

# Risk factors and prognostic role of an electrical storm in patients after myocardial infarction with an implanted ICD for secondary prevention

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## Abstract

**Introduction:** The aim of our study was to determine the risk factors for electrical storm (ES) and to assess the impact of ES on the long-term prognosis in patients after myocardial infarction (MI) with an implantable cardioverter-defibrillator (ICD) for secondary prevention of sudden cardiac death (SCD).

**Material and methods:** We retrospectively analyzed 416 patients with coronary artery disease after MI who had an implanted ICD for secondary prevention of SCD. Fifty (12%) patients had one or more incidents of an electrical storm – the ES (+) group. We matched the reference group of 47 patients from 366 ES (–) patients.

**Results:** We analyzed 3,408 episodes of ventricular arrhythmias: 3,148 ventricular tachyarrhythmic episodes in the ES (+) group (including 187 episodes of ES) and 260 in the ES (–) group. Multivariate logistic regression showed that inferior wall MI (RR = 3.98, 95% CI: 1.52–10.41) and the absence of coronary revascularization (RR = 2.92, 95% CI: 1.18–7.21) were independent predictors of ES ( $p = 0.0014$ ). During 6-year observation of 97 patients, there were 39 (40%) deaths: 25 (50%) subjects in the ES (+) group and 14 (30%) in the ES (–) group ( $p = 0.036$ ). Independent predictors of death were: the occurrence of ES (HR = 1.93), older age (HR = 1.06), and lower left ventricular ejection fraction (HR = 0.95) (for all  $p < 0.001$ ).

**Conclusions:** Electrical storm in patients after MI with ICD for secondary prevention is a relatively common phenomenon and has a negative prognostic significance. Myocardial infarction of the inferior wall and the absence of coronary revascularization are predisposing factors for the occurrence of an ES.

**Key words:** implantable cardioverter-defibrillator, electrical storm, sudden cardiac death.

## Introduction

The term electrical storm (ES) was used for the first time in the early 1990s and was defined as a state of high electrical instability of the heart, which is manifested by numerous episodes of ventricular tachycardia (VT)/ventricular fibrillation (VF) within a short period of time. Electrical storms are associated with a very high mortality (80–90%) both during

the incident alone and during further observation [1, 2]. The use of an implantable cardioverter-defibrillator (ICD) changes the natural course of the disease. The main cause of death in this group is heart failure, and sudden cardiac death occurs in approximately 2–4% of cases. However, electrical storms are still an important problem, especially since the clinical presentation of an ES is often very dramatic with recurrent VT/VF that requires multiple defibrillator shocks. Eighty percent of patients with an ES are hospitalized [3, 4]. Most data about electrical storms come from patients with an implanted ICD for secondary prevention. By definition, an ES signifies the occurrence of three or more episodes of VT/VF that require ICD intervention within 24 h in which the interval between the distinct incidents is longer than 5 min [5].

Due to the increasing number of ICD recipients, electrical storms are observed more often and require adequate management that is specific for an ischemic and non-ischemic etiology of cardiac arrhythmia. However, pharmacological treatment is not always effective. In addition to pharmacological treatments, which are based on blockage of the sympathetic nervous system and the administration of class I agents, radio frequency ablation (RF ablation), neurosurgical sympathetic denervation and general anesthesia are used [6, 7]. On the other hand, ICD implantation for secondary prevention is the strongest predictor of ES [8, 9]. One of the major tasks is to select a group of patients who are at high risk of ES. There are limited data about ES, especially in high-risk patients who are homogeneous in terms of etiology and indications for ICD implantation. This encouraged us to investigate ES using our many years of experience in electrotherapy.

The aim of our study was to identify the risk factors for an ES in patients after myocardial infarction with an implanted ICD for the secondary prevention of SCD. We attempted to determine the influence of an ES and the type of therapy administered by an ICD, antitachycardia pacing (ATP) or shocks, on the long-term prognosis.

