorts from MADIT-II and DEFINITE,[62,63] confirming a significant decrease in unnecessary ICD therapies associated with statin use in patients with either ischaemic or nonischaemic heart failure aetiology. Aldosterone blockers have shown to be effective in the primary prevention of SCD in high-risk individuals with systolic left ventricular dysfunction[64,65] and may be effective as an adjunct to ICDs and/or CRT, both in the primary and the secondary prevention of SCD.[66,67]

Device programming

Implantable cardiac defibrillators can terminate ventricular tachyarrhythmias either by ATP or delivery of electrical shocks.[7,68,69] Antitachycardia pacing is preferred if possible, and all efforts should be made to avert inappropriate shock therapies because they cause patient discomfort are potentially proarrhythmic and decrease battery life. In fact, clinical studies confirmed that ATP and strategic ICD programming are effective and safe in reducing the number of shocks and improving the quality of life.[70,71] This section provides recommendations for the programming of ICD devices aiming to reduce the number of unnecessary and inappropriate shock therapies. It is important to point out that programming of devices always needs to be adjusted to the individual patient.

Minimizing unnecessary shocks

Ventricular tachycardia and ventricular fibrillation detection zones

Up to three ventricular rate-detection zones can be programmed to allow delivery of different ICD therapies depending on the rate of ventricular tachyarrhythmia. In patients with structural heart disease, recent studies have shown that it is useful to set upper boundary for slower VT around 188 bpm (320 ms), zone for fast VT between 188 and 250 bpm (320–240 ms), and VF zone from 200 bpm on.[70–72] These studies demonstrated high effectiveness of ATP in terminating both slow and fast VTs. There is solid evidence that ATP is equally effective in termination of fast VTs in secondary as well as in primary prevention patients. In primary prevention, programming a slow VT monitor zone can be helpful to detect other than fast ventricular tachyarrhythmias. In patients without structural heart disease (e.g. patients with genetic disorders), VT zones should be adapted to the expected higher heart rates reached during exercise, in particular in younger patients.

Antitachycardia pacing

Optimization of ATP is the most effective strategy to reduce appropriate shocks.[70,73] In the Pain-free-RX II study,[70] a VT zone from 188 to 250 bpm was programmed and patients were randomized to receive one sequence of eight ATP pulses at 88% of the tachycardia cycle length followed by shock therapy, if necessary, or an immediate shock. In the ATP group, 81% of fast VTs could be terminated successfully by ATP only. This reduced defibrillator shocks and translated into a significantly better quality of life. These findings were further corroborated by the results of other studies.[71,72,74]

