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## Electrical Storm

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### 1. Introduction

Electrical storm (ES) is usually defined as a clustering of destabilizing episodes of ventricular tachycardia or ventricular fibrillation in a short period of time, requiring multiple cardioversions or defibrillations. Many criteria have been proposed to define ES since the early 1990s, when the term was first introduced to indicate a state of electrical instability with several ventricular tachycardias (VT) or ventricular fibrillations (VF) over a few hours. At present an official and widespread definition of ES is not available. ES has been defined differently, from two or more to twenty or more episodes within 24 hours. Some definitions were based on number of hemodynamically unstabilizing episodes, others relied on number of shocks needed (or just delivered, whether appropriate or not), and some others on time between each single episode. More imaginative definitions include “VT recurring immediately after termination”, “VT for an half of each of three days” and “VT resulting in more total ventricular ectopic beats than sinus beats in 24 hours” (Israel et al, 2007). Fortunately, nowadays most cardiologists define ES as recurrent VT or VF, at least 3 in a 24 hours period, as this is probably the most appropriate and solid definition. This definition does not include the presence of haemodynamic instability, since the intervention of modern implanted cardioverter-defibrillator (ICD) usually terminates the arrhythmia before its clinical and hemodynamical consequences.

Similarly to definitions, many names have been used as synonymous to ES, such as arrhythmic storm, recurrent short-term ventricular arrhythmia, VT clusters and electrical instability. In this chapter we will use the term ES, as the most used and widespread in clinical practice.

ES is more frequently seen as an acute complication of myocardial infarction, as a not-so-uncommon adverse event in ischemic and non ischemic dilated cardiomyopathy and in genetic arrhythmia syndromes, such as congenital long QT syndrome and Brugada syndrome. However, the highest incidence of ES is reported in heart failure patients with ICD. This incidence, which ranges from 4% to 60% according to different studies, is mainly due to two factors. First, ICD can detect the VT or VF underlying a clinical episode of dizziness, syncope or even aborted sudden cardiac death, thus making the diagnosis far easier. Second, and most important, it can effectively treat the first and second VT or VF, thus saving the patient from sudden cardiac death and making him able to withstand more arrhythmic episodes.

The current chapter will summarize the current epidemiology and diagnosis of electrical storm, as well as give some insight on current recommendation regarding pharmacological and non pharmacological treatment.

2. Epidemiology of electrical storm

2.1 Incidence of electrical storm and its different causative arrhythmias

In the last twenty years, several studies were carried out to determine the incidence of ES (table 1).

Author	Year	Definition	Population	%
Wood	1995	$\geq 3$ VT/VF $\leq 24$ h	ICD (secondary prevention)	10
Kowey	1996	$\geq 2$ VT/VF $\leq 24$ h	All ES patients	-
Villacastin	1996	$\geq 2$ shocks for VT	ICD (secondary prevention)	20
Fries	1997	$> 2$ VT $\leq 1$ h	ICD (secondary prevention)	60
Credner	1998	$\geq 3$ VT/VF $\leq 24$ h	ICD (secondary prevention)	10
Nademanee	2000	$\geq 20$ VT/VF $\leq 24$ h $\geq 4$ VT/VF $\leq 1$ h	All ES patients	-
Greene	2000	$\geq 3$ VT/VF $\leq 24$ h	ICD (secondary prevention)	18
Bansch	2000	$\geq 3$ VT/VF $\leq 24$ h	ICD (secondary prevention)	28
Exner	2001	$\geq 3$ VT/VF $\leq 24$ h	ICD (secondary prevention)	20
Verma	2004	$\geq 2$ VT/VF $\leq 24$ h	ICD (secondary prevention)	10
Arya	2005	$\geq 3$ VT/VF $\leq 24$ h	ICD (30% primary prevention)	13
Brigadeau	2006	$\geq 2$ VT/VF $\leq 24$ h	ICD (secondary prevention)	58
Honhloser	2006	$\geq 3$ VT/VF $\leq 24$ h	ICD (secondary prevention)	23
Sesselberg	2007	$\geq 3$ VT/VF $\leq 24$ h	ICD (primary prevention)	4
Nordbeck	2010	$\geq 3$ VT/VF $\leq 24$ h	ICD (55% primary prevention)	7
Streinert	2011	$\geq 3$ VT/VF $\leq 24$ h	ICD (81% primary prevention)	7

Table 1. Incidence of electrical storm according to different definitions and populations.

However, due to different definitions of ES and different populations, results vary widely. Most of the evidences come from ICD populations implanted after sustained ventricular arrhythmias, in which ES incidence ranges from 10 to 60 %. ICD recipients implanted for primary prevention, either with underlying ischemic or idiopathic dilated cardiomyopathy, have a lower incidence of ES (from 4 to 7%).

Little is known about incidence after acute myocardial injury, except that is more common in acute ST-elevation myocardial infarction (STEMI), when STEMI is associated with an important left ventricular dysfunction and when reperfusion therapy (either fibrinolysis or percutaneous angioplasty) is late or ineffective. Similarly, exact incidence and prevalence of ES in genetic arrhythmic syndromes such as Brugada syndrome, familiar long QT syndrome, short-coupled variant of torsade de pointes and right ventricular arrhythmogenic cardiomyopathy is still unknown. Albeit they are all rare conditions, patients afflicted by genetic arrhythmic syndromes are typically young and otherwise healthy subjects, therefore in immediate need for ES primary prevention. More than 8 out of 10 arrhythmic episodes constituting ES are monomorphic VT (mVT), with polymorphic VT (pVT) and VF far behind in prevalence (Table 2).

Author	Year	mVT (%)	pVT+VF (%)
Credner	1998	71	29
Greene	2000	97	3
Bansch	2000	91	8
Exner	2001	86	14
Verma	2004	52	48
Brigadeau	2006	90	10
Honhloser	2006	91	9
Sesselberg	2007	78	22
Nordbeck	2010	85	15
Streinert	2011	73	27

Table 2. Prevalence of different ventricular arrhythmia types in ES.