## Material and methods

We retrospectively analyzed 416 patients with coronary artery disease after myocardial infarction who underwent ICD implantation for the secondary prevention of SCD in the years 1997–2004. Four hundred sixteen patients were included in the study. All were consecutive patients after MI implanted for secondary prevention in 1997–2004. Secondary prevention of SCD was defined as a history of cardiac arrest due to VF or hemodynamically not tolerated VT and also after recurrent sustained VT, without reversible causes.

The observation was carried out until December 2006. The median length of the observation was 66 months.

Fifty (12%) patients in the study population had one or more incidents of ES: the ES (+) group. A representative control group was composed of 47 subjects who were matched with respect to age, sex and implantation date from the 366 patients without a history of ES.

An electrical storm was defined as the occurrence of three or more episodes of VT or VF that required the intervention of a device (ATP or defibrillation) within 24 h in which the intervals between distinct arrhythmias lasted more than 5 min [5].

Before an ICD implantation procedure, the following medical data were collected for all patients: age, sex, NYHA class, comorbidities (hypertension, hypercholesterolemia, diabetes mellitus, chronic renal disease), atrial fibrillation/atrial flutter, actual pharmacotherapy, indications for ICD implantation.

We evaluated the factors that might be of potential prognostic value for ventricular arrhythmia and an ES: the location of myocardial infarction (anterior/inferior/other), the extent of coronary artery disease (single or multi-vessel), any previous coronary revascularization and the type of revascularization (surgical/percutaneous), revascularization of the infarct-related artery, left ventricular ejection fraction and left ventricular end diastolic diameter (LVEDD) using echocardiography, the width of the QRS complex and heart rate in a resting electrocardiogram, the quantity of number of ventricular arrhythmic events and the average heart rate in 24-hour ECG Holter monitoring.

We analyzed all arrhythmic events that had been recorded in the ICD's memory. Patients were followed up at 3- to 6-month intervals after the ICD implantation. Clinical evaluation and device testing were carried out at each follow-up visit. Information about arrhythmic events, the device's current parameters and the current pharmacotherapy were noted during each visit.

Each case of ES was considered individually. All available methods, including changes in the pharmacological and non-pharmacological treatment, were used. Crucial decisions concerning coronary revascularization and ICD programming were made individually for each patient by the same team.

None of the patients died during an acute incident of ES. Two of the patients in the 50 ES (+) group had percutaneous coronary intervention (PCI) during follow-up. Among the 50 ES (+), 15 patients had RF ablation; 4 patients had more than one RF ablation. There was no correlation be-

tween RF ablation and survival. We did not analyze the impact of ablation on the recurrence of VT. During the follow-up none of the patients had an upgrade to cardiac resynchronization therapy (CRT).

### Implantable cardioverter-defibrillator implantation and programming

Devices were implanted in the left subclavicular area. Device cans were placed on the surface of the pectoralis major muscle. Transvenous electrodes were equipped with one or two coils and had either an active or passive fixation. Electrodes were implanted at the right ventricular apex. Commercially available devices (Biotronik: Phylax AV Phylax XM AH, Phylax O6 AH, AH Mycrophylax, Tachos DR and Medtronic: Micro Jewel II, GEM II VR, GEM II DR) were implanted in the study population.

Devices were programmed for two detection zones (VT and VF):

- VT zone at a heart rate of 150/min or 10–20 bpm fewer than the previously documented ventricular arrhythmia, 25–30 intervals in the zone required for detection;
- VF zone at 200/min or more, 16–18 intervals in the zone required for detection.

Antitachycardia pacing in the VT zone was followed by shock therapy if pacing did not terminate the arrhythmia. In the VF zone, shocks of 30–40 J were programmed. In most cases atrial discriminators for supraventricular arrhythmias were turned on.

All of the devices were programmed in the same way (single center study, the same management in all patients). Each episode of arrhythmia was analyzed by experienced staff through the assessment of IEGM recordings. Each episode of arrhythmia was verified with regard to the adequacy of the therapy that was delivered. Inadequate interventions were not taken into account.

