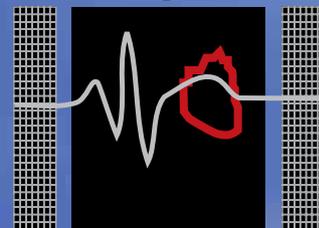


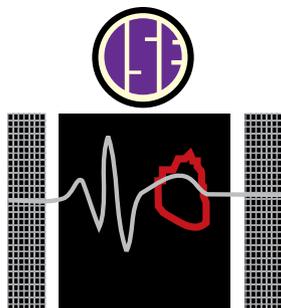
2011 : Vol. 2



INDIAN JOURNAL OF  
*Electrocardiology*

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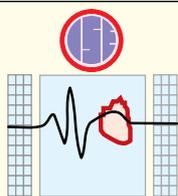
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# Editorial

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*Dear Friends,*

As we release this issue of the IJE, we are at the threshold of ISE Goa. The scientific committee has prepared an excellent arrhythmia course encompassing interesting topics relevant in day to day clinical practice as well as some rare clinical scenarios. Various utility of device therapy is also covered in this comprehensive course. I am sure their teachings and your interest will create the right mix for a good understanding of pathophysiology from the perspective of cardiac conduction system. We are very excited to bring you the current issue of IJE since it includes interesting ECG related original manuscripts as well as clinical relevant review articles.

Dr. Maheswari's group has presented an interesting case of RV infarct presenting as ST elevation in the precordial leads. They have presented the various criteria to differentiate RVMI vs Anterior wall LVMI, two different entities presenting as ST elevation in anterior precordial leads. Dr. Talele's group has presented an interested case of pseudoatrial dissociation related to diaphragmatic signals picked up on ECG. They demonstrate various criteria to confirm the diaphragmatic origin of these high frequency signals. Dr. Pandurangi's group has presented a review of SVT presenting as wide QRS tachycardia. Rare clinical scenario of VT and SVT in the same patient has also been demonstrated in this review.

The number of device implants are increasing all over the country and all of us encounter increasing number of patients with device related problems. Dr. Banchs et al. present an interesting case of inappropriate ICD shock due to electromagnetic interference. Furthermore, we have a comprehensive review on Dronaderone, a drug which is making rapid inroads into clinical practice. The authors have presented a review of the mechanism of action, clinical efficacy, side effects and clinical outcomes. The authors have made a strong evidence based argument for its role in AF management.

Drs. Proetti and Sagone have presented an excellent review of electrical storm and its management. Their thought provoking review will assist all of us in better management of this rare but high risk clinical event. The editors wish their comprehensive review included the role of Thoracic epidural anesthesia for management of electrical storm. This article has been re-printed with permission from the web-based Indian Pacing and Electrophysiology journal.

The ECG Quiz is very tricky this time around.

Happy reading and we hope to have more contributions from you for future issues.

**Jignesh Shah**  
*Guest Editor*

**Yash Lokhandwala**  
*Editor*

**Ulhas Pandurangi**  
*Editor*

## From Vice President's Desk

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*Dear Members,*

It is our great pleasure in bringing out the 2nd issue of Indian Journal of Electrocardiology of the year 2011 on the eve of Goa Arrhythmia Course 2011 – The Midterm Conference of Indian Society of Electrocardiology.

ISECON-2011 was organized by Dr. Prakash Kamath, Dr. K.U. Natarajan and the team at Kochi from 1st to 3rd April 2011. It was a great scientific feast.

Indian Society of Electrocardiology also organized many programs during the year :

- a. “ECG Learning Course” for postgraduate students at Indore on 23rd and 24th April 2011, at Hyderabad on 18th and 19th June 2011, at Agra on 6th and 7th August 2011, and at Goa on 16th and 17th September 2011. About 80-100 delegates participated in each course and successful candidates were awarded the Certificate of Competence for ECG reading

My sincere thanks to Dr. Yash Lokhandwala, Dr. Ulhas Pandurangi, Dr. Jignesh Shah and the Editorial Team for bringing out the ISE Journal – 2011, 2nd Volume.