Sustained monomorphic VT is usually related to structural heart disease. Monomorphic VT self-propagation is made possible by a reentry mechanism around a fixed anatomic barrier, which is often represented by a myocardial scar from a previous MI. The same ischemic cardiomyopathy, as it progresses, leads to fibrosis which in turn leads to conduction slowdowns, creating the perfect pathway for reentrant VT. In this particular setting an otherwise harmless trigger such as a premature ventricular contraction is necessary to start monomorphic VT. Monomorphic reentrant VT does not require active ischemia as a trigger, and is an uncommon cause of ES in acute myocardial infarction. VF and pVT are due to different mechanisms, comprehending multiple activation of different ventricular foci (pVT) or chaotic activation of the whole endocardial ventricular surface (VF). These two arrhythmias are more common in acute myocardial infarction or

when a long QT interval is present. Therefore QT interval should be assessed in all patients with ES due to pVT or VF as soon as sinus rhythm has been restored. Acquired causes of long QT, such as hypokalemia, hypocalcemia, hypomagnesemia, hypothyroidism and medication known to prolong QT interval, should be corrected as soon as possible. Inherited causes of long QT should be suspected whether an acquired cause cannot be found.

## **2.2 Short and long-term prognosis of electrical storm**

Available data strongly suggest that patients who experience an ES have a poor outcome. Only a few trials were inconclusive regarding the role of ES as a mortality marker, and those usually had a wider definition of ES or a shorter follow-up. In secondary prevention populations such as the one of the AVID trial (Exner et al, 2001), patients with ES had a higher risk for non-sudden cardiac death (OR 2.4). In a substudy of the MADIT-II trial, which enrolled primary prevention patients, the risk of death was even higher (OR 7.4). Consistent findings suggest that the increase in mortality is mainly due to the worsening of ventricular dysfunction leading to end stage heart failure, with only a small proportion of sudden cardiac or other deaths (Gatzoulis et al, 2005). This hypothesis is also supported by the fact that risk of death after ES reaches its peak around 2-3 months after the acute event.

Another main clinical consequence of ES is hospitalization, which is required for a proper treatment in approximately 80% of patients (Bänsch et al, 2000). Hospitalization rates grow higher with each shock delivered, reaching 100% if 3 or more shocks are needed. Hospitalization is in turn associated with a poorer quality of life and higher costs.

Patients who experience an ES are more likely to have multiple ES over time. Recent data showed that recurrence of ES happens in more than half of all patients with ES, and is more common within the first year after the original ES episode (Streitner et al, 2011).

## **2.3 Pseudo-storm**

Sometimes multiple recurrent ICD discharges are not associated with ES but are due to device malfunctioning. Pseudo-storm is defined as recurrent inappropriate ICD discharges over 24 hours. Far from being a minor complication, pseudo-storm is usually physical and psychological harmful and potentially lethal. The most common causes of inappropriate ICD shock include supraventricular tachycardia with high ventricular response and oversensing of peaked T waves, myopotentials or electrical noise (Gradaus et al, 2003).

Recurrent ICD shocks can cause myocardial injury by direct electrocution cell injury and by activation of signaling pathways in the molecular cascade of HF, the most important of all being adrenergic neurohormonal system. Adrenergic hyperactivity may then synergize with recurrent ventricular arrhythmias in exacerbating ventricular dysfunction and worsening heart failure. In a recent paper on a typical ICD population, Sweeney et al. demonstrated that electrical shocks were associated with an increased risk of death independently of underlying ventricular arrhythmia (Sweeney et al, 2010). Authors esteemed that for every delivered shock, whether appropriate or not, the risk of death increases by 20%. On the other hand, no increased risk was associated with anti-tachycardia pacing (ATP) therapies. Pseudo-storm does not only cause myocardial damage, but can deplete a full device battery within hours, potentially leaving the patient unprotected from life-threatening arrhythmic events. Although a very rare complication, fatal arrhythmias actively caused by pseudo-storm are possible. Messali and coworkers

described a patient who, three months after an ICD replacement, received six consecutive shocks related to detection of noise interpreted as VF. Unfortunately, the sixth shock triggered a true VF, which was not treated due to the end of the therapeutic sequence, and which led to the patient's death (Messali et al, 2004).

Pseudo-storm should be treated by immediate intervention to suppress ICD shocks. Moreover, inappropriate discharges from ICD should be avoided at all cost by an optimal device programming. ATP therapy should be preferred over shock in the therapeutic sequence, due to its more favorable risk profile.

### **3. Clinical presentation of electrical storm**

#### **3.1 Electrical storm in acute myocardial infarction**

Electrical storm, when present, is often the initial manifestation of ischemia and usually starts in the first 48 hours of an ST-elevation MI. In that case, pVT is almost always the ventricular arrhythmia underlying ES. Ischemia and adrenergic activation increase Purkinje cell automaticity, thus letting multiple spontaneous firing. Necrosis, altered myocyte membrane potential, electrolytic imbalance and even reperfusion damage can all contribute to increase dispersion of refractory periods between epicardium and endocardium, thus facilitating the propagation of multiple reentry waves.

ES is a strong negative prognostic factor in acute myocardial infarction. In one study where ES was defined as  $\geq 20$  VT or VF episodes/day or  $\geq 4$  VT/VF episodes/hour, one-week incidence of non-sudden death was as high as 50%. The majority of patients who survived the acute phase, however, have a prognosis relatively comparable with other ES populations. Along with pharmacological therapy, swift and effective reperfusion is crucial to end arrhythmic episodes. Retrospective studies and registry data suggest the hypothesis that, with modern reperfusion therapy currently available, the incidence of ES could be associated with reperfusion time.

#### **3.2 Electrical storm in heart failure**

Heart failure is an important risk factor for ES. Most of the times, idiopathic dilated cardiomyopathy is the structural heart disease underlying heart failure in ES patients, followed in prevalence by ischemic cardiomyopathy (Gasparini et al, 2008). Hypertrophic cardiomyopathy and valvular cardiomyopathy, albeit uncommon, has been associated with heart failure worsening leading to ES (Credner et al, 1998).

