Review

What do implantable cardioverter/defibrillators teach us about the mechanisms of sudden cardiac death?

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Abstract

Almost two decades of experience with the ICD have resulted in a better understanding of the nature of life-threatening ventricular tachyarrhythmias. The most pertinent achievements in this respect stem from clinical follow-up observations in patients with important clinical entities such as dilated cardiomyopathy or the Brugada syndrome. For instance, patients with dilated cardiomyopathy presenting with syncope and a negative electrophysiological study have been shown to have a high incidence of appropriate usage of their ICD. On the other hand, relative little has been gained from the extensive electrogram storage capabilities of third and fourth generation ICDs. Basically, careful evaluation of these electrograms has confirmed previous anecdotal data obtained from analysis of Holter recordings of patients who died suddenly while wearing the ECG recorder. In most instances, VT/VF episodes are triggered by relatively late-coupled premature beats whereas short–long–short sequences have been observed only in few patients. One future research avenue concerns the more detailed analysis of changes in cardiac autonomic tone preceding the occurrence of sustained ventricular tachyarrhythmias which can be assessed from the electrogram storage of the ICD. © 2001 Elsevier Science B.V. All rights reserved.

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1. Introduction

Sudden cardiac death (SCD) remains one of the leading killers in the industrial world, responsible for an estimated 300 000–400 000 deaths every year in the United States. Episodes of ventricular tachycardia (VT) and ventricular fibrillation (VF) are the underlying causes in the majority of victims of SCD. At the time of emergency intervention, VF is found in approximately 40% of SCD victims, with asystole and electromechanical dissociation becoming more frequent the longer the intervention is delayed [1]. In patients in whom the time interval between the clinical event and the first electrocardiogram was less than 4 min, the incidence of VF was documented to be as high as 95%. However, the specific types of arrhythmias preceding the onset of VF in the majority of patients is not precisely known.

Reentry is the mechanism underlying VT and VF particularly in patients with structural heart disease. Reentry requires the presence of a fixed or functional circuit capable of unidirectional block in one limb and slow conduction in the other. Theoretically, reentrant arrhythmias can be initiated by several mechanisms. For instance, spontaneous premature beats may invade a reentrant circuit, slow conduction in one limb and create unidirectional block in the other resulting in VT. Alternatively, an increase in dispersion of repolarization as a consequence of short–long–short cycles may facilitate reentrant arrhythmias [2]. Finally, modulating factors such as the autonomic nervous system may render the ventricles more susceptible to sustain reentrant arrhythmias [3].

To evaluate the conditions and precursors resulting in sustained ventricular tachyarrhythmias and ultimately in SCD in the clinical setting has been extremely difficult in the past. The availability of third-generation implantable cardioverter/defibrillators (ICD) which have extensive...
data storage capabilities of RR intervals and intracardiac electrograms has provided the opportunity to study the initiating sequences of ventricular tachyarrhythmias in greater detail. Moreover, follow-up of ICD recipients may allow insights in the ‘natural course’ of their disease and a more precise determination of specific arrhythmia-related risk. It is the focus of the present review to summarize results of studies utilizing the ICD as a tool to increase our knowledge on sudden arrhythmic death.

2. SCD documented during Holter monitoring

For many years, data on electrical triggers of sustained VT/VF could only be obtained from analysis of Holter recordings or from telemetry recordings in intensive care units [4–6]. Despite the fact that all of these studies comprised only small patient populations, some important features of VT/VF induction could be delineated (Table 1). Whereas bradyarrhythmias were found as the cause of SCD in approximately 20% of cases (except in patients with advanced heart failure [7]), the most frequently found arrhythmic event was monomorphic VT degenerating into VF. It was therefore postulated that in some patients VT-induced hypotension, ischemia, and left ventricular dysfunction eventually causes the rhythm to deteriorate to VF. Interestingly, short-coupled ventricular premature beats were only seen in exceptional cases whereas rather late-coupled VPBs initiated the VT much more frequently [4–6]. So-called short–long–short cycles resulting in increased disparity of ventricular repolarization were only seen in spurious cases [6]. Thus, for a long time data on electrical triggers of VT/VF were only based on fortuitous observations in a very small number of patients.

3. Lessons about SCD learned from the ICD

3.1. Clinical observations

Over the past 10–15 years, device therapy has become the mainstay of therapy for secondary prevention of SCD. Three large, prospective, randomized trials have convincingly demonstrated that ICD therapy compared to therapy with antiarrhythmic drugs (mainly amiodarone) is associated with improved survival [10–12]. The widespread use of ICD therapy has therefore enabled electrophysiologists to study additional issues related to the clinical problem of SCD.