Long Live Indian Society of Electrocardiology



**Dr. S.B. Gupta**

*Vice President*

*Indian Society of Electrocardiology*





## Review Article

# Dronedarone in Atrial Fibrillation

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### Introduction

Atrial fibrillation (AF), the most common sustained arrhythmia, is strictly age dependent: 0.1% of all individuals under 55 years, but up to 9.0% of those over 80 years are affected. AF is associated with multiple cardiovascular conditions including arterial hypertension, dyslipidemia, heart failure, valvular heart disease, coronary artery disease, and diabetes mellitus. AF is known to be associated with poor prognosis in patients with cardiovascular diseases. It is associated with increased risk of serious cardiovascular complications like heart failure, stroke, cardiovascular hospitalization and death.

Management of AF is a three pronged approach of : stroke prevention, rate control and rhythm control. Role of oral anticoagulation in AF patients with risk factors for stroke is well established. Rate control as well as rhythm control reduces symptoms and improve the quality of life However, data from randomized controlled trials have failed to establish superiority of either rate or rhythm control.<sup>1,2</sup> There is a small subset of patients who are symptomatic with rate controlled AF. These patients are ideally suited for rhythm control strategy. Amiodarone is the most frequently used antiarrhythmic drug to achieve and maintain sinus rhythm. However, the use of amiodarone is limited by significant adverse effects including pulmonary toxicity, skin discoloration, thyroid toxicity, corneal deposits, and optic neuropathy. Consequently, research has focused on developing more favourable agents with equal efficacy and less adverse effects. Dronedarone was the result of this research. It is a modified amiodarone intended to combine the efficacy of amiodarone without its myriad of side effects. The current article reviews the current literature on this new agent.

### Pharmacology

Dronedarone is a benzofuran molecule, which is chemically related to amiodarone. Unlike amiodarone, it does not contain the iodine moieties causing thyroid problems. Moreover, the addition of a methyl sulfonyl group decreases its lipophilicity and shortens its plasma half-life and hence the decrease in the organ toxicity due to cumulative effects.

Dronedarone is well absorbed after oral administration, with a bioavailability of approximately 15% after extensive first pass metabolism. As with amiodarone, the drug is extensively metabolized by cytochrome P-450 (CYP) 3A4 and excreted in the bile with minimal renal excretion. Thus, concurrent use of

medications that inhibit CYP3A4 can increase exposure to the drug and result in potentially serious drug-drug interactions. Given that the drug is highly bound to plasma proteins, the steady-state terminal elimination half-life is approximately 30 hrs compared to that of amiodarone (approximately 58 days) due to its extensive tissue deposition. Approximately 10% to 15% increase in serum creatinine can be seen with dronedarone, due to inhibition of tubular secretion of creatinine.<sup>3</sup>

### Role of Dronedarone in rhythm control

Dronedarone was expected to have rhythm control effects of amiodarone without its side effects. The antiarrhythmic efficacy of dronedarone has been evaluated in 4 placebo-controlled and 1 active-control randomized trials..

### Delay in recurrences of AF or maintenance of sinus rhythm

The DAFNE study was a phase 2 dose-ranging study that established a 400 mg twice daily dose to have optimal efficacy and safety.<sup>4</sup> The EURIDIS and ADONIS studies were identical sister trials performed under the same protocol that assessed the efficacy of dronedarone to maintain sinus rhythm in patients with a history of nonpermanent AF/AFL who were in sinus rhythm at the time of randomization and had no clinically significant structural heart disease or heart failure.<sup>5</sup> Although the ATHENA study was designed to primarily evaluate the impact of dronedarone on clinical outcomes, data on arrhythmia recurrence were also assessed. In all above mentioned four trials, dronedarone delayed the time to the first recurrence of arrhythmia and decreased recurrence of these events. Pooled data from all 4 studies demonstrate that 43% of dronedarone-treated patients were estimated to have experienced a first AF/AFL recurrence at 1 year, compared with 54% of placebo-treated patients ( $p < 0.0001$ ).<sup>6</sup>

In another meta-analysis, Piccini et al compared four placebo controlled trials of Amiodarone, 4 placebo controlled trials of Dronedarone and one trial comparing amiodarone to dronedarone. Though there was a significant reduction in recurrent AF with amiodarone versus placebo (odds ratio [OR]: 0.12; 95% confidence interval [CI]: 0.08 to 0.19) there was no reduction in case of dronedarone versus placebo (OR: 0.79; 95% CI: 0.33 to 1.87). A direct comparison between the 2 drugs showed amiodarone to be superior to dronedarone (OR: 0.49; 95% CI: 0.37 to 0.63;  $p < 0.001$ ) for the prevention of recurrent AF.<sup>7</sup> Even the previous estimates of only 57% patients having freedom from AF need to put into perspective.