### Statistical analysis

Statistical analysis was performed using Statistica 6 software. A *p*-value of less than 0.05 was considered statistically significant. The Shapiro-Wilk test was used to verify whether the variable had a normal distribution. The basic characteristics of the groups were analyzed using the ANOVA test, the Mann-Whitney *U* test and the  $\chi^2$  test. A logistic regression model was used to predict the outcome of dependent variables. The Hosmer-Lemeshow goodness-of-fit test for the logistic regression was performed. Survivals were estimated using the Kaplan-Meier method and compared with a log-rank test. A proportional hazard Cox model was performed to evaluate the

effect of an independent variable on the risk of death.

### Results

#### Clinical characteristics from the period before the ICD implantation procedure

A comparison of the ES (+) and ES (–) groups did not show significant differences in age, NYHA class, concomitant diseases, the incidence of atrial fibrillation/flutter or the type of implanted device. VF in the history as an indication for ICD implantation was more frequent in the ES (–) than in the ES (+) group (27% vs. 6%, *p* = 0.003). An analysis of the pharmacotherapy showed significantly higher use of statins in the ES (–) group (53% vs. 32%, *p* = 0.034) (Table I).

#### Factors of potential prognostic value for an ES

Our analysis showed that patients in the ES (+) group had less frequent revascularization than subjects in the ES (–) group (36% vs. 56%, *p* = 0.049) as well as in relation to the infarct-related artery (patent or bypassed infarct-related artery) – 30% vs. 51%, relatively; *p* = 0.034. Moreover, in the ES (+) group, myocardial infarction was more frequently located at the inferior wall (46% vs. 21%, *p* = 0.007) (Table II).

#### Arrhythmic events during follow-up

We recorded and analyzed a total of 3,408 episodes of ventricular arrhythmias that required the intervention of an ICD. There were 3,148 episodes in the ES (+) group and 260 in the ES (–) group. ATP was successful in 2,302 (67%) episodes – 2,141 (68%) in the ES (+) group and 161 (62%) in the ES (–) group. Shock was required in 1,106 (33%) episodes – 1,007 (32%) in the ES (+) group and in 99 (38%) in the ES (–) group. Five patients in the ES (–) group had no intervention during follow-up. In the ES (+) group, there were 187 incidents of an ES – in 159 cases the therapy consisted of ATP and shocks and in 28 cases ATP alone was sufficient. In the ES (+) group the mean number of electrical storms was  $3.74 \pm 3.27$ , the median 3, range: 1–15. The average number of arrhythmic events during a storm was 16.8 18.6, median: 6, range: 3–140. The average interval between MI and device implantation was  $125 \pm 87$  months. The average time from implantation to the first occurrence of an ES was 400 days, median 172 days.

The average cycle length of an arrhythmia in the entire study population was  $347 \pm 59$  ms (median: 347 ms). We found a statistically significant difference in the length of a cycle between the ES (+) and ES (–) groups ( $358 \pm 54$  ms vs.  $327 \pm 62$  ms, *p* = 0.023; median 365 ms vs. 330 ms, respectively).

**Table I.** Clinical characteristics from the period before ICD implantation's procedure

Parameter	Study population (n = 97)	ES (+), n = 50	ES (-), n = 47	P-value
Age [years]	60.8 ±9.4 (40–79)	60.7 ±9.1 (40–79)	60.8 ±9.8 (44–78)	0.95
Gender male, n (%)	88 (91)	45 (90)	43 (91)	0.86
NYHA I, n (%)	19 (19.6)	11 (22)	8 (17)	0.53
NYHA II, n (%)	50 (51)	23 (46)	27 (59)	0.79
NYHA III, n (%)	27 (28)	16 (32)	11 (24)	0.38
Arterial hypertension, n (%)	61 (62)	33 (66)	28 (59)	0.47
Hypercholesterolemia, n (%)	50 (52)	28 (56)	22 (47)	0.37
Diabetes mellitus, n (%)	11 (11)	4 (8)	7 (15)	0.28
Chronic kidneys disease, n (%)	7 (7)	4 (8)	3 (6)	0.70
Atrial fibrillation/flutter, n (%)	14 (14)	5 (10)	9 (19)	0.20
Dual-chamber ICD, n (%)	6 (6)	1 (2)	5 (11)	0.07
Pharmacotherapy:				
Amiodarone, n (%)	64 (66)	33 (66)	31 (66)	1.0
Sotalol, n (%)	11 (11)	8 (16)	3 (6)	0.12
β-Blocker, n (%)	70 (72)	33 (66)	37 (79)	0.15
Statins, n (%)	41 (42)	16 (32)	25 (53)	0.034
ACE inhibitors, n (%)	86 (89)	46 (92)	40 (85)	0.28
Aspirin, n (%)	91 (94)	49 (98)	42 (89)	0.07
Indication for ICD				
VF, n (%)	16 (17)	3 (6)	13 (27)	0.003
VT, n (%)	49 (50)	27 (54)	22 (47)	0.49
VF + VT, n (%)	32 (33)	20 (40)	12 (26)	0.14