Left ventricular ejection fraction (LVEF) is the most widespread and easy to obtain marker of systolic dysfunction and has been used to test the association between heart failure and ES. Several studies have found an altered LVEF as an independent risk factor for ES, along with older age, chronic renal failure, causative arrhythmia and electrolytic imbalances (Exner et al, 2001 and Brigadeau et al, 2006). On the other hand, Streitner and coworkers found that a LVEF lower than 30% was not predictive for the initial ES event, but brought a 2.2-fold increased risk for ES recurrence (Streitner et al, 2011).

Current evidence suggests that, more than the presence or absence of heart failure by itself, it is the progression of heart failure and structural cardiomyopathy the real factor in promoting ES and ES recurrence. Therefore, prevention of further systolic function deterioration after an initial ES is mandatory and must be achieved through optimization of medical and resynchronization therapy.



### 3.3 Electrical storm in patients with congenital pro-arrhythmic diseases

Genetic arrhythmic syndromes or inherited arrhythmic disorders comprise a group of syndromes with unique genetic abnormalities and presentations but with very similar clinical outcomes, the most terrifying of which are life-threatening arrhythmias and sudden cardiac death. Some of them, such as Brugada syndrome and long QT syndrome, affect structural normal hearts, whereas others, such as arrhythmogenic right ventricular cardiomyopathy and left ventricular non-compaction affect deeply the myocardial tissue, and are included in the cardiomyopathies classification. ES has been described in the Brugada syndrome, in the familial long QT syndrome, in the short-coupled variant of torsade de pointes, in catecholaminergic polymorphic ventricular tachycardia, in arrhythmogenic right ventricular cardiomyopathy and in myocardial non-compaction. Overall, these syndromes are quite rare, being Brugada syndrome the most prevalent with 5 cases out of 10.000, and association with ES is even rarer. However, patients affected by these syndromes are typically young and otherwise completely healthy, making diagnosis and treatment of these conditions challenging.

## 4. Laboratory and electrical storm

Although electrolytic imbalances, such as hypokalemia, hyperkalemia and hypomagnesemia are a well known risk factors for ventricular arrhythmias and hence ES, many papers report that an evident trigger in a majority of patients cannot be identified by laboratory alone. Credner et al. underlined the presence of hypokalemia, along with acute coronary syndrome and worsening heart failure as potential triggers in 26% of the patients in his case-records (Credner et al, 1998). Similarly, Bänsch et al. found hypokalemia as a potential cause of ES in 20% of their cohort. According to the SHIELD trial, electrolytic imbalance was responsible for storm triggering in only 4% of patients (Hohnloser et al, 2006).

In 2006 for the first time Brigadeau et al. highlighted a possible role of creatinine in predicting ES, identifying a storm trigger in 36% of the whole cohort. Among most common triggers (such as acute coronary syndrome, high body temperature, hypokalemia or hyperkalemia, hyperthyroidism and acute heart failure) chronic renal failure, identified as a creatinine clearance lower than 60 ml/min, was independently associated with ES occurrence. Thus, this study concluded that the patients with a defibrillator who are likely to undergo electrical storm are those who have both low left ventricular ejection fraction and chronic renal failure (Brigadeau et al, 2006). In a recent MADIT II chronic kidney disease was associated with a 2.1-fold increase in risk for ES in both primary and secondary prevention patient (Sesselberg et al., 2007).

Thyroid disorders (both hypothyroidism and hyperthyroidism) have been suggested as a trigger for ES. However, at present there is scarce evidence of an important role of thyroid hormones in ventricular arrhythmias pathogenesis. In one case report ES was attributed to amiodarone-induced thyrotoxicosis (Marketou et al, 2001). The patient was unresponsive to medical therapy and the ES was successfully terminated by thyroidectomy.

In conclusion, in an ES setting laboratory could play an important role. Electrolytic abnormalities such as hypokalemia, hyperkalemia and hypomagnesemia should be promptly diagnosed and corrected, as potential triggers of arrhythmic events. High serum levels of BNP, creatinine and PCR are valid markers for respectively decompensated heart failure, chronic renal insufficiency and pro-inflammatory state, and should be assessed in every patients experiencing ES as useful stratification tools.

## 5. Electrical storm therapy

### 5.1 Pharmacological therapy

#### 5.1.1 Amiodarone

Amiodarone is widely used in the treatment of ventricular arrhythmias. Intravenous amiodarone appears to be the most effective agent for ES, and may even suppress ventricular tachycardia that recurs despite chronic oral amiodarone therapy. In acute ES, a rapid intravenous amiodarone administration (300 mg or 5 mg/kg rapid push followed by repeated boluses at half the above doses for breakthrough episodes) blocks fast sodium channels in a use-dependent fashion (producing more channel blockade at faster heart rates), inhibits norepinephrine release, and blocks L-type calcium channels without prolonging ventricular refractoriness. On the other hand, prolonged ventricular refractory periods are seen in oral amiodarone therapy over periods ranging from days to weeks. Amiodarone has few negative inotropic effects and is safe in patients who have depressed systolic function. It is in fact the only anti-arrhythmic drug with solid safety evidences in patients with NYHA class III and IV or recently decompensated heart failure. Moreover, the incidence of torsades de pointes is low in such patients despite the potential significant prolongation of the QT interval. Amiodarone efficacy in terminating ES is approximately 60%. When compared with placebo in the ARREST trial, amiodarone improved survival to hospital admission in patients who had a cardiac arrest that involved VF or pulseless VT (Kudenchuk et al, 1999). Amiodarone can be effective even when other agents have been ineffective. Levine and colleagues examined 273 hospitalized patients who had electrical storm that was refractory to lidocaine, procainamide, and bretylium therapy (Levine et al, 1996). When amiodarone was given, 46% of the patients survived for 24 hours without another episode of VT, and another 12% improved after taking amiodarone plus lidocaine or procainamide. Side effects of short-term amiodarone intravenous use are rare. A combination of intravenous amiodarone and propranolol improves survival rates and should be the mainstay of therapy in acute management of ES.