For eg. quinidine has 50% efficacy in maintaining sinus rhythm compared with 25% for placebo at 1 year.<sup>8</sup> The DIONYSOS trial compared the efficacy and safety of dronedarone (400 mg twice a day) versus amiodarone (600 mg daily for 28 days, then 200 mg daily thereafter) for at least 6 months for the maintenance of sinus rhythm in patients with AF.<sup>9</sup> This study showed that recurrences of AF were more frequent in the dronedarone group than in the amiodarone group (63% vs. 42%; relative risk [RR]: 1.51, 95% confidence interval [CI]: 1.27 to 1.80). Furthermore, previous studies with sotalol and amiodarone have demonstrated attenuation of treatment effect with longer follow-up. There is no evidence to suggest that this might not be the case with dronedarone as well. Thus, these data suggest that dronedarone has modest antiarrhythmic efficacy.

### Role of Dronedaronerone in Rate control

The ERATO trial was a placebo-controlled study to evaluate the efficacy of dronedarone 400 mg bid given for 6 months in controlling the ventricular rate in patients with symptomatic permanent AF at rest.<sup>10</sup> The primary end point, decrease from baseline in 24-h Holter heart rate on day 14, was significantly more pronounced in the dronedarone group (mean of 86.5 to 76.2 beats/min) than in the placebo group (90.6 to 90.2 beats/min). This rate-controlling effect of dronedarone was sustained throughout the 6-month trial and was additive to the effect of other rate-control therapies. A similar pattern was seen among patients with AF recurrence in the DAFNE trial (89.7 vs. 102.9 beats/min,  $p < 0.001$ ),<sup>4</sup> the EURIDIS and ADONIS trials (102 vs. 117 beats/min,  $p < 0.001$ ),<sup>5</sup> and the ATHENA trial (75 vs. 84 beats/min,  $p < 0.001$ ).<sup>11</sup> These findings demonstrate that dronedarone reduces the ventricular rate of patients with both permanent and non permanent AF. Thus, in aggregate, these studies establish that dronedarone has the ability to control both rhythm and rate in patients with AF/AFL. However, the antiarrhythmic efficacy is quite modest compared with placebo and only half as effective compared with the gold standard amiodarone.

### Safety of Dronedaronerone

The safety of dronedarone has been evaluated in 2 randomized controlled trials. The ANDROMEDA study was designed to establish the safety in a high risk population. The trial enrolled patients with recently symptomatic decompensated heart failure (NYHA functional class II to IV) who may or may not have had AF.<sup>12</sup> Approximately 25% of patients had AF on randomization and 37% of patients enrolled had a history of AF. The primary end point was time to mortality or hospitalization for worsening heart failure. The trial was terminated prematurely because of excess mortality among dronedarone-treated patients.<sup>12</sup> The excess mortality appeared to be predominantly related to worsening heart failure, followed by arrhythmia and sudden death. The outcome of the ANDROMEDA study resulted in a recommendation by the FDA to restrict its use in patients with recent heart failure related hospitalization.

The ATHENA study was a randomized trial to evaluate the long-term effect of dronedarone 400 mg bid versus placebo on the combined risk of cardiovascular hospitalization or all-cause mortality in patients with a recent or current history of nonpermanent AF/AFL and additional risk factors.<sup>11</sup> Although patients with stable heart failure were included, the trial excluded patients who were clinically decompensated. Treatment with dronedarone was associated with a 24% reduction of the combined risk of cardiovascular hospitalization or all cause death (RR: 0.76, 95% CI: 0.69 to 0.84) compared with placebo over a follow-up of 21 months. Based largely on the results of the ATHENA study, the FDA approved dronedarone to reduce the risk of cardiovascular hospitalization in the treatment of AF/AFL. Taken together with the findings of the ANDROMEDA trial, these observations suggest that caution is warranted in considering dronedarone for patients who have heart failure in general, and that the use of dronedarone in patients with NYHA class IV heart failure or NYHA class II or III heart failure with recently decompensated heart failure is contraindicated, resulting in a boxed warning by the FDA.<sup>13</sup>

More recently, PALLAS (Permanent Atrial Fibrillation Outcome Study Using Dronedaronerone on Top of Standard Therapy) was an international phase IIIb study compared dronedarone 400 mg twice daily (the approved dose) to placebo in about 3,000 patients with permanent AF, who were over age 65 and had comorbidities such as previous myocardial infarction, documented coronary artery disease, previous stroke, symptomatic heart failure, or diabetes. Patients with New York Heart Association class IV or unstable NYHA class III heart failure were excluded. This trial was suspended early due to an increase in cardiovascular events with dronedarone. There was a 2.3 fold increase in major cardiovascular events defined as a composite of stroke, MI, systemic embolism, or cardiovascular death and 1.5 fold increase in unplanned CV hospitalisation and death in the dronedarone arm as compared to the placebo arm.<sup>14</sup> Thus, recent times have seen increasing documentation of poor outcomes with dronedaronerone built up and heretofore undiscovered adverse effect come to light in post marketing phase.