ATP interventions are based on trains of pacing impulses with an equal (burst) or decrementing (ramp) interstimulus interval. Studies investigating the relative efficacy of these two ATP patterns showed similar effects regarding VT termination.[75,76] However, in fast VTs (cycle length <300 ms), burst pacing is more likely to terminate the tachycardia than ramp pacing.[72] ATP efficacy also depends on a sufficient number of attempts and duration of pacing trains that can be programmed according to the patient's haemodynamic tolerance during VT episodes. The underlying left ventricular function is an important parameter in this respect.[77] In patients with fast VT, a maximum of two ATP attempts is
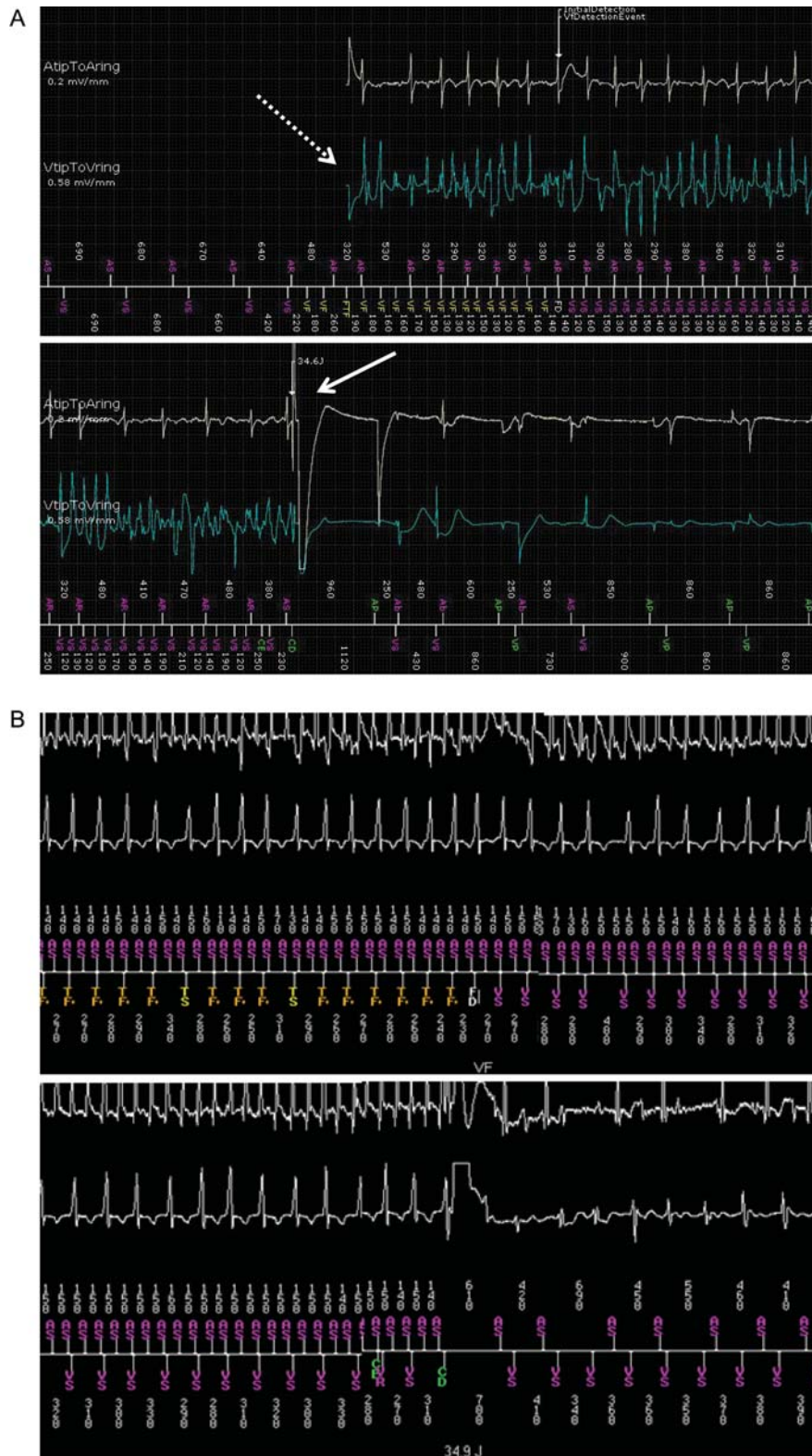
Figure 3 (*A*) Appropriate ICD discharge. Fast VT is detected and a successful shock delivered (arrow). (*B*) Inappropriate ICD discharge. Fast ventricular rate caused by AF is detected in the VT zone (see marker 'Ts') triggering an inappropriate shock delivery. The device has only a ventricular lead making the discrimination between AF and VT difficult. Irregular and short cycle lengths are typical for fast conducted AF (see numbers below annotations). Atrial fibrillation persists, however, with a transiently slower AV conduction. (*C*) T-wave oversensing. Inappropriate shock caused by T-wave oversensing. In the marker channel (on the top), correct sensing of QRS complexes (Vs) is followed by sensing of T-waves (VF). Ventricular signals have a higher amplitude compared with smaller T-wave signals (lower channel). (*D*) Artefacts, lead fracture. Inappropriate shock therapy in a patient with lead fracture. Oversensing of noise in the intracardiac electrograms (arrow). In the marker channel (bottom), non-physiological short intervals (<200 ms) are registered (see numbers below annotations).
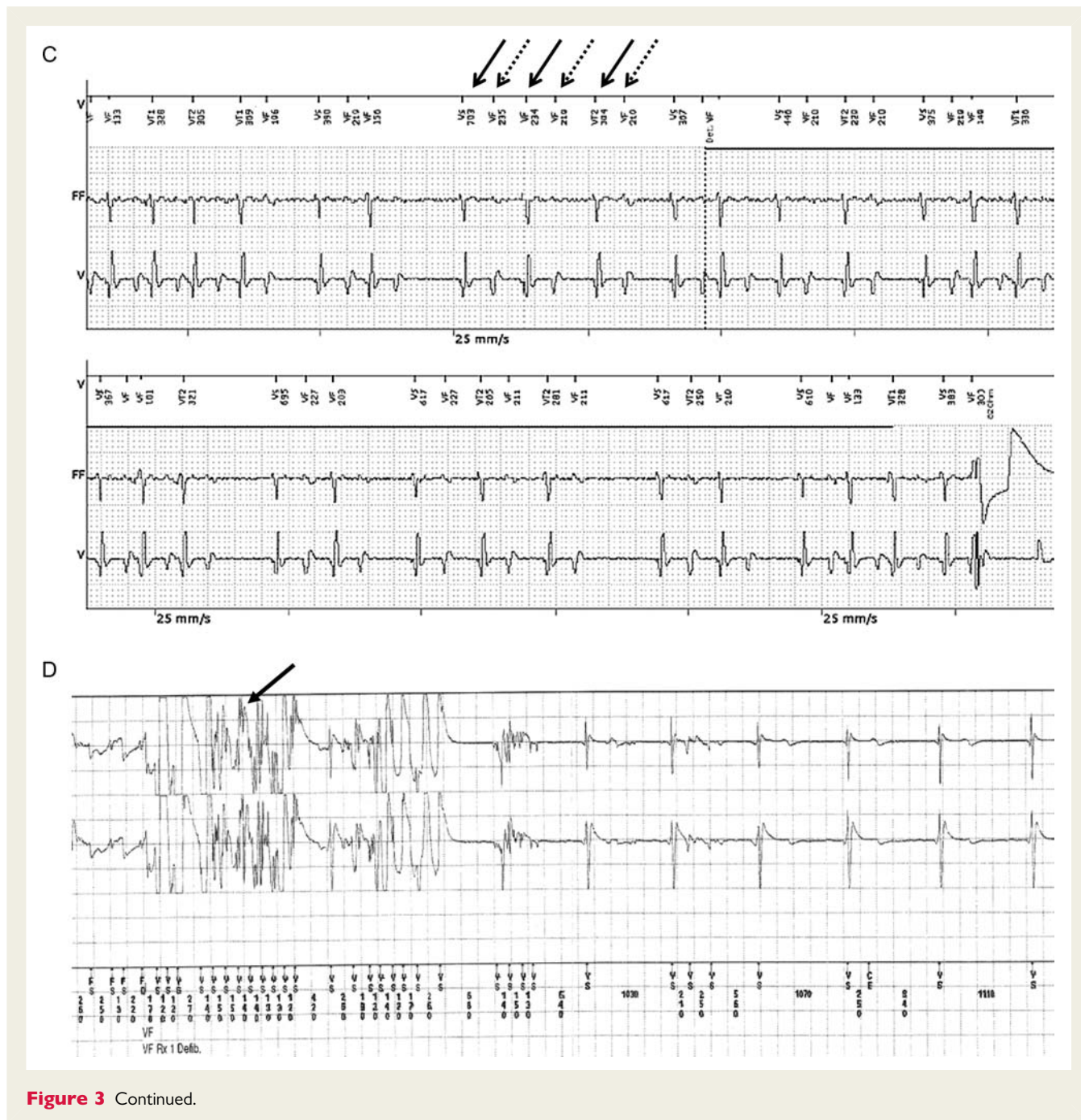
Figure 3 Continued.