3.2. Recurrent arrhythmias in patients resuscitated from VT

It is well-known that VF survivors differ clinically from patients with a history of monomorphic VT. Compared to the latter, VF survivors tend to have a better preserved left ventricular function [13], are less likely to have a history of myocardial infarction [13,14], are less likely to have late potentials detected by signal-averaged ECG [15], and are less likely to have monomorphic VT induced at programmed ventricular stimulation [13–16]. However, the incidence of spontaneous monomorphic VT in unselected patients resuscitated from VF remained unknown. The ICD provided the opportunity to study this issue and therefore

Table 1
Arrhythmias at the time of cardiac arrest / sudden cardiac death documented by Holter monitoring or telemetry

<table>
<thead>
<tr>
<th>Study (Reference)</th>
<th>Patients</th>
<th>Bradyarrhythmia</th>
<th>Tachyarrhythmia</th>
<th>VPB coupling prior to VT/VF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pratt et al. (1983) [4]</td>
<td>15</td>
<td>None</td>
<td>VT → VF n=15 (100%)</td>
<td>VPB prematurity index 1.27±0.28</td>
</tr>
<tr>
<td>Panidis et al. (1983) [5]</td>
<td>15</td>
<td>n=3 (20%)</td>
<td>Slow VT n=2 (13%)</td>
<td>R on T n=1 (7%)</td>
</tr>
<tr>
<td>Kempf et al. (1984) [6]</td>
<td>27</td>
<td>n=7 (26%)</td>
<td>VT → VF n=20 (74%)</td>
<td>Long–short cycle n=2 (7%)</td>
</tr>
<tr>
<td>Luu et al. (1989) [7]</td>
<td>21</td>
<td>n=13 (62%)</td>
<td>VT → VF n=7 (33%)</td>
<td>R on T n=4 (15%)</td>
</tr>
<tr>
<td>De Luna et al. (1989) [8]</td>
<td>157</td>
<td>n=26 (16.5%)</td>
<td>VT → VF n=98 (62.4%)</td>
<td>VT most often included by late coupled VPB</td>
</tr>
<tr>
<td>Olshausen et al. (1991) [9]</td>
<td>61</td>
<td>n=11 (18%)</td>
<td>Mono VT n=26 (43%)</td>
<td>R on T n=13 (26%)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Poly VT n=15 (25%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Tdp n=5 (8%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Primary VF n=3 (5%)</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>296</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Study comprised only patients with advanced congestive heart failure. VPB, ventricular premature beat; VT, ventricular tachycardia; VF, ventricular fibrillation; mono VT, monomorphic VT; poly VT, polymorphic VT; Tdp, torsade de pointes.
to gain further insight in the mechanisms of cardiac arrest in VF survivors.

Two studies examined this issue by reviewing data obtained from electrogram storages of the ICDs [17,18]. Raitt et al. [17] reviewed the records from 111 consecutive ICD recipients. Fifty five of these patients had a history of only VF whereas in the remaining patients an episode of monomorphic VT was documented prior to ICD placement. During a 14-month follow-up period, spontaneous VT was documented in 18% of VF survivors compared to 54% of patients with a history of VT (P=0.002). In the first group, spontaneous VT was recorded in 11% of patients. The authors concluded from their observations that patients with a history of only VF should be considered a unique population and not simply a subset of VT patients and therefore suggested that the deterioration of rapid VT to VF is not the only clinically important mechanism of VF induction [17].

In a similar study, Rüppel et al. [18] collected data on 40 patients who had cardiac arrest secondary to VF (and no other arrhythmia documented) and who were fitted with an ICD capable of intracardiac electrogram recording and storage. Seventy percent of their patients suffered from coronary artery disease and the average LVEF was 42±18%. Over a follow-up period of 23±11 months, 36 VT episodes were recorded in 11 patients and five episodes of VF were documented in the remaining two patients. Accordingly, the main conclusion of this study was that VT is the most common ventricular arrhythmia recorded on device-incorporated electrograms. This findings may account together with the relatively well-preserved LV function for the ability of these patients to survive at the time of the index arrhythmia. The difference to the findings of Raitt et al. [17] was explained by the lack of full electrogram storage in their study and by differences in patient referral patterns.