Oral amiodarone is effective as adjunctive therapy to prevent recurrent ICD shocks. The OPTIC (Optimal Pharmacological Therapy in Implantable Cardioverter) study compared amiodarone (200 mg maintenance dose following 6 weeks of loading) plus  $\beta$ -blocker with sotalol (240 mg adjusted for renal function) or  $\beta$ -blocker alone (Connolly et al, 2006). Appropriate shocks were reduced by amiodarone compared with  $\beta$ -blocker therapy only by 70%, inappropriate shocks were reduced by 78%, appropriate shocks and ATP were reduced by 70%, and all-cause shocks excluding the first 21 days were reduced by 82%. The mean number of shocks per year was 4.32 in the beta-blocker only group, 0.93 in the sotalol group, and 0.51 in the amiodarone group. Although long-term amiodarone therapy is usually successful, substantial side effects include pulmonary fibrosis, hypothyroidism, liver toxicity, and corneal deposits. In addition, amiodarone may increase the energy required for successful defibrillation, so patients with ICDs should undergo repeated defibrillation threshold testing. Patients who have episodes of electrical storm despite amiodarone therapy may benefit from  $\beta$ -blockers adjunctive therapy or undergo RF ablation.

#### 5.1.2 $\beta$ -blockers

$\beta$ -blockers play a key role in the management of electrical storm. Their effects were discovered in the 1970s, when they were studied as therapy for acute MI. First evidences regarding a possible role as anti-arrhythmic drugs come from canine models (Anderson et



al, 1983). All  $\beta$ -blockers increased 6-fold the fibrillation threshold and made the animals less susceptible to fibrillation under ischemic and non-ischemic conditions. The improvement was greater with the use of more potent  $\beta$ -blockers and with those that antagonized both  $\beta_1$  and  $\beta_2$  receptors. Although several  $\beta$ -blockers decrease susceptibility to VF, most of the studies have focused on propranolol. Propranolol consistently decreases the incidences of fatal VF during acute MI and sudden cardiac death after MI (Tsagalou et al, 2005). In patients with congestive heart failure, propranolol decreases sympathetic outflow more than does metoprolol, perhaps because  $\beta_2$  receptors prevail in failing hearts (Newton et al, 1996). The lipophilic nature of propranolol enables active penetration of the central nervous system and the blockade of central and prejunctional receptors in addition to peripheral  $\beta$  receptors. Propranolol may effectively suppress an electrical storm even when metoprolol has failed (Tsagalou et al, 2005). Therefore, propranolol, given at a dose of 0.15 mg/kg intravenous bolus over 10 minutes followed by a 3-5 mg dose every 6 hours, is a first line therapy in emergency ES setting. Nademanee and coworkers investigated the efficacy of sympathetic blockade in electrical storm comparing propranolol, esmolol, and left stellate ganglionic blockade to combined lidocaine, procainamide, and bretylium therapy (Nademanee et al, 2000). All their patients have experienced a recent MI and more than 20 episodes of VT within 24 hours or more than 4 episodes per hour. Sympathetic blockade provided a marked survival advantage (78% versus 18% at one week, and 67% versus 5% at one year). Despite the high doses of propranolol, an increase in heart failure progression was not reported in this study, although it is known from previous studies that propranolol can exacerbate heart failure in patients with poor systolic function and use in these patients should be carefully monitored. The short acting intravenous esmolol may also be used but its dosing and titration are somewhat more complicated. Miwa et al. have demonstrated that landiolol, an ultra-short acting  $\beta_1$ -selective blocker is useful as a life-saving drug for amiodarone-resistant ES (Miwa et al, 2010). Landiolol in ES is used intravenously with an initial dose of 2.5  $\mu\text{g}/\text{kg}\cdot\text{min}$ , which can be doubled every 10 minutes if first dose is ineffective, up to a maximum dose of 80  $\mu\text{g}/\text{kg}\cdot\text{min}$ . Landiolol has a shorter plasma half-life (4 minutes) than esmolol (9 minutes) or propranolol (2 hours) and higher  $\beta_1$  selectivity. These properties suggest that adverse respiratory effects, such as bronchial asma, are less likely to develop and to persist with landiolol, which make it more suitable for emergency medical care. When amiodarone was ineffective, landiolol inhibited ES in 33 patients (79%) at a mean dose of 7.5  $\mu\text{g}/\text{kg}\cdot\text{min}$ . All patients in whom landiolol was ineffective died of arrhythmia. Of the 33 patients in whom landiolol was effective, 25 survived and were discharged (60% of all patients). At present, landiolol is not available in most European countries and its safety profile in ES still needs to be assessed, making this drug a promising alternative to propranolol when amiodarone alone is ineffective.

### 5.1.3 Lidocaine

Intravenous sodium-channel blockers (lidocaine, procainamide) are minimally effective in suppressing shock-resistant VT/VF and ES (Credner et al, 1996 and Nademanee et al, 2000). Lidocaine binds to fast sodium channels in a use-dependent fashion. Binding increases under cellular conditions that are common in ischemic VT, such as reduced pH, faster stimulation rate and reduced membrane potential. However, lidocaine has relatively weak antiarrhythmic properties outside the ischemic setting: conversion rates from VT to sinus rhythm range from 8% to 30%. In one study enrolling patients with out-of-hospital, shock-

resistant VT or VF, only 12% of those randomized to lidocaine survived to hospital admission, versus 23% who received amiodarone. On the basis of this and other findings, amiodarone has replaced lidocaine as first line therapy for refractory VT and VF. Actual recommendations suggest intravenous lidocaine only in the treatment of polymorphic VT associated with acute ischemia. If lidocaine is used, it should be administered as an intravenous bolus of 0.5 to 0.75 mg/kg that is repeated every 5 to 10 min as needed. A continuous intravenous infusion of 1 to 4 mg/min maintains therapeutic levels. The maximum total dose is 3 mg/kg over 1 hr.