commonly recommended. Each sequence typically consists of 8 beats; however, 15 beats were documented equally effective and even preferable in the subgroup of secondary prevention subjects with preserved left ventricular ejection fraction.[78] Strategic ATP programming can be performed empirically with preset parameters and such approach is not inferior to tailored ATP programming.[72]

Time to detection and therapy

Studies also showed that it is important to increase the time to detection and therapy to allow spontaneous termination of non-

sustained ventricular arrhythmias. It may vary from 16/18 to 30/40 beats without compromising safety.[71,79] Painless termination of fast VTs by ATP is associated with a significant improvement in quality of life. Still, in patients with haemodynamically compromising VTs, immediate 'shock therapy' may be required to terminate the arrhythmia quickly.

Minimizing inappropriate shocks

There are two main causes of inappropriate shocks: supraventricular arrhythmias and device dysfunction. Inappropriate therapy delivery is most frequently triggered by atrial arrhythmias, usually

AF (*Figure 3*B), resulting in high ventricular rates. This may be prevented by adequate rate control therapy with AV-nodal slowing agents, usually β-blockers or (in patients without heart failure) non-dihydropyridine calcium channel blockers in combination with digitalis glycosides. In refractory patients, amiodarone or dronedarone can be effective in lowering the ventricular response rate. Especially in patients receiving CRT, ablation of the AV-nodal conduction should be considered. In some patients, rhythm control therapy may be preferred[80] either with pharmacological treatment or, in selected patients, using pulmonary vein ablation. If atrial flutter, AVNRT, AVRT, or other supraventricular tachyarrhythmias are the cause for inappropriate shocks, catheter ablation should be considered. In cases of inappropriate shocks due to sinus tachycardia, increased β-blockade usually suffices, whereas the addition of non-dihydropyridine calcium channel blockers may be occasionally necessary in selected patients without heart failure.