**Table II.** Comparison of potential prognostic factors for ES in the study groups

Variable	ES (+), n = 50	ES (-), n = 47	P-value
Myocardial infarction of anterior wall, n (%)	26 (52)	36 (77)	0.01
Myocardial infarction of inferior wall, n (%)	23 (46)	10 (21)	0.007
Another location of myocardial infarction, n (%)	1 (2)	1 (2)	0.97
Single vessel coronary artery disease, n (%)	12 (24)	13 (28)	0.65
Multivessel coronary artery disease, n (%)	38 (76)	34 (72)	0.65
Previous coronary revascularization – surgical, n (%)	12 (24)	20 (43)	0.051
Previous coronary revascularization – percutaneous, n (%)	10 (20)	10 (21)	0.90
Absence of coronary revascularization, n (%)	28 (56)	17 (36)	0.049
Patent or bypassed infarct-related artery, n (%)	15 (30)	24 (51)	0.034
LVEF (%)	35.2 ±10.2	36 ±10.4	0.70
LVEDD [mm]	63.8 ±6.8	62.2 ±6.3	0.23
QRS width [ms]	120 ±42	115 ±32	0.51
Mean HR in resting ECG [min <sup>-1</sup> ]	68 ±7	70 ±8	0.19
Mean HR in 24-hours Holter [min <sup>-1</sup> ]	67 ±11	69 ±11	0.37
PVC > 10/h, n (%)	26 (55)	24 (48)	0.49
PVC > 30/h, n (%)	17 (34)	17 (36)	0.83
nsVT, n (%)	24 (48)	18 (38)	0.32

For the overall population, the average time until the first occurrence of intervention was 305 ±438 days (median: 140 days) and was longer in the ES (-) group than the ES (+) group (537 ±582 days, median: 314 days; and 166 ±238 days, median: 77 days, respectively). These differences were statistically significant ( $p = 0.0001$ ) (Figure 1).

### Logistic regression model

Multivariate logistic regression analysis showed that myocardial infarction of the inferior wall (RR = 3.98, 95% CI: 1.52–10.41) and the absence of coronary revascularization (RR = 2.92, 95% CI: 1.18–7.21) are independent predictors of an ES ( $p = 0.0014$ ). In the Hosmer-Lemeshow goodness-of-fit test,  $p = 0.96$ .