In summary, therefore, survivors of VF or of VT differ with respect to several important clinical features. In both patient groups, however, a significant proportion of recurrent ventricular tachyarrhythmias are monomorphic VT episodes. Consequently, the use of antitachycardia pacing (ATP) as a modality to treat arrhythmia recurrences is recommended even in patients whose sole clinically documented arrhythmia was VF.

3.3. Relation of induced to spontaneous VT in ICD patients

The understanding of the relation between VT induced at electrophysiological study or at ICD predischarge testing and that occurring spontaneously is still incomplete. Accordingly, there is an ongoing debate concerning the validity of predischarge ICD testing in an attempt to tailor ATP modalities according to induced VT. Whereas some centers rely on this approach, others do not routinely perform predischarge testing but rely on empiric ATP algorithms. Another issue of controversy relates to the predictive value of electrophysiological testing with respect to subsequent VT or VF events. This latter question was addressed by the aforementioned study of Rüppel et al. [18] who performed invasive EP testing prior to ICD implantation. In that study, programmed ventricular stimulation proved to be a poor predictor of arrhythmia recurrence as well as of the specific type of subsequent spontaneous arrhythmias (i.e. VT versus VF) [18]. Specifically, only 29% of patients with inducible arrhythmias at EP testing were also found to have spontaneous arrhythmia recurrences during follow-up.

Monahan et al. [19] examined the issue of induced versus spontaneous VT episodes in a cohort of 19 patients with coronary artery disease who received an ICD capable of storing far-field electrograms. Electrograms obtained at VT induction at predischarge testing and those from spontaneous subsequent VT events were carefully compared. This comparison revealed that in 13 of 19 patients (68%) induced and spontaneous VT differed significantly in terms of VT cycle lengths and morphologies [19]. In patients in whom induced and spontaneous VTs were different, the induced arrhythmias were significantly faster compared to the spontaneous ones. This finding was in agreement with observations during attempts of VT ablation where it was demonstrated that the induction of rapid non-clinical VTs is common in patients with a history of VT. It is commonly accepted that these rapid nonclinical VTs may not warrant specific therapy.

Similar observations were recently made by Orlov et al. [20]. In 148 ICD recipients who were followed for 44±23 months, there was also no correlation between inducible and recurrent VT in terms of VT cycle lengths. Moreover, clinical or inducible VF were not associated with recurrent VF during follow-up.

The reason for this discrepancy between VT induced at predischarge and observed during follow-up is not entirely understood. There is speculation that increased levels of endogenous catecholamines at the time of laboratory testing may account for the faster VT rates [19]. However, convincing experimental evidence in support of this is still lacking. Monahan et al. [19] therefore argue that the most likely explanation for the observed discrepancy is probably the induction of rapid non-clinical arrhythmias.

4. Natural course of specific types of heart disease associated with an increased risk of sudden death

Recent years have seen an enormous increase in our knowledge concerning risk stratification for sudden death in survivors of acute myocardial infarction [21]. Several prospective studies have shown that primary prevention of arrhythmic death in well-defined patient populations is feasible [22,23]. On the other hand, no such progress could be obtained in other important patient subsets. Anecdotal
data or uncontrolled smaller studies using the ICD in patients deemed to be at high risk for sudden cardiac death may be at least helpful in better defining the role of device therapy in such individuals.

4.1. Nonischemic dilated cardiomyopathy

One important example is represented by patients who suffer from dilated cardiomyopathy and present with syncope of unknown origin. Electrophysiological testing is frequently performed in these patients in an attempt to determine the cause of syncope but results have generally been disappointing with a high percentage of patients being non-inducible [24–27]. There have been two studies of patients with DCM treated with an ICD [28,29]. However, in each of these studies only a limited number of patients who presented with syncope had been included. Nevertheless, shock therapy was documented in 42 and 49% over a follow-up period of 18 and 36 months, respectively [28,29]. More recently, Knight et al. [30] specifically addressed the patient population with DCM and syncope of unknown origin. They treated 14 such patients — all of whom had a negative EP test — with an ICD and followed them for an average of 24±13 months. During this observation period, 7/14 patients received an appropriate ICD shock for termination of VT or VF. This incidence was comparable to the rate of ICD therapy in 19 DCM patients who had presented with cardiac arrest (8/19 patients or 42% with appropriate ICD therapies). The authors conclude from their observations that patients with DCM, unexplained syncope, and a negative EP test should be treated with an ICD due to the high likelihood of sudden arrhythmic death [30].