#### **5.1.4 Procainamide**

Procainamide blocks fast sodium channels in a use-dependent fashion and is metabolized to N-acetylprocainamide, which in turn blocks potassium channels and accounts for much of the antiarrhythmic effect in vivo. When given as a loading dose of 100 mg over 5 min, procainamide is a reasonable choice for terminating monomorphic VT. In patients with depressed systolic function procainamide can cause hypotension or prolong QRS width by more than 50%, either of which would necessitate discontinuation of the drug. Procainamide prolongs the QT interval and therefore could cause torsade de pointes. Its use is contraindicated in patients with impaired renal function, because N-acetylprocainamide is excreted by the kidneys.

#### **5.1.5 Azimilide**

Azimilide is an experimental class III antiarrhythmic drug that blocks calcium channels and prolongs the energy potential and refractory periods. The recently published SHIELD trial showed that azimilide is effective and helps to reduce the number of ICD discharges, though not mortality (Stefan et al, 2006). A secondary analysis of the SHIELD data found that during a prospective one-year follow up azimilide significantly reduced the incidence of ES in comparison with placebo. Azimilide could become an alternative for the treatment of ES whenever it becomes commercially available.

#### **5.1.6 Polypharmacological approach**

Optimization of  $\beta$ -blocker therapy is the first important step, particularly when the electrical storm is triggered by ischemia or increased sympathetic tone. The next step is initiating antiarrhythmic therapy if patient still experiences ES. In the absence of contraindications (such as QT lengthening or polymorphic ventricular tachycardia), amiodarone is generally the antiarrhythmic drug of choice and has been validated in numerous clinical trials (Kowey et al, 1995 and Wood et al, 1995). Most of the time, optimized  $\beta$ -blocker therapy plus intravenous amiodarone will control the electrical storm within 24 to 48 hours. This appears to be the most effective therapy for electrical storm. If the intravenous combination of amiodarone and  $\beta$ -blockers proves inefficacious, the addition of lidocaine is a reasonable option. Although controlled data are not available, combination of antiarrhythmic drug allows lower and better tolerated doses of individual drugs, and offers the potential of synergistic effects.

#### **5.1.7 Sedation**

The physical and emotional stress that patients experience in association with electrical storm and multiple electrical cardioversions increases adrenergic tone and often perpetuates

arrhythmias. All patients who have electrical storm should be sedated. Sedation or general anesthesia are needed in resistant cases where repeated shocks or anti-tachycardia transthoracic pacing are needed. Short-acting anesthetics such as propofol, benzodiazepines, and some agents of general anesthesia have been associated with the conversion and suppression of VT (Burjorjee & Milne, 2002). Left stellate ganglion blockade and thoracic epidural anesthesia have also suppressed electrical storms that were refractory to multiple antiarrhythmic agents and  $\beta$  blockade (Nademanee et al, 2000). These therapeutic approaches directly target nerve fibers that innervate the myocardium, and a reduced adrenergic tone is most likely responsible for the reported efficacy. It is currently unknown whether sedative and anesthetic agents may have direct antiarrhythmic effects.

## **5.2 Implantable defibrillators: First-line therapy and first-line diagnostic?**

ES is quite common in ICD recipients. In different studies the incidence of ES in patients with an ICD ranges from 10 to 60% when ICDs are implanted for secondary prevention (Arya A et al, 2005) and 4% to 7% when ICDs are implanted for primary prevention (Sesselberg et al, 2007). A higher incidence of ES in ICD patients is related to an higher cardiovascular mortality and morbidity among ICD recipients. As previously said the presence of ICD allows the physician to recognize more arrhythmias and the patient to survive the arrhythmia and hence manifest more episodes.

Occurrence of ES after ICD implant has changed over time. A paper from the late 1990s reported the peak of incidence of ES between 4 and 5 months after implant (Credner et al, 1998). On the other hand recent evidences pushed the onset of ES to 26 months after implant (Streitner et al, 2011). Total number of episodes varied over time too: as much as 55 mean episodes per patient have been reported (Greene et al, 2000), whereas more recent papers describe far less VT/VF per single patient (Streitner et al, 2011). Both these findings could be explained by changes in antiarrhythmic therapy and ICD technology occurred in this last decade which in turn led to fewer total arrhythmic episodes and improved overall prognosis.

There is a multitude of etiologies of ES in ICD patients: hypokalemia or other electrolytic imbalance, drugs (diuretics,  $\beta$ -adrenergic drugs, alcohol), ischemia, medication noncompliance and less common causes such as fever or stress (Huang et al, 2005 and Israel et al, 2007). The most common cause of ES in ICD patients is exacerbation of heart failure, as underlying ischemic or idiopathic dilated cardiomyopathy could progress despite medical therapy and arrhythmic prevention. Nevertheless, most ES have no clear etiology and even an exhaustive search for acute cause may prove fruitless.

Clinical presentation can vary dramatically depending on arrhythmias typology (monomorphic VT, polymorphic VT or VF) and patient's characteristics (EF, NYHA class, comorbidities). The presence of syncope associated with arrhythmia depends both on hemodynamic factors and on ICD settings (mostly shock charging time). The most important acute consequence related to ES is hospitalization that is required in more than 80% of patients, particularly when shocks are delivered (Bansch et al, 2000). ICD patients presenting ES have higher morbidity and mortality, hence determination of predictors is needed to identify high risk patients.

Data on risk factors of ES are far from comprehensive or conclusive but most studies consider low EF and secondary prevention as major risk factors for developing ES in ICD recipients. High NT-proBNP and hs-CRP, history of atrial fibrillation before implant or single/dual chamber pacing over CRT are also described as predisposing to ES (Streitner et al, 2011).

ICD patients who experienced a first ES are also more likely to experience one or more ES recurrence. Recurrence rate is as high as 80% within 12 months after the first episode, according to Steinert, who described LVEF < 30%, age > 65 years, chronic obstructive pulmonary disease and lack of ACE inhibitors therapy as independent predictors of ES recurrence (Steinert et al, 2011).

Most authors report poor prognosis associated with ES with a risk of death increased from 1.9 to 17.8 fold. Death rate is usually low during hospitalization and acute episode but increases afterward, particularly during the first year after ES.