### Risk of death

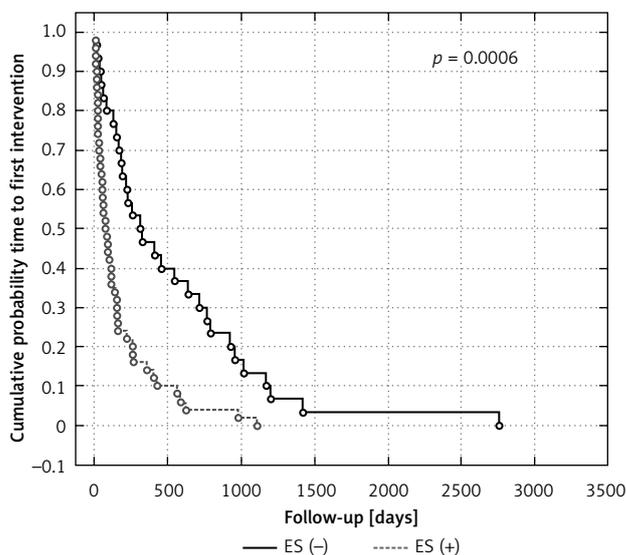
During the long-term observation of 97 patients, there were 39 (40%) deaths. In the ES (+) group 25 subjects (50%) died and in the ES (-) group 14 (30%) subjects died. The Kaplan-Meier survival curves showed that patients in the ES (+) group had a higher cumulative mortality than patients in the ES (-) group;  $p = 0.036$  (Figure 2). The multivariate proportional hazard model by Cox identified the following independent predictors of death ( $p < 0.001$ ) – the occurrence of an ES (HR = 1.93), older age (HR = 1.06), and lower LVEF (HR = 0.95).

### Type of therapy and risk of death in ES (+) group

In the ES (+) group, there was a correlation between the type of therapy administered (ATP vs. shocks) and survival. Among the patients who were treated with ATP alone, 2 of them (20%) died, and this number was significantly lower than in the patients who were treated with high-energy therapy – 23 (57%) subjects died;  $p = 0.033$ . In comparison to the ES (-) group, survival in the subpopulation of the ES (+) group treated with ATP alone was similar (Figure 3).

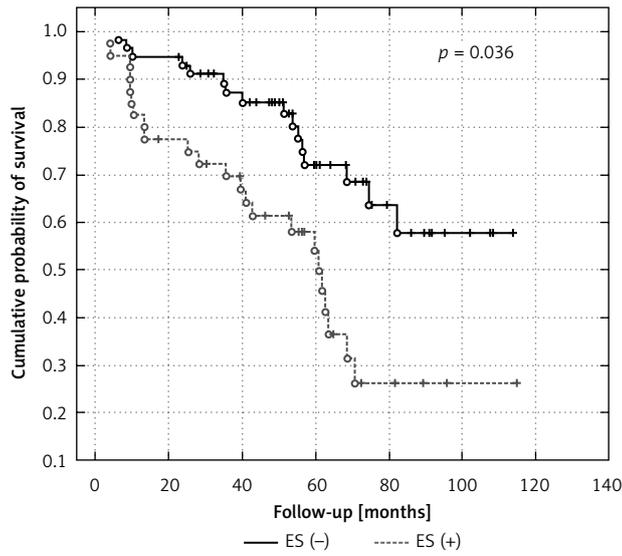
### Discussion

Our study presents the factors that predispose a patient to an ES and the clinical significance of an ES in patients after myocardial infarction with an implanted ICD for the secondary prevention of SCD. The study population comprised a relatively large, homogeneous (in terms of etiology and indications for ICD implantation) group of patients. During a few years of follow-up, an ES occurred in 12% of the patients, and this prevalence is similar to the results described in the literature [10–12]. During a few years of follow-up, an ES occurred in 12% of the patients. This prevalence is similar to the results described in the literature between 1997 and 2004 [10–12], when the study was carried out. Nowadays, widespread use of coronary interventional treatment is likely



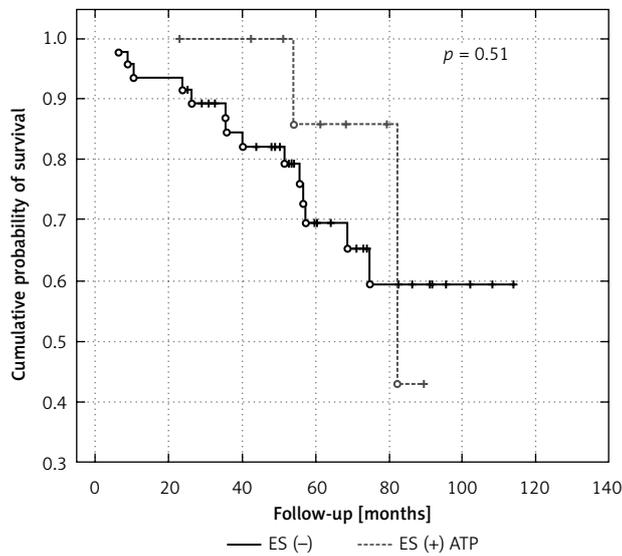
Cumulative percent time to first intervention ES (-)									
100.00	50.00	36.66	23.33	6.66	3.33	3.33	3.33	3.33	3.33
Number of patients ES (-)									
30	15	11	7	2	1	1	1	1	1
Cumulative percent time to first intervention ES (+)									
100.00	16.00	6.00	4.00	0	0	0	0	0	0
Number of patients ES (+)									
50	8	3	2	0	0	0	0	0	0

Figure 1. Kaplan-Meier survival curves for time to first adequate ICD intervention in ES (+) group and ES (-) group



Cumulative percent time of survival ES (-)										
100.00	93.62	89.31	82.44	79.78	69.60	59.66	59.66	59.66	59.66	59.66
Number of patients ES (-)										
47	44	41	34	27	17	9	7	3	0	0
Cumulative percent time of survival ES (+)										
100.00	82.00	77.90	73.57	68.75	46.87	37.50	29.99	29.99	29.99	29.99
Number of patients ES (+)										
50	41	37	33	26	11	7	2	1	1	1

Figure 2. Kaplan-Meier survival curves for ES (+) group and ES (-) group



Cumulative percent time of survival ES (-)										
100.00	93.62	89.26	82.31	79.56	68.95	59.10	59.10	59.10	59.10	59.10
Number of patients ES (-)										
47	44	40	34	25	17	9	7	4	1	1
Cumulative percent time of survival ES (+) ATP										
100.00	100.00	100.00	100.00	87.50	87.50	87.50	43.75	0	0	0
Number of patients ES (+) ATP										
10	10	9	9	6	4	3	0	0	0	0

Figure 3. Kaplan-Meier survival curves for patients treated with ATP alone in the ES (+) and ES (-) groups

to reduce the incidence of ES in the population of patients with ICD.

We identified the factors which predispose a patient to the occurrence of an ES – a previous myocardial infarction other than at the anterior wall and the absence of coronary revascularization. On the other hand, the occurrence of an ES was a negative prognostic factor for death, especially in elderly patients and patients with impaired left ventricular systolic function.

In the study group, the most common indication for ICD implantation was a history of sustained ventricular tachycardia, and the presence of ventricular fibrillation was less frequent. In total, we analyzed more than 3,400 incidents of ventricular arrhythmias. The median heart rate during tachyarrhythmias was 170/min. What is important is that an ES was less frequent in patients with a history of VF as the indication for ICD implantation. Ventricular tachycardias were slower in the ES (+) group than in the ES (–) group (167/min and 183/min, respectively). Whether a history of VT alone (without VF) contributes to the occurrence of an ES at the same level as in patients with VF is still an unresolved problem. Some studies have presented results that are similar to ours [10, 13, 14].

It seems that the underlying etiology and transient factor are the main determinants of the nature of an arrhythmia. The above-mentioned factor, triggering VF, has a temporary effect, only under certain conditions. The re-entry loop for VF is unstable electrically. A post-infarct scar in ischemic cardiomyopathy predisposes a patient to a stable, re-entry VT. Therefore, the majority of ES include slow VT. The effect of antiarrhythmic drugs also plays an important role by modifying the electrophysiological substrate and the frequency of VT.

Half of the patients from the ES (+) group had an electrical storm in the first 6 months after ICD implantation. Some authors have reported the average time from implantation to the occurrence of an ES of between 133 and 270 days. In their studies, no relationship between ICD implantation and the severity of arrhythmia was found [10–12]. In our study we found that the time until the first adequate ICD intervention was significantly shorter in patients in the ES group (+) than in patients with single, isolated arrhythmias. A similar relationship was described by Villacastin *et al.* in one of the first studies about ES in patients with ICD, but those results were not confirmed by subsequent researchers [14]. Only in the subanalysis of the MADIT II study was it found that single, isolated episodes of VT/VF are predictors of an ES. It seems that a single VT/VF (especially VT) episode that requires an ICD intervention that occurs shortly after ICD implantation may be a risk factor for an ES [15].

## Risk factors for an electrical storm

Our study demonstrated that myocardial infarction of the inferior wall and the absence of coronary revascularization are independent risk factors for an electrical storm. The number of patients who underwent CABG was significantly lower in the ES (+) group than in the ES (–) group. Similarly, patients who had either a patent or bypassed infarct-related artery were less frequent in the ES (+) group.

It seems that the location of infarct scar tissue within the inferior wall predisposes a patient to a re-entry ventricular tachycardia and is strongly influenced by the autonomic nervous system [16, 17]. A partial loss of innervation during MI leads to an imbalance between the sympathetic and parasympathetic tone of the autonomic nervous system [18, 19]. The Purkinje fibers system can also play a role in the creation of the VT re-entry circuit in patients after inferior MI [20].

Pascale *et al.* [21] reported on a population of 252 patients with ischemic cardiomyopathy who were eligible for ICD implantation. They selected a group of post-MI patients with recurrent ventricular tachycardia which was dependent on the infarct scar. Patients with a history of MI of the inferior wall constituted 81% [21].

Myocardial ischemia is one of the factors that lead to malignant ventricular arrhythmias and cardiac arrest. Coronary revascularization, which is performed in acute coronary syndrome as well as in patients with stable angina and multi-vessel coronary artery disease, together with optimal medical therapy, is associated with a significant reduction in the risk for an SCD in short- and long-term follow-ups [22–24]. The patency of the infarct-related artery results in increased density of capillaries and delayed cardiomyocyte apoptosis in the infarct and periinfarct zones. These factors are responsible for the limitation of the infarct scar size and the reduction in left ventricular remodeling [25–27].

Heart failure and a reduced left ventricular ejection fraction (LVEF) are two of the most important risk factors for an SCD. Their significance has been proven in a number of clinical trials (MADIT, MADIT II, SCD-HeFT) [28–30].

In our study, there was no effect of HF and LVEF on the risk of occurrence of an ES. In the ES (+) group, there were more patients in the NYHA III class, but the difference was not statistically significant. However, some authors consider heart failure as a possible cause of an ES [31]. Among all of the cases of an ES, decompensation of heart failure was found in 10–20% of patients [13, 32]. There are conflicting literature data regarding a possible relation between LVEF and ES. Several studies have confirmed that a lower LVEF constitutes an inde-

pendent factor for ES [9, 10, 32], while others have published opposite results [11–13]. Standard limitations of echocardiography in the assessment of LVEF as well as shock-related transient systolic dysfunction may influence these conflicting findings.

Atrial fibrillation (AF), especially permanent AF, is associated with higher rates of mortality and ICD discharge (appropriate and inappropriate) [33, 34]. In our study, we found no statistically significant difference in the number of AF patients in ES (+) and ES (–) groups.

The study groups did not differ in pharmacotherapy, except for the use of statins. Statins were recommended more often in the ES (–) group. The difference may be caused by the small number of people who were receiving statins in the entire study population. The low usage of statins is mainly due to economic reasons, but it is also due to the fact that hypolipidemic drugs were only becoming important in the treatment of coronary artery disease at the time of the study.

#### Prognostic role of an electrical storm

During the more than five years of follow-up, we found a significant difference in mortality between the ES (+) and ES (–) groups. In a multivariate Cox analysis, an ES was shown to be related to a lower survival rate. An ES increases the risk of death 1.9-fold. This observation is in accordance with the literature data [11–13, 35]. In the first meta-analysis on this topic, an ES is associated with a three-fold increased risk of death [9] in whole populations of patients with both ischemic and non-ischemic etiology of cardiac arrhythmia.

Apart from ES, older age and reduced LVEF were related to higher mortality in our population.

A possible contribution to the higher mortality could come from the shocks themselves, and we attempted to determine the influence of the type of therapy administered by an ICD (ATP or shocks) on the long-term prognosis.

Sixty-seven percent of all ventricular arrhythmias were successfully treated with ATP, but only 14% of electrical storms did not require high-voltage therapy. We found that there is a better prognosis for patients in the ES (+) group if ATP is successful. The survival curve for this subpopulation of patients is similar to the ES (–) group. The vast majority of ventricular incidents disappeared after the first therapy. Recently, some studies have shown a better prognosis if arrhythmias are resolved using ATP, and therefore the number of high-voltage therapies is reduced [36, 37]. In some cases, the ineffectiveness of ATP may be due to the focal nature of the arrhythmia [38].

We did not evaluate the impact of inappropriate shocks on the risk of death in patients with an

implanted ICD. However, a subanalysis of MADIT II and SCD-HeFT showed that inadequate therapy is also associated with a higher risk of death [39, 40]. The mechanism of the harmful effect of numerous discharges on the myocardium still remains unclear and requires further investigations. Increased troponin levels reflect damage to the myocardium. Moreover, ventricular arrhythmia alone has a negative influence on the myocardium [41, 42]. The total number of VT/VF episodes was much greater in the ES (+) group. Recurrent ventricular arrhythmia affects the metabolism of cellular calcium, which leads to the accumulation of calcium within the myocyte and apoptosis. An ES causes  $Ca^{2+}$ /calmodulin-dependent protein kinase II activation and phospholamban dephosphorylation, which can explain the vicious cycle of arrhythmia promotion and mechanical dysfunction that characterizes ES [43, 44].

This mechanism impairs the left ventricle and increases the risk of recurrence of a ventricular arrhythmia. Death is most often not sudden, but results from the aggravation of heart failure [45].

The study is a retrospective analysis of data collected over a very long period. Due to the lack of data regarding the direct causes of death, the study only analyzed total mortality. The history of arrhythmic events was carried out only after ICD implantation, and was primarily based on the memory in the ICDs. The results of all of the examinations (echocardiography, 24-hour Holter recording and coronary angiograms) were interpreted by different researchers. Moreover, different machines were used to perform particular examinations. We did not analyze any inadequate therapies that were delivered by an ICD. The long period between the observation and the manuscript preparation limited the interpretation of our results. However, the limited number of reports on this topic and the final results prompted us to write it. In our opinion, changes in guidelines of treatment of coronary artery disease influenced the incidence of ES in the general population. The impact on prevalence in ES (+) and ES (–) remains the same, due to the fact that both groups were treated in the same way. Pharmacological treatment when the study was performed (1997–2004) and pharmacological treatment currently being administered do not differ significantly. In recent years, no new antiarrhythmic drugs have been introduced to the treatment of ventricular arrhythmias. In the cardiology center where the follow-up was performed, each patient with an ES was evaluated for eligibility of RF ablation of ventricular arrhythmias. During an incident of ES, each patient was evaluated for ACS, and in most of them control coronary angiography was performed. Therefore, it seems that treatment ap-

plied during the study (1997–2004) does not differ significantly from the current one.

In conclusion, an electrical storm was found in 12% of the patients after myocardial infarction with an ICD for the secondary prevention of SCD.

Our study showed that myocardial infarction of the inferior wall and the absence of coronary revascularization are independent risk factors for the occurrence of an ES. A patent or infarct-related artery and coronary revascularization reduce the risk of occurrence of an ES.

An electrical storm, especially one that results in a large number of shocks, together with older age and a reduced LVEF, is an independent predictor of death in patients after myocardial infarction with an implanted ICD for secondary prevention. Effective ATP therapy may reduce the risk of death among ES patients.

### Acknowledgments

Study data have been accepted and presented at the Congress of Heart Rhythm Society in 2009 in Boston, USA and the Congress of European Heart Rhythm Association EUROPACE in 2011 in Madrid, Spain.

### Conflict of interest

The authors declare no conflict interest.

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