### Adverse Effects

In the DIONYSOS study, which compared 400 mg bid dronedaronerone with 200 mg amiodarone, dronedaronerone was associated with a reduced risk of thyroid disorders, sleep disorders, and tremor, and fewer episodes of bleeding due to less interference with oral anticoagulants, but the risk of adverse gastrointestinal events was increased. However, premature discontinuations due to treatment-related adverse events (the primary tolerability end point) were not statistically different—10.4% versus 13.3% (RR: 0.78, 95% CI: 0.48 to 1.27).<sup>8</sup> Although no pulmonary or liver toxic effects were seen with either agent, the short duration (6 months) of the study precludes any definitive conclusions regarding long-term safety. Of reported adverse

Table 1 :

Adverse Reaction	Dronedarone vs. Placebo		Amiodarone vs. Placebo	
	Dronedarone	Placebo	Amiodarone	Placebo
Pulmonary toxicity	32/3,205 (1.0)	20/2,802 (0.7)	2/419 (0.5)	1/245 (0.4)
Thyroid toxicity	128/3,205 (4.0)	83/2,802 (3.0)	12/161 (7.5)	0/113 (0)
Hepatic toxicity	112/3,205 (3.5)	69/2,802 (2.5)	1/161 (0.1)	0/113 (0)
Symptomatic bradyarrhythmia	89/3,205 (2.8)	31/2,802 (1.1)	6/161 (3.7)	0/113 (0)
Increase in serum creatinine	128/3,205 (4.0)	32/2,802 (1.1)	NR	NR

\*Data are pooled from those studies in which the authors reported organ-specific adverse reactions requiring discontinuation of drug therapy.

effects of dronedarone include diarrhoea, nausea or vomiting, and rash. Dronedarone produces electrocardiographic changes consistent with its pharmacodynamic activity; there is no evidence of a proarrhythmic effect of dronedarone, with only 1 case of torsades de pointes identified so far. A benign transient increase in serum creatinine has been observed with dronedarone. Unlike amiodarone, dronedarone is not associated with endocrinological, neurological, or pulmonary toxicity in the pooled AF/AFL studies, although a mean follow-up of 12 months may be an insufficient duration for observing the type of pulmonary toxic effects seen with long-term amiodarone use.<sup>7</sup> In January 2011, the manufacturers reported rare but severe hepatic injury secondary to dronedarone in the post marketing phase. Two patients had to undergo liver transplant due to significant hepatic injury.<sup>15</sup>

Thus, while dronedarone has been shown to be well tolerated compared with placebo, when compared with amiodarone, it has a modest, but non-significant, tolerability advantage (Table 2). Long-term studies are required to conclusively establish the superior safety and tolerability of dronedarone over amiodarone.

### Conclusions and Implications

Dronedarone is envisioned and marketed as a safer alternative to amiodarone for maintaining sinus rhythm in patients with AF. However, its relatively modest efficacy in preventing AF/AFL recurrence as well as questions regarding its short and long-term safety in at-risk patients leave its role in the management of this arrhythmia uncertain. What role do we see for this drug in clinical practice? In general, a rhythm control strategy is advisable only for a subset of AF patients. For many AF patients, rhythm control with antiarrhythmic drugs should generally be considered only when symptoms persist despite adequate rate control. When a rhythm-control strategy is desired for patients with no or minimal heart disease, including patients with hypertension but without substantial left ventricular hypertrophy, flecainide,

propafenone, and sotalol are recommended as first-line agents by guidelines<sup>13</sup> based on their proven safety and efficacy in this population. The use of dronedarone might merit consideration for these patients as an alternative to amiodarone or dofetilide (recommended as second line agents), especially for patients intolerant to these drugs.