In addition, ICD devices offer a variety of algorithms for the discrimination between SVT and VT aiming to withhold therapy delivery. These vary between single- and dual-chamber devices. Dual-chamber ICDs take advantage of atrial sensing capabilities to enhance discrimination between SVT and VT. Single-chamber devices have limited capabilities for arrhythmia discrimination[81] and largely rely on sudden onset or stability criteria. Recently, morphology criteria are more commonly used in addition.

Accordingly, appropriate programming of these tools may help to reduce the number of inappropriate ICD therapies. *Table 3* summarizes the discrimination algorithms commonly implemented in current ICDs, and *Table 4* shows causes of inappropriate shock delivery due to signal misinterpretation and provides solution strategies. *Figure 3A−D* gives typical examples for appropriate and inappropriate shock delivery.

Device-related causes of arrhythmia

When analysing the cause of ventricular arrhythmias leading to ICD therapies, clinicians should be aware of several mechanisms by which failure but also ordinary operation of the device may provoke arrhythmia.[82] As described in *Table 4* and *Figure 3D*, lead failure and connector-block issues are a frequent cause of inappropriate therapies. Furthermore, the delivery of ATP or inappropriate shocks may trigger the onset of malign arrhythmia. Additionally, short−long−short sequences facilitated by bradycardia pacing might constitute an important mechanism of device-related ventricular proarrhythmia.[83] Although a rare event, the possibility of pacing-related monomorphic and polymorphic VTs in patients receiving CRT, the latter possibly resulting from an increased dispersion of refractoriness and heterogeneity in conduction patterns, should be ruled out.[84] Finally, lead dislodgement or local lead effects with mechanical irritation and late fibrosis may be a potential mechanism for VT.[83] Therefore, cardiac imaging should be performed to exclude mechanically induced VT/VF especially in the early period after implant.

Catheter ablation

Although antiarrhythmic drugs may reduce recurrence rates of VTs and ICD shocks, their use is often limited by decreased efficacy and significant side effects.[58,85] Over the past two decades, the understanding of ventricular arrhythmogenic substrates and electrophysiological techniques and tools for their ablation have been greatly improved. As a result, catheter ablation of VTs is

Table 3 Algorithms for discrimination of atrial tachycardia and VT

Algorithm	Principle algorithm	Comment
SVT detection	Compares atrial and ventricular rates before further discrimination criteria are applied (dual-chamber ICDs)	Possible outcomes are that atrial and ventricular rates are equal (A = V), atrial rate is faster (A > V), or vice versa (V > A). In the case of V > A, the diagnosis is VT. If A = V, additional criteria are required for discrimination
Sudden onset (SO)	The SO criterion is fulfilled if the RR interval shortens by a programmed percentage when compared with the average number of preceding beats	Allows discrimination of gradually accelerating sinus tachycardia from suddenly occurring VT. May fail if VT occurs during sinus tachycardia (e.g. exercise) with a small decrease in RR interval
Rate stability (RS)	RS is expressed as %deviation between RR intervals	Confirms AF. However, AF with pseudo-regular ventricular rhythm and irregular VTs can be falsely classified as VT or SVT, respectively, which may cause delivery of inappropriate therapy or delay of appropriate therapy
Sustained rate duration (SRD)	Initiates shock delivery after a programmable time interval (e.g. 1 min) even if the episode has been classified as SVT	Aims to prevent that VT therapies are erroneously inhibited by sudden onset and stability criteria. SRD may cause inappropriate shocks. Activation should be avoided, if possible, or programmed to a long-time interval
Morphology discrimination (MD)	Based on the comparison of intracardiac electrograms in sinus rhythm and during VT[111]	Composes templates of electrogram morphology during normal rhythm that is expected to differ from that registered during ventricular arrhythmia. Especially useful in single-chamber devices due to the lack of atrial lead information. MD algorithms should not be activated in patients with bundle branch block or rate-dependent electrogram changes