4.2. Idiopathic ventricular fibrillation

A second patient group in whom the ICD has helped to better define the natural course of the disease in terms of the inherent risk of sudden death concerns patients who present with a history of cardiac arrest in the absence of significant structural heart disease. It is estimated that these patients account for 3–9% of out-of-hospital cases of VF. Although these individuals represent an inhomogeneous group, recent clinical, basic and genetic studies have highlighted some specific disease entities. Examples comprise patients with the so-called Brugada syndrome [31–33], the sudden unexplained death in Thai men syndrome [34], or abortive forms of the congenital long QT syndrome.

Previously, some studies have suggested that survivors of idiopathic VF may have a benign clinical course when treated with betablockers, class III agents or even left untreated [35]. Other investigators have reported favorable outcomes of survivors of idiopathic VF on therapy aiming at antagonizing high sympathetic tone [36].

More recently, however, data have accumulated which indicate that patients with a history of VF in the absence of recognizable structural heart disease have a substantial risk of subsequent ventricular tachyarrhythmic events. For instance, Meissner et al. [37] performed a ten-center retrospective study on 28 survivors of idiopathic VF who received an ICD. They found that survival was excellent in these patients since there were no cardiac deaths. However, 4/28 patients received appropriate ICD shock therapy emphasizing the risk of recurrent sustained ventricular tachyarrhythmias. Wever et al. [38] prospectively followed 19 consecutive VF survivors without structural heart disease and observed major arrhythmia recurrences in 37%. Similarly, data from the unexplained cardiac arrest registry indicate a VF recurrence rate of approximately 25–30% during an average follow-up period of 28±23 months [39].

One of the best studied disease entities in this context is the so-called Brugada syndrome [31]. These patients present with an ECG pattern of right bundle branch block, ST segment elevation in leads V1–V3, and a history of VF in the absence of structural heart disease. The largest series of such patients comprises 63 patients who are followed at 33 centers worldwide [31]. Patients were treated differently: 35 received an ICD, 15 received pharmacological therapy (betablockers or amiodarone), and 13 were not treated at all. The incidence of arrhythmic events was similar in all three groups but there was a striking difference in total mortality which was 0% in the ICD group, 26% in the group treated pharmacologically, and 31% in the no treatment group. The authors therefore concluded that the risk of recurrent tachyarrhythmias is high in these patients and that only the ICD offers protection from recurrent sudden death. Recently, Nademanee et al. [34] reported on 27 Thai men referred because they had cardiac arrest due to VF. The authors noted striking similarities with the Brugada syndrome; as with this condition, the risk of recurrent sudden death turned out to be inordinate.

Although there are no prospective randomized trials on the efficacy of ICD therapy in the aforementioned groups of patients, the ICD itself has therefore helped to increase our knowledge concerning future arrhythmic risk in afflicted individuals. The relative rarity of these diseases will most likely also in the future exclude the conduct of scientifically sound trials.

5. Analysis of ICD stored data

5.1. Mode of induction of ventricular tachyarrhythmias

Initially designed primarily as safety tool to evaluate appropriateness of ICD therapy, the improved electrogram and data storage logs of contemporary ICDs have been extensively used to evaluate in more detail the onset mechanisms of VT/VF (Table 2). In one of the first such
Table 2
ICD electrogram storage: analysis of mode of induction of ventricular tachyarrhythmias

<table>
<thead>
<tr>
<th>Study (Reference)</th>
<th>Patients</th>
<th>VT events analyzed</th>
<th>Short–long–short VT initiation</th>
<th>VT initiation</th>
</tr>
</thead>
</table>
| Roelke et al. (1994) [40] | 27 | 73 | 14% | Mean VPB interval 364 ms  
Mean prematurity index 0.56 |
| Meyerfeldt et al. (1997) [41] | 38 | 286 | 70 (25%) | Mean prematurity index 0.64±0.12 |
| Saeed et al. (2000) [42] | 52 | 268 | NA | Three onset mechanisms:  
(a) Sudden onset→75 VT episodes  
(b) Extrasystolic onset→177 episodes  
(c) Paced onset→16 episodes  
Prematurity index  
(a) 0.75±0.12  
(b) 0.80±0.40  
(c) 0.58±0.30 |
| Taylor et al. (2000) [43] | 90 | 260 | 5 (2%) | 222 (85%) of VT episodes initiated by late-coupled PVC  
33 (15%) by an early-coupled PVC |