Lately, new tools have become available to detect and manage ES in ICD patients. All major ICD companies now offer some sort of home or remote monitoring along with their devices. Home monitoring offers the physician reports for arrhythmic events, device battery and parameters status in real-time. Asymptomatic or lightly symptomatic ES could be then promptly recognized and the patient immediately called in for a check-up without the need to wait for the next programmed ambulatory visit. Figure 1 shows how an ES looks like on auto-generated home monitoring report: the patient experienced 37 arrhythmic episodes in less than 24 hours, 36 VT terminated with a shock after ATP sequence was ineffective and 1 VF episode terminated with ATP.

Figure 2 shows how EGM can be seen and interpreted via remote monitoring. In this EGM VT has been correctly detected and treated by ICD shock.

### 5.3 Is cardiac resynchronization therapy useful in preventing electrical storm?

Cardiac resynchronization therapy (CRT) is a well established therapy for treatment of moderate to severe heart failure. Several studies assessed benefits from biventricular pacing resulting in prevention of left ventricular remodeling and improvement of hemodynamic, ejection fraction, NYHA class, quality of life, morbidity and mortality (Cleland et al, 2005). Effects of CRT on ventricular arrhythmias are less well established. Some evidence suggests that pacing itself might cause arrhythmias and some authors reported an increase in incidence of atrial fibrillations, ventricular arrhythmias or even electrical storms after biventricular pacing (Kantharia et al, 2006). However, currently available large-scale trials showed no significant proarrhythmic effects of CRT. On the other hand, there is no strong evidence of a direct antiarrhythmic effect of CRT over single or dual chamber pacing either. Nordbeck et al. compared incidence of ES in 168 CRT and 561 ICD patients. They found significant lower incidence of ES in CRT group (0.6% versus 7%), suggesting that, beside the well known hemodynamic improvements, cardiac resynchronization therapy may reduce the arrhythmia burden in heart failure patients (Nordbeck et al, 2010). An Italian group found a higher incidence of ES (11.3% vs 5.3%) in patients non responder to CRT therapy, defined as minor improvement in NYHA class and ejection fraction (Gasparini et al, 2008). These data support the hypothesis that CRT may have an indirect antiarrhythmic effect, due to factors which are still unclear. Nordbeck suggests that the reduction of arrhythmic burden could be due to improvement of cardiac output and ejection fraction from CRT, as ejection fraction is a known risk factor for ventricular arrhythmias. Another hypothesis by Kowal found some evidences in clinical cases reporting a specific role of cardiac pacing site in development or suppression of VT. He hypothesized that the mechanism of arrhythmia suppression under biventricular pacing could be ascribed to preexcitation of the area of slow conduction responsible for the reentrant arrhythmia (Kowal et al, 2004).

Nevertheless, electrophysiologic effects of CRT are still poorly understood. Biventricular pacing remains a major therapeutic tool in the treatment of heart failure but additional data are required to assess its efficacy as antiarrhythmic therapy.



To:		
Report	Patient ID: S            P	
from 28-Jul-2011	Device / Device SN:	Patient device SN:
11:45	Lumax 300 HF-T / 60462319	46817123

Recordings

Recordings - Episode list:

No.	Detection time	Type	Details	Predeflection PP/RR	Pretermination PP/RR
84	29-Apr-2011 10:41:41	VT1	ATP: 3; Shocks delivered: 1	772 / 390	835 / 668
83	29-Apr-2011 10:37:42	VT1	ATP: 3; Shocks delivered: 1	772 / 387	816 / 719
82	29-Apr-2011 10:14:07	VT1	ATP: 3; Shocks delivered: 1	773 / 387	845 / 763
81	29-Apr-2011 10:06:14	VT1	ATP: 3; Shocks delivered: 1	770 / 387	922 / 998
80	29-Apr-2011 10:01:45	VT1	ATP: 3; Shocks delivered: 1	787 / 394	795 / 785
79	29-Apr-2011 09:51:22	VT1	ATP: 3; Shocks delivered: 1	787 / 393	801 / 823
78	29-Apr-2011 09:49:28	VF	ATP: 1; ATP One Shot: YES; Shocks aborted: 1	> 1998 / 271	599 / 553
77	29-Apr-2011 09:47:14	VT1	ATP: 3; Shocks delivered: 1	889 / 390	663 / 646
76	29-Apr-2011 09:41:47	VT1	ATP: 3; Shocks delivered: 1	784 / 383	695 / 880
75	29-Apr-2011 09:34:03	VT1	ATP: 3; Shocks delivered: 1	662 / 383	1409 / 917
74	29-Apr-2011 09:20:11	VT1	ATP: 3; Shocks delivered: 1	853 / 379	437 / 654
73	29-Apr-2011 09:04:47	VT1	ATP: 3; Shocks delivered: 1	582 / 387	1049 / 797
72	29-Apr-2011 08:50:27	VT1	ATP: 3; Shocks delivered: 1	786 / 393	> 1998 / 922
71	29-Apr-2011 08:27:16	VT1	ATP: 3; Shocks delivered: 1	672 / 390	555 / 898
70	29-Apr-2011 08:23:31	VT1	ATP: 3; Shocks delivered: 1	638 / 392	1244 / 915
69	29-Apr-2011 08:10:59	VT1	ATP: 3; Shocks delivered: 1	685 / 395	601 / 554
68	29-Apr-2011 06:46:20	VT1	ATP: 3; Shocks delivered: 1	751 / 374	931 / 804
67	29-Apr-2011 06:00:11	VT1	ATP: 3; Shocks delivered: 1	1008 / 405	1225 / 976
66	29-Apr-2011 05:30:13	VT1	ATP: 3; Shocks delivered: 1	784 / 390	566 / 851
65	29-Apr-2011 05:19:23	VT1	ATP: 3; Shocks delivered: 1	690 / 384	727 / 856
63	29-Apr-2011 04:52:44	VT1	ATP: 3; Shocks delivered: 1	766 / 383	1310 / 842
62	28-Apr-2011 23:59:30	VT1	ATP: 3; Shocks delivered: 1	588 / 386	584 / 598
61	28-Apr-2011 22:08:25	VT1	ATP: 3; Shocks delivered: 1	525 / 387	> 1998 / 747
60	28-Apr-2011 20:56:48	VT1	ATP: 3; Shocks delivered: 1	872 / 387	> 1998 / 868
59	28-Apr-2011 19:44:17	VT1	ATP: 3; Shocks delivered: 1	649 / 396	754 / 703
58	28-Apr-2011 19:16:43	VT1	ATP: 3; Shocks delivered: 1	677 / 387	655 / 740
57	28-Apr-2011 19:10:38	VT1	ATP: 3; Shocks delivered: 1	758 / 397	475 / 896
56	28-Apr-2011 19:06:48	VT1	ATP: 3; Shocks delivered: 1	671 / 382	610 / 536
55	28-Apr-2011 19:03:33	VT1	ATP: 3; Shocks delivered: 1	801 / 392	670 / 804
	28-Apr-2011 17:11:56	Follow-up			
54	28-Apr-2011 16:53:24	VT1	ATP: 3; Shocks delivered: 1	873 / 389	508 / 728
53	28-Apr-2011 16:43:14	VT1	ATP: 3; Shocks delivered: 1	1101 / 390	827 / 680
52	28-Apr-2011 16:31:38	VT1	ATP: 3; Shocks delivered: 1	1267 / 390	> 1998 / 878
51	28-Apr-2011 16:15:21	VT1	ATP: 3; Shocks delivered: 1	> 1998 / 385	> 1998 / 783
50	28-Apr-2011 15:49:35	VT1	ATP: 3; Shocks delivered: 1	> 1998 / 394	774 / 768
49	28-Apr-2011 15:34:34	VT1	ATP: 3; Shocks delivered: 1	885 / 396	538 / 760