Table 4 Inappropriate shocks due to signal misinterpretation

Problem	Cause	Comment
T-wave oversensing (*Figure 3C*)	False-positive arrhythmia detection. ICD misclassifies T-wave as R-wave	Common cause of inappropriate shocks[112] often in the setting of low sensing thresholds due to low-amplitude R-waves. Can be addressed by (i) decreased ventricular sensing (sensing of low-amplitude VF must be enabled), (ii) lengthening of the refractory period to suppress T-wave detection (may limit the detection of VT), (iii) changing sensing decay parameters (not available in all devices). Nevertheless, lead revision is often required. A true bipolar defibrillation lead may be preferred compared with integrated bipolar leads.[113] Insertion of a sensing/pacing lead may be considered
Electromagnetic interference (EMI; *Figure 3D*)	Owing to electromagnetic radiation from external sources. EMIs can be misinterpreted by the ICD as intracardiac signals resulting in electrical shocks and/or withhold of pacing	Household appliances (microwave oven, portable telephone, personal computers, etc.) do not interfere with modern ICDs. Cellular phones should be held on opposite side and close contact with the device should be avoided.[114] Electromagnetic sources potentially interfering with (especially older generation) ICDs are electronic article surveillance devices, metal detectors, improperly grounded appliances held in close contact to the body, high-voltage power lines, transformers, welders, electric motors, induction furnaces, degaussing coils, MRI scanners, electrocautery during surgery, defibrillation, neuro-stimulators, TENS units, radiofrequency catheter ablation, therapeutic diathermy, radiotherapy, and lithotripsy. Very strong EMI fields may reset the ICD to a 'safety' or 'stand-by' mode
Lead- and connector-block failure	Includes lead fracture, lead insulation failure, conductor coil fracture, and loose set screws	Frequent cause of inappropriate shocks. Lead fracture usually causes high lead impedance (1000–2000 Ω) and outer insulation failure low impedance. Inner insulation failure allows the two conductor coils to communicate. A conductor coil exposed to skeletal muscle may cause inappropriate shock due to oversensing (*Figure 3D*). Can be revealed by an analysis of intracardiac electrograms and event markers (oversensing, non-physiological short intervals <150 ms). Modern ICDs provide alert features for lead integrity. Remote device monitoring is helpful to identify malfunction early. Reprogramming the ICD is usually not sufficient and replacement or reconnection of the lead is necessary. Insertion of a sensing/pacing lead may be considered
Double or triple sensing of ventricular signals	Can occur in patients with wide ventricular complex	May be prevented by changing the sensing decay algorithm, lengthening of the refractory period, or lowering of the sensitivity. Newer devices allow for increasing the blanking period
Myopotentials	Detection of pectoral myopotentials	Very uncommon. Has been reported due to reversal of the proximal and distal coil in the header. Oversensing of diaphragmatic myopotentials occurs more frequently with integrated bipolar leads. Decreasing the sensitivity is the initial step. Repositioning of the lead or implantation of a true bipolar lead may be required
Double tachycardias	Atrial and ventricular arrhythmias exist at the same time	May result in withholding therapy, e.g. if AF is sensed at a higher rate than concomitant VT. Morphology criteria may help to discriminate SVT vs. VT

today considered earlier in the management of patients with recurrent VTs.[13,30]

Strategies to ablate VTs in patients with different morphological substrates have been discussed in detail in other documents.[42]

Principally, the mechanism of VT determines the selection of an appropriate mapping strategy to identify target sites for catheter ablation. A 12-lead electrocardiogram of the clinical VT is essential to document the morphology. If not available, the VT cycle length

from stored intracardiac electrograms may be the only available information. In these patients, substrate identification using electrophysiological mapping techniques and voltage criteria may enable substrate modification to suppress all inducible VTs. After an acutely successful ablation procedure, long-term recurrences may still occur and most patients with structural heart disease will continue to have a standard indication for ICD therapy. More recently, the concept of prophylactic catheter ablation of VT in post-myocardial infarction patients with a secondary ICD indication has been introduced.[13,14] However, the results of larger studies should be awaited before prophylactic ablation can be recommended.

It has to be emphasized that catheter ablation of ventricular arrhythmias in structural heart disease requires special training and should be reserved for expert centres.

Catheter ablation of ventricular tachycardia is recommended

(1) For symptomatic sustained monomorphic VT necessitating frequent ICD therapies despite antiarrhythmic drug therapy or when antiarrhythmic drugs are not tolerated or not desired (especially when VT recurrences fulfil definition of ES).

(2) For control of recurrent symptomatic or incessant monomorphic VT not suppressible by antiarrhythmic drug therapy, regardless whether VT is stable or unstable, or multiple VTs are present.

(3) For bundle branch re-entrant or interfascicular VTs.

(4) For recurrent sustained polymorphic VT and VF refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted by ablation.

Ventricular tachycardia catheter ablation is contraindicated

(1) In the presence of a mobile ventricular thrombus, epicardial ablation may be considered.

(2) For VT due to transient, reversible causes, such as acute ischaemia, hyperkalaemia, or drug-induced torsades de pointes.