For patients with hypertension and substantial left ventricular hypertrophy, amiodarone should be used as first-line treatment<sup>13</sup> with consideration for dronedarone only for patients who are intolerant of amiodarone. For patients with coronary artery disease and without overt heart failure, for whom dofetilide and sotalol are recommended as first-line treatment option,<sup>13</sup> dronedarone might be a reasonable alternative to these drugs or to amiodarone (second line therapy). Among patients with heart failure for whom a rhythm-control strategy is desired, amiodarone or dofetilide is recommended as a first-line agent based on its neutral effect on survival in these patients. In general, dronedarone should be avoided in patients with heart failure, especially those with advanced or recently decompensated heart failure for which it carries a “boxed” warning. However, for patients with less advanced and without recently (within the last month) decompensated heart failure (NYHA functional class II or less, or EF >35%, dronedarone could potentially offer a reasonable alternative, particularly for patients who are intolerant of low dose amiodarone or dofetilide. Thus, the available data support only limited use of dronedarone for select patient populations, mostly as a second- or third-line agent in lieu of amiodarone.

Following the suspension of phase 3 PALLAS trial because of an increase in CV events in those randomized to dronedarone, recently The European Medicines Agency (EMA) and FDA reminded to be careful to use dronedarone as indicated and not to prescribe it for patients with permanent AF.<sup>15</sup>

Unlike amiodarone, dronedarone has *not been studied for management of other arrhythmias such as ventricular arrhythmia*, another common arrhythmia in heart failure patients.

To further understand how dronedarone will fare against amiodarone in the wider population with heart disease, more studies with longer follow-up are needed. At the very least, these studies need to demonstrate superior tolerability of dronedarone without unacceptable loss of efficacy in the maintenance of sinus rhythm and quality of life, or without an increase in morbidity or mortality compared with amiodarone.

Due to its modest antiarrhythmic efficacy, lack of significant well-established safety advantage over amiodarone, a huge cost disadvantage compared with amiodarone, we conclude that dronedarone can be used in a highly selected population as a second or third line agent especially for those who are intolerant to amiodarone.

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# Electrical storm: Incidence, Prognosis and Therapy

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## Abstract

Implantable defibrillators are lifesavers and have improved mortality rates in patients at risk of sudden death, both in primary and secondary prevention. However, they are unable to modify the myocardial substrate, which remains susceptible to life-threatening ventricular arrhythmias. Electrical storm is a clinical entity characterized by the recurrence of hemodynamically unstable ventricular tachycardia and/or ventricular fibrillation, twice or more in 24 hours, requiring electrical cardioversion or defibrillation. With the arrival of the implantable cardioverter-defibrillator, this definition was broadened, and electrical storm is now defined as the occurrence of three or more distinct episodes of ventricular tachycardia or ventricular fibrillation in 24 hours, requiring the intervention of the defibrillator (anti-tachycardia pacing or shock). Clinical presentation can be very dramatic, with multiple defibrillator shocks and hemodynamic instability. Managing its acute presentation is a challenge, and mortality is high both in the acute phase and in the long term. In large clinical trials involving patients implanted with a defibrillator both for primary and secondary prevention, electrical storm appears to be a harbinger of cardiac death, with notably high mortality soon after the event. In most cases, the storm can be interrupted by medical therapy, though transcatheter radiofrequency ablation of ventricular arrhythmias may be an effective treatment for refractory cases.

This narrative literature review outlines the main clinical characteristics of electrical storm and emphasises critical points in approaching and managing this peculiar clinical entity. Finally focus is given to studies that consider transcatheter ablation therapy in cases refractory to medical treatment.

**Keywords:** Electrical storm, incessant arrhythmias, radiofrequency transcatheter ablation

The term electrical storm (ES) was introduced in the 1990s to describe a state of electrical instability of the heart characterized by a series of malignant ventricular arrhythmias in a short period of time.<sup>1</sup> This condition has been described in patients with post-infarction ischemic heart disease, various forms of cardiomyopathy, valve disease, corrected congenital heart disease and genetically determined heart diseases with no apparent structural alteration, as for example in Brugada syndrome.<sup>2</sup> The rapid succession of life-threatening ventricular arrhythmias leads to increased mortality and requires intensive care, hemodynamic study, multiple cardioversions and cardiopulmonary resuscitation.<sup>2</sup>