Fig. 1. 78 year-old patient with dilated idiopathic cardiomyopathy. In this report the patient experienced 37 arrhythmic episodes in less than 24 hours. Physicians can check the report directly online in real time, allowing fast diagnosis of ES and, hence, immediate treatment of ES.



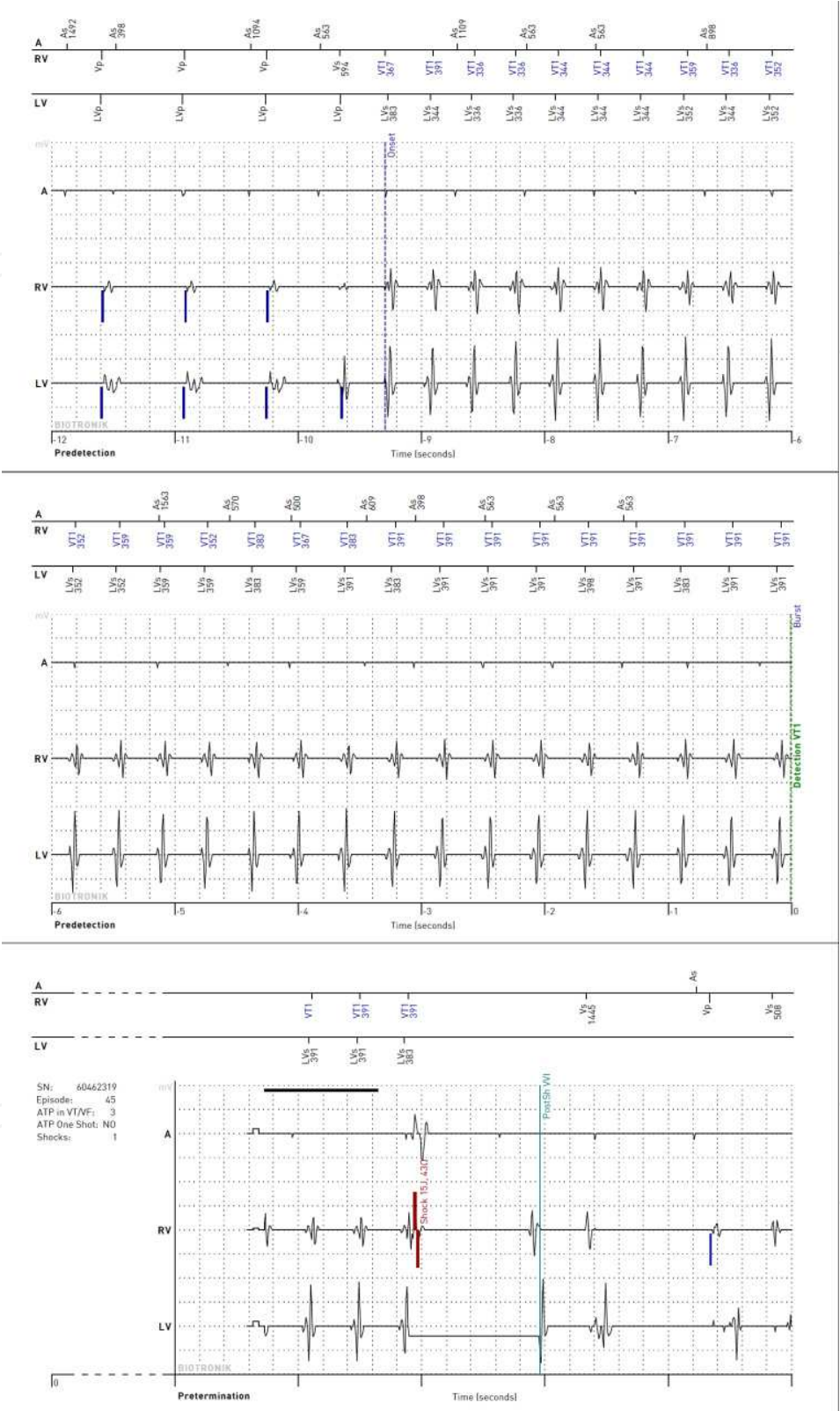


Fig. 2. One of the arrhythmic episodes reported in Fig.1. In this EGM a ventricular tachycardia in VT zone starts abruptly following a premature ventricular contraction. Atrio-ventricular dissociation is easily visible. ATP was ineffective whereas 15J delivered shock successfully terminated VT.