studies using RR interval recordings, Roelke et al. [40] showed that 45% of VT/VF episodes were induced by a single premature ventricular beat (VPB) whereas 22% were preceded by couplets and another 33% by multiple VPBs. Interestingly, these investigators described 14% of episodes (included in the first group) where the arrhythmia was preceded by a short coupled VPB resulting in a compensatory pause followed by another short coupled beat resulting in a short–long–short sequence (Fig. 1). This specific induction sequence has most often been associated with the occurrence of polymorphic VT from the torsade de pointes type [2,44]. However, a short–long–short stimulation sequence was found to also facilitate the induction of monomorphic VT in patients in whom this arrhythmia could not be induced at constant cycle lengths [45]. Meyerfeldt et al. [41] reported on the mode of onset in 286 VT episodes from 38 ICD recipients. Monomorphic VT initiated by short–long–short intervals was found as a patient specific phenomenon in 25% of patients. After these descriptive studies, the short–long–short interval gained increased clinical interest, since dedicated anti-bradycardia pacing algorithms were developed to prevent compensatory pauses reducing the likelihood of VT initiation.

In a recently published retrospective analysis, it was found that continuous biventricular pacing diminished the need for ICD therapy [46]. As discussed in that paper, these results may be related to — among other potential factors — the rate smoothing effect of continuous pacing. Automatic rate stabilization algorithms have already been implemented in dual chamber ICDs. In a prospective randomized study, however, no clinical benefit of this feature could be demonstrated in a group of 309 ICD recipients who were not dependent on continuous pacemaker therapy [47]. A possible explanation for this disappointing result may be an overestimation of the incidence of short–long–short sequences in the earlier reports. This hypothesis is supported by results from the largest systematic study reviewing VT-onset electrograms. Taylor et al. [43] analyzed 260 episodes of VT onset in 90 patients fitted with an ICD with extended electrogram storage capabilities. In 85% of arrhythmic episodes, a late coupled single VPB triggered the arrhythmia (Fig. 1); of note, only 2% of VT events were preceded by short–long–short cycles. Analysis of the patients according to the type and severity of the underlying heart disease did not reveal any significant clues as to the various types of arrhythmia onset. Thus, as emphasized by Wood and Ellenbogen [48] in an accompanying editorial, more complex trigger factors appear to be involved in the induction of VT since the observed late coupled premature beats occur much more frequently in the same patients without inducing any sustained arrhythmia. The analysis of coupling intervals preceding VT/VF alone, therefore, may be of limited value to explain the complex multifactorial pathophysiological situation associated with the genesis of life threatening ventricular tachyarrhythmias.

5.2. Time patterns, cluster arrhythmias, and electrical storm

The advanced ICD data log provides detailed data on the time of onset as well as the RR intervals of a longer time period (i.e., >1000 beats) preceding the onset of a tachyarrhythmic episode. Such data constituted the basis of studies on the circadian variance and the time patterns in the occurrence of VT/VF [49]. Interestingly, in many patients after long periods of clinically uneventful follow-up, a clustered incidence of arrhythmic events is found during a very short time [50]. The clinically most challenging situation in ICD recipients consists in the occurrence of an electrical storm defined as recurrent VT or VF occurring two or more times within 24 h and usually requiring electrical cardioversion or defibrillation [51]. In a prospec-
tive observational study from our institution, a 10% incidence (14/136 patients) was found with the majority of events occurring during the first 6 month after ICD implant [52]. Most episodes could be managed with a combination of amiodarone and beta-blockers. During follow-up, a previous episode of electrical storm did not independently confer increased mortality in these patients.

As the applied definition included only patients with multiple shocks and most VTs can be terminated by ATP, we recently studied the time patterns of any recurrent ICD
therapy including episodes of asymptomatic ATP. In a consecutive series of 150 patients with a follow-up of 6 months, a total of 642 spontaneous episodes with EGM classified as appropriate and successfully terminated VT were analyzed [53]. Eighteen patients had only one episode, whereas in 34 patients 590 episodes occurred in clusters. Overall, two thirds of episodes occurred within 60 minutes after successful termination of the previous VT and 14/34 patients had at least 50% of their episodes within 10 minutes of termination of a previous episode. We found no clinical differences between patients with clustered or separate recurrence of ICD therapy [53]. Summarizing these findings, it seems conceivable that additional factors contribute to a temporal vulnerability for ventricular arrhythmias in patients with organic heart disease.