Long-term follow-up

Patient education and counselling are important components of an integral follow-up strategy and mandatory for the overall success of ICD therapy. This applies in particular to patients having received shocks. Recent guidelines recommend in-office follow-up visits within 72 h and again 2–12 weeks after implant, followed by regular in-office or remote follow-up visits every 3–6 months.[49] However, patients with a recent shock event may require intensified in-office or remote follow-up in order to evaluate the clinical course of the arrhythmia, the underlying heart disease and the response to drug therapy or other interventions.

In-office follow-up

Apart from the technical device assessment, in-office follow-up visits offer the important opportunity to provide oral and written information. The ICD patient and his family should have

a basic understanding of the technical aspects of treatment as well as a realistic expectation about its capabilities and limitations. It is prudent to discuss the potential scenario of shock delivery and to provide information how to act in such situation.

Remote follow-up

Remote monitoring is being increasingly used in ICD follow-up and offers the continuous surveillance of technical device performance and diagnostic information stored by the device.[16,86] With last generation technology, sustained or self-limited VTs and delivered therapies can be notified by automatic wireless data transmission to a central server or service centre that forwards an alert to the clinical staff by e-mail or short messages service (sms). The clinician can then evaluate episode details on a website. In non-wireless devices, patients may manually send the data and inform the ICD clinic by phone about the transmission in the case of significant events. This allows a prompt evaluation of the appropriateness of detection and effectiveness of delivered therapy. If device function is deemed appropriate and clinical status is stable, the patient can be reassured and further follow-up scheduled. In the opposite case, patients can be directed to a hospital or the ICD clinic. Remote monitoring can also alert on clinical or device-related issues that are associated with an increased risk for appropriate or inappropriate shock delivery, e.g. recurrent self-limited VT, AF with high ventricular heart rate response, lead failure among other things.[87] Timely treatment of such conditions may prevent clinical deterioration and/or subsequent shock therapies.

In an Italian multicentre study using the non-wireless CareLink™ Network system,[88] 81% of ventricular tachyarrhythmia episodes could be analysed remotely, and in 85% of them, no further action was needed. The HRS recommends that the cardiac rhythm management device manufacturers develop and utilize wireless and remote monitoring technologies to identify abnormal device behaviour as early as possible and to reduce under-reporting of device malfunction.[89]

Remote monitoring has been introduced as a standard clinical practice in a recent expert consensus[49] stating that it can replace standard in-office visits. In-office visits, however, are recommended for the post-implant follow-up, after 1 month and at least once a year.

Anxiety and depression

Psychological distress: prevalence, severity, and chronicity

Implantable cardiac defibrillator therapy is generally well accepted by patients and, on average, associated with preserved or improved quality of life when compared with antiarrhythmic treatment.[90–92] However, a subgroup has difficulty adjusting to the new demands of living with the heart disease itself and the ICD, with approximately one-third of ICD patients experiencing anxiety and depression.[12] Anxiety is the most important psychological side effect of ICD therapy, attributable to the device being able to provide an uncontrollable shock.[12,93] The level of psychological distress varies in severity, ranging from low (normal) levels of

Table 5 Continuum of shocks, coping, and distress

	Arrhythmia	Coping	Distress	Feelings, thoughts, and behaviours
Continuum	No arrhythmia	Optimism	Reassurance	ICD as 'guardian angel'
		Active coping	Successful adjustment	ICD doesn't bother me
	ATP only	Faith in doctors	Realistic fear	ICD may fail
	Single shock	Depressive coping	Adjustment disorder	Uncertain if ICD keeps me safe
			Shock phobia	Avoid activities that might trigger shocks
	Multiple shocks	Distraction/denial	Moderate depression/agoraphobia	Avoid any activities, withdraw
	Electric storm	Catastrophizing	Dysthymia/generalized anxiety	Lose interest/confidence in life, permanent worry
		Resignation	PTSD/personality change	Permanent threat and arousal
			Severe/recurrent depression	Wanting to be dead

Modified from Sears and Conti.[12]

distress to severe disorder[12] such as post-traumatic stress disorder.[94] Levels of anxiety and depression generally decrease during the first year following implantation, indicating that patients adapt to ICD therapy over time.[93] However, if anxiety is present at the time of implantation, it may persist up till 12 months in as many as 50% of patients.[95]

The vicious cycle of shocks, psychological distress, and health outcomes

Data from observational studies and clinical trials show that the experience of ICD shocks is linked to poor psychological and quality of life outcomes, although the evidence supporting the impact of shocks on these outcomes is not consistent.[92,96] The appropriateness and the number of shocks may also have a differential impact on patient distress and device acceptance. Awareness of having received inappropriate therapy and/or an ES may compromise patient trust in the device, and increase levels of psychological distress.[97] A continuum of the type of ICD therapy, how patients cope with the therapy, and the associated distress, feelings, and thoughts are presented in *Table 5*.