ES was formerly defined as the recurrence of hemodynamically unstable ventricular tachycardia and/or ventricular fibrillation, twice or more in 24 hours, requiring electrical cardioversion or defibrillation.<sup>3-6</sup> With the arrival of the ICD (implantable cardioverter-defibrillator) this definition was broadened, and ES is now defined as the occurrence of three or more distinct episodes of ventricular tachycardia (VT) or ventricular fibrillation (VF) in 24 hours, requiring the intervention of the defibrillator (anti-tachycardia pacing [ATP] or shock).<sup>7-21</sup> It should be noted that this latter definition does not include the presence of hemodynamic instability, since the intervention of the defibrillator generally prevents the arrhythmia from becoming hemodynamically significant. Obviously, an inappropriate intervention of the device is not considered.<sup>10</sup> Moreover, the episodes of VT must be separate, meaning that the persistence of ventricular tachycardia

following ineffective intervention is not regarded as a second episode.<sup>10</sup> By contrast, a sustained ventricular tachycardia that resumes immediately after ( $\geq 1$  sinus cycle and within 5 minutes) efficacious therapeutic intervention by the defibrillator is regarded as a severe form of electrical storm.<sup>10</sup> The electrical instability that may arise in the first week following ICD implantation is thought to be caused by an irritative stimulus and is not strictly interpreted as an electrical storm.<sup>10</sup> The inclusion of anti-tachycardia pacing in the defining criteria of ES requires particular attention for two reasons: first, the fact that it does not arouse immediate alarm and may cause the real incidence of the phenomenon to be underestimated; secondly, a case of a single shock by the defibrillator requires careful evaluation by the cardiologist, since it might in reality, conceal an ES in which other tachyarrhythmias have been treated by means of anti-tachycardia pacing. ES is deemed to be resolved if the patient is free from VT for at least two weeks.<sup>14</sup>

## Incidence

Between 1996 and 2006, several studies were carried out in order to investigate the incidence and prognosis of ES in ICD recipients (Table 1). However, the definition of ES was not homogeneous in the studies examined. This fact, together with the differences in the considered observation period and in the populations assessed, yielded great variability in the incidence of ES reported in the various studies. According to the above-mentioned definition (3 defibrillator interventions in 24 hours), the reported

**Table 1 : Incidence of electrical storm**

Author	Electrical storm definition	% Incidence
Wood <sup>5</sup>	>3 VT in 24 hrs	10
Villacastin <sup>7</sup>	>2 shock for single VT	20
Fries <sup>8</sup>	>2 VT in 1 hr	60
Credner <sup>9</sup>	>3 VT in 24 hrs	10
Exner <sup>11</sup>	>3 VT in 24 hrs	20
Sesselberg <sup>12</sup>	>3 VT in 24 hrs	4
Arya <sup>13</sup>	>3 VT in 24 hrs	14
Greene <sup>14</sup>	>3 VT in 24 hrs	18
Bänsch <sup>15</sup>	>3 VT in 24 hrs	28
Stuber <sup>16</sup>	>3 VT in 2 weeks	24
Hohnloser <sup>17</sup>	>3 VT in 24 hrs	23
Verma <sup>18</sup>	>2 VT interrupted by shock in 24 hrs	10
Brigadeau <sup>19</sup>	>2 VT in 24 hrs	40
Gatzoluis <sup>20</sup>	>3 VT in 24 hrs	19
Gasparini <sup>25</sup>	>3 VT in 24 hrs	7

VT=ventricular tachycardia, hrs=hours

incidence of ES varies from 10 to 28% in an observation period of 1-3 years in those studies in which ICD implantation was carried out for secondary prevention.<sup>9,11,15</sup> In the MADIT II study, which concerned primary prevention, the incidence proved to be substantially lower- about 4%.<sup>12</sup>

The time from ICD implantation to the onset of electrical storm also differs in the various caserecords. Indeed, the time-lapse seems to be particularly affected by the population considered, the myocardial substrate, the medical therapy undertaken and the indications for device implantation. In one of the earliest studies,<sup>9</sup> the average time of ES onset was reported to be 4-5 months after device implantation. By contrast, more recent studies have reported a period of 2-3 years.<sup>16,18</sup>

The number of VT that occurs during an episode of electrical storm varies substantially among the different studies. Verma reported an average of 5±5 shocks,<sup>18</sup> while Green<sup>14</sup> recorded an average of 55 VT. Most of the arrhythmic episodes that occur during ES are episodes of monomorphic ventricular tachycardia, while polymorphic VT and VF are unusual causes.<sup>10</sup> Verma<