Myocardial ischemia, despite being a frequent cause of arrhythmias in acute coronary syndromes, has only rarely been found in ICD recipients as a cause of electrical storm but worsening of congestive heart failure or electrolyte imbalances may contribute more significantly to such acute electric instability [52].

5.3. Mode of death in ICD recipients

From an epidemiological standpoint, sudden cardiac death would ideally describe patients dying of ventricular tachyarrhythmic events only. Electrogram storage of an ICD allows verification of the suspected mode of death in a growing number of patients. Interestingly, data from a large database with follow-up of 834 ICD patients, arrhythmias associated with ICD discharge were found only in seven of 17 patients classified on clinical grounds as sudden cardiac death [54]. Another investigation of the electrical phenomena occurring with the terminal arrhythmic event confirmed these results with the majority of deaths (69%) being not the immediate result of an arrhythmia. In the remaining 31% of deaths, electrograms were wide in 89% of stored episodes. The authors concluded that these electrograms represent electromechanical dissociation not susceptible to ICD therapy [55]. In contrast, a more recent study on the same topic [56] found that sudden death was associated with VT or VF events in two thirds and death occurred despite ultimately successful, although often protracted, device therapies. The worsening clinical status of their patients also suggested acute cardiac mechanical dysfunction as a frequent terminal factor [56]. In summary, the clinical classification of sudden cardiac death as it is commonly used in large clinical trials may be challenged.

5.4. Changes in autonomic tone prior to ICD therapy

Abnormalities in cardiac autonomic tone have been convincingly demonstrated to be associated with increased mortality in patients after myocardial infarction [57] with or without a history of sustained ventricular tachyarhythmias [58]. Analysis of heart rate variability from short- or long-term ECG recordings has been found to be an excellent noninvasive tool to evaluate changes in cardiac autonomic tone [59]. Accordingly, there have been attempts to derive information on autonomic tone from stored ICD data as well.

Mani et al. [60] used data from ICDs storing 1024 R–R intervals preceding a ventricular tachyarrhythmia. Power spectrum analysis was performed on 135 sets of 1024 RR intervals prior to ventricular arrhythmia and controls from 78 patients. RR intervals that led to VT/VF-onset had a significantly higher total spectral power than controls ($P<0.001$) during the course of 12 min, followed by a sudden elevation in spectral power within 100 s before the onset of VT/VF. These findings were recently substantiated by observations obtained in a multinational trial [61]; these investigators studied heart rate variability from similar ICD data logs in 58 post-myocardial infarction patients. A state of increased sympathetic tone was suggested by a significant reduction in HRV before VT/VF-onset compared with control conditions. Betablockers and dl-sotalol enhanced HRV in control recordings; nevertheless, HRV declined before ventricular tachyarrhythmia onset independent of antiarrhythmic drugs. Although sotalol has been shown to reduce the incidence of ICD therapy in a placebo-controlled, prospective study [62], further studies are required to assess whether the benefit of any specific drug may be at least in part due to changes in autonomic tone directly preceding an arrhythmia.

5.5. Association of atrial fibrillation with VT/VF

In patients with organic heart disease, atrial and ventricular arrhythmias may coexist. In this context, the presence of permanent or paroxysmal atrial fibrillation is of major clinical relevance, since rapid AV-node conduction of atrial fibrillation leads to inappropriate therapy in as many as 15% of ICD recipients [63]. Even in patients without previously known supraventricular arrhythmias stored electrograms reveal paroxysms of atrial fibrillation during follow-up. Newly designed dual chamber ICD algorithms are able to detect and report atrial fibrillation and atrial tachycardia during continuous monitoring with a high specificity [64] thereby significantly reducing the incidence of inappropriate ICD therapy.

With respect to the pathophysiological mechanisms underlying sudden cardiac death, a recent observation from our laboratory is of potential interest [65]. We could demonstrate that ICD recipients presenting with permanent atrial fibrillation at the time of implantation are at a higher risk of recurrent ventricular tachyarrhythmias compared to patients who present in permanent sinus rhythm [65]. Specifically, in a series of 250 consecutive patients, the presence of permanent atrial fibrillation at implant almost doubled the risk for appropriate ICD-therapy during follow-up (relative risk 1.8; 95% CI: 1.2–2.9; Fig. 2). On
Holter recordings of patients who died suddenly while wearing the ECG recorder. One future research avenue concerns the more detailed analysis of changes in cardiac autonomic tone as assessed from the electrogram storage of the ICD.

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