Not only the actual ICD shock but also the fear of shock incurs an increased risk of distress[98] in particular in patients with a specific psychological profile—such as the *distressed* (Type D) personality.[93] This emphasizes the importance of taking the psychological profile of the patient into account, including factors such as Type D personality,[99] depressive coping,[100] and lack of optimism.[101] The presence of one or more of these factors has been shown to be at least as important as the event of an ICD shock itself in predicting adjustment problems of ICD patients. Patients with a high-risk psychological profile may have a tendency to interpret ICD shocks as malignant, suggesting that it is not the actual shock that leads to distress but more the patient appraisal and interpretation of the shock.[12] Importantly, the manifestation of psychological distress may lead to avoidance behaviours, a sedentary lifestyle, sexual problems, and poor quality of life,[12] but also to an increased risk of life-threatening arrhythmias[102,103] and mortality in ICD patients.[94,104] Therefore, breaking this vicious circle as hypothesized in *Figure 4*, which also summarizes the underlying physiological mechanisms, is important.
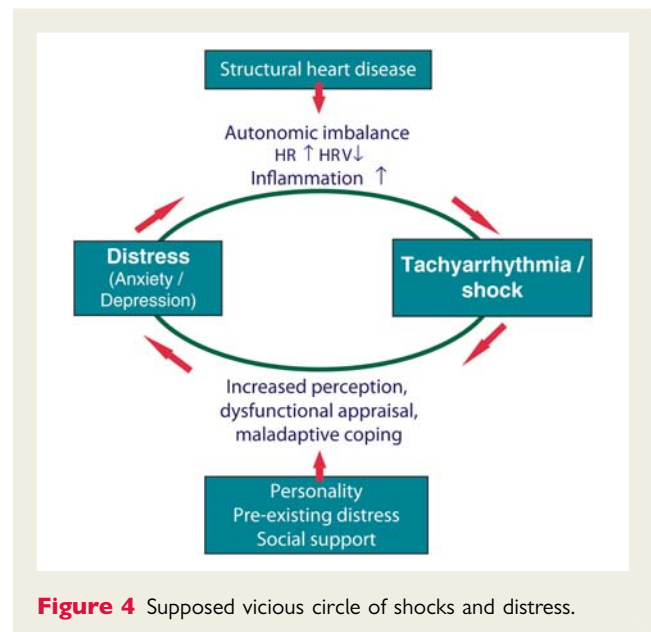

Figure 4 Supposed vicious circle of shocks and distress.

Preventive measures

One way of breaking this vicious cycle is to prevent the manifestation of anxiety and depression. This can be facilitated by reducing shock rates and by supporting the patients' coping resources. Therefore, psychological symptoms and maladaptive personality traits or coping strategies should be identified as early in the course of disease as possible, preferably even before ICD implantation. In clinical practice, this can be done by means of asking the patient to complete a self-report measure, with our recommendation of a list of such measures provided in *Table 6*. Since faith in physicians is paramount to effective coping in ICD patients, it is essential to build up a trustful physician–patient relationship and to offer easy access to physician support and advice in the case of problems and uncertainties (*Table 7*). Merely listening to patient concerns may reduce their worries and can by itself improve coping. Active problem-oriented coping is often helpful for adapting to new challenges such as shocks and can be supported by providing information about heart disease, the ICD, and how the ICD works. Even a few minutes more can make a difference, reduce patients' uncertainties and their suffering. Hence, when patients have received an ICD shock, sufficient time

Table 6 Recommended measures to identify high-risk patients post-shock

Risk factor	Questionnaire	Number of items	Minutes for patients to complete	Available in ≥3 languages
ICD concerns	ICDC[a]	8	3–5	Yes
Anxiety	FSAS[a]	10	3–5	Yes
	HADS-A[b]	7	3	Yes
	STAI (state only)[b]	20	3–6	Yes
Depression	HADS-D[b]	7	3	Yes
	PHQ-9[b]	9	3	Yes
Post-traumatic symptoms	IES-R[b]	22	10	Yes
Type D personality	DS14[b]	14	5	Yes

DS14, Type D Scale; FSAS, Florida Shock Anxiety Scale; HADS, Hospital Anxiety and Depression Scale; ICDC, ICD Concerns Questionnaire; IES-R, Impact of Event Scale Revised; PHQ-9, Patient Health Questionnaire; STAI, Spielberger's State-Trait Anxiety Inventory.
[a]Disease-specific.
[b]Generic.

Table 7 Ten practice tips for preventing and reducing shock-induced distress

General measures
  Establish trustful relationship with patients and partners
  Establish trustful relationship with mental health expert inside or outside the team
  Listen to patients and leave room for questions and emotions
Preventive measures
  Provide information (including patient brochures) and answer patients' questions about the ICD and related topics before and after implantation. Explain that the ICD may save the patient's life but emphasize that it does not cure heart disease
  Discuss participation in comprehensive rehabilitation and encourage regular exercise adapted to individual preference and physical capacity
Assessment and basic treatments
  Regularly explore beliefs, health resources, and distress (both pre-existing and newly developed) in person or via telephone calls
  Offer quick help if shocks and/or distress occur
  Ask for subjective effects of shock (e.g. sensory and emotional perception, interpretation, behavioural consequences) and encourage resumption of activities as soon as possible
  Explore patient needs for more information and support
  Provide reassurance and referral to a mental health expert, structured psychosocial intervention, and/or support group as appropriate

should be spent with patients to provide relevant information, answering patients' questions, and listening to worries and concerns.

During phases of acute distress, patients may be unable to process information effectively, which may necessitate repeated explanations. Patients should be asked what they have understood and whether they still have concerns and unanswered questions.

Family support and support groups

Family support is critical for the ICD patient, both in terms of instrumental and emotional support. However, families and in particular partners of ICD patients are faced with a number of

uncertainties and fears that may lead to even higher levels of distress than in patients. Advice how to cope with a partner's ICD and heart disease has been published elsewhere.[105]

One further option can be the referral to self-help groups, if they are locally available, or implementation of a clinic-associated support group for ICD patients. Patients with sufficient internet experience can also be invited to join forums for ICD patients on the internet.

Treatment and management of distress in clinical practice

Data on the treatment of comorbid distress in ICD patients are limited. Current reviews and treatment recommendations mainly advocate the use of techniques for psychological crisis intervention, cognitive behavioural therapy, relaxation, telephone counselling, exercise, and SSRIs.[94,106,107] It is important to keep in mind that some psychotropic substances may exert a marked QT-prolonging effect. There is no evidence to support differential assignment of individual patients to specific treatments or combinations. Careful individual assessment and counselling (see above) is therefore recommended for all ICD patients receiving shocks. Patients should be encouraged to maintain or resume normal life as soon as possible in order to prevent phobic avoidance behaviour.[108] In the case of persisting distress or psychopathology, a mental health expert should be involved. In the absence of data comparing different psychotherapeutic approaches, any treatment of individual psychopathology by means of established psychotherapeutic techniques can be recommended depending on local availability.

Psychotropic drug treatment should be prescribed by physicians knowledgeable of the specific demands of psychopharmacotherapy in cardiac patients. For theoretical reasons, antidepressants without anticholinergic, or class I antiarrhythmic effects such as SSRIs, appear preferable, although no specific evidence from controlled trials in ICD patients exists.

Summary

The management of ICD patients after shock therapy varies, depending on the number of shocks, their nature (appropriate

or inappropriate), and the patient's clinical condition. The continuum of care ranges from single shocks without clinical instability that permit assessment in due course performed either in-office or remotely to repeated shocks with haemodynamic instability that mandate emergency hospitalization. Implantable cardiac defibrillator interrogation establishes the diagnosis of supraventricular or ventricular arrhythmias and recognizes device malfunction. Programming the device algorithms that discriminate supraventricular from ventricular arrhythmias reduce inappropriate shocks. Appropriate shocks can be reduced by reprogramming ATP parameters, based on the response of stored VTs to ATP attempts and on the haemodynamic tolerance of previous VT episodes. Clinical assessment aims at the identification of triggering factors, such as myocardial ischaemia, decompensated heart failure, and electrolyte abnormalities. Antiarrhythmic agents can reduce episodes of supraventricular and ventricular arrhythmias and may decrease the VT rate, rendering rapid VTs amenable to successful ATP. If antiarrhythmic regimens fail, substrate modification by catheter ablation in an experienced centre decreases arrhythmic burden. Patient management after ICD shocks needs to be re-evaluated during frequent follow-up visits, and device reprogramming and/or medical treatment adjustments are occasionally required. Particular emphasis should be given to psychological distress, commonly observed after ICD shocks. Reassurance often suffices, but specialized assessment and counselling may be necessary in selected patients.

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