#### 5.4 Specific therapy in congenital pro-arrhythmic diseases

Although electrical storm associated with Brugada syndrome is exceptional, it is a major and life threatening event that requires rapid and effective treatment. In most cases presented to date, infusion of isoproterenol (as a 1-2 µg bolus injection followed by continuous infusion at 0.15 µg/min, or at a rate of about 0.003 µg/kg/min titrated to result in a 20% increase in heart rate), was used to terminate electrical storm. Other cases reported intravenous orciprenaline infusion or quinidine as effective in terminating ES. Direct β adrenergic stimulation by isoproterenol and orciprenaline increases the L-type calcium current, which restores the epicardial action potential dome, normalizes ST segment elevations and suppresses ventricular arrhythmias. It should be emphasized that orciprenaline or quinidine use as last resort approach in ES is limited to cases of confirmed Brugada syndrome, while in the majority of ES associated with ischemic or dilated cardiomyopathy, orciprenaline or quinidine application may result in fatal outcome.

In the congenital long QT syndrome, high dose β-blockers can suppress the occurrence of ES and frequent ICD discharges. In refractory cases, left cardiac sympathetic denervation results in marked reduction in ES incidence.

In the ES associated with the short-coupled variant of torsade de pointes ventricular tachycardia, verapamil or the combination of verapamil and mexiletine is somewhat effective. Intravenous magnesium and overdrive pacing are the treatment of choice for drug-induced torsade de pointes.

Arrhythmogenic right ventricular cardiomyopathy and myocardial non-compaction deeply modify heart structure, and ES associated with these conditions is usually resistant to medical therapy. Heart transplantation represents nowadays the only viable option for terminating recurrent, haemodynamically destabilizing arrhythmias in these patients.

#### 5.5 Last resource therapies. Radio-frequency ablation and heart transplantation

##### 5.5.1 Radio-frequency ablation

Although radiofrequency catheter ablation (CA) has an established role in the treatment of recurrent VT, only recently it has been suggested as a method of choice in management of ES, especially when pharmacological and ICD therapies fail. The best candidates for CA are those ventricular arrhythmias in which the initiating beat or premature ventricular contractions morphologically identical to the initiating beat can be localized with electroanatomical mapping. In patients affected with ischemic cardiomyopathy the typical site of the initiating beat is around the border zone of the scar tissue. The procedure can be performed under light anesthesia or deep sedation, according to the hemodynamic state of the patient. If the VT is not incessant, a stimulation protocol from right and left ventricle and up to three extrastimuli is usually applied to induce clinical VT and determine its characteristics. Mapping and ablation is usually performed by an irrigated-tip catheter introduced into the right ventricle or left ventricle by direct femoral vein approach or retrograde transaortic or transseptal approach, respectively. Electroanatomical mapping is nowadays the standard of care, being safe and effective.

The largest series of patients undergoing CA for refractory ES has been described by Carbucicchio and coworkers. Solid electrophysiological evidence of the effective treatment of the presenting VT was achieved in 89% of patients, whereas a transient effect of CA causing short-term stabilization but ineffective in long-term ES prevention was observed in the remaining 11% of patients (Carbucicchio et al, 2008). In this latter group, CA acted only

as a temporary bailout, with no impact on ES recurrence. In a recent Czech study RF ablation proved effective in suppression of ES in 84% of cases; however, repeated procedures were necessary in 1 out of 4 patients (Kozeluhova et al, 2011). Severely depressed left ventricular ejection fraction, highly-dilated left ventricle, renal insufficiency, and ES recurrence after previous ablation procedure were independently associated with adverse outcome within the first 6 months after the procedure. Is it still unclear whether inducibility testing of the VT at the end of the study is predictive of mortality and arrhythmic recurrences, as currently available data are controversial.

Successful CA of refractory ES in the absence of a detectable trigger has also been described. Schreieck and colleagues reported a case series of 5 ischemic patients with unmappable recurrent VTs, in which CA was attempted targeting delayed local potentials guided by voltage mapping and pace mapping (Schreieck et al, 2004). These isolated delayed potentials are found exclusively in areas of dense scar, making this kind of technique ineffective in idiopathic dilated cardiomyopathy.

On a side note, patients with pseudo-storm due to inappropriate ICD shocks induced by atrial tachyarrhythmias can benefit from CA of atrial flutter, atrial fibrillation or even AV node ablation.

### 5.5.2 Heart transplantation

Patients with no significant comorbidities except for recurrent ES who experienced no improvements from pharmacological, device-related and surgical treatment should be considered for cardiac transplantation. Patients with refractory ES associated with a genetic arrhythmia syndromes may also be reasonable candidates for heart transplantation as these patients are typically young, otherwise healthy individuals with good quality of life and prognosis.

In haemodynamically instable patients, intraaortic balloon pump and cardiac assist devices should be used as bridge-to-transplant, as potentially lifesaving.

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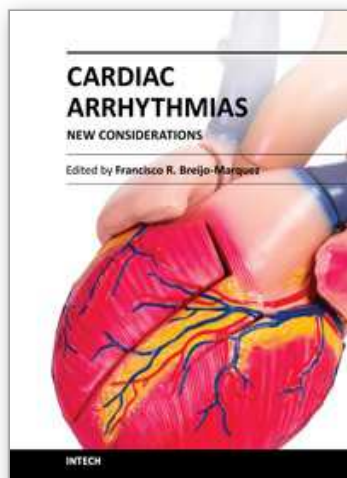
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## **Cardiac Arrhythmias - New Considerations**

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The most intimate mechanisms of cardiac arrhythmias are still quite unknown to scientists. Genetic studies on ionic alterations, the electrocardiographic features of cardiac rhythm and an arsenal of diagnostic tests have done more in the last five years than in all the history of cardiology. Similarly, therapy to prevent or cure such diseases is growing rapidly day by day. In this book the reader will be able to see with brighter light some of these intimate mechanisms of production, as well as cutting-edge therapies to date. Genetic studies, electrophysiological and electrocardiographic features, ion channel alterations, heart diseases still unknown, and even the relationship between the psychic sphere and the heart have been exposed in this book. It deserves to be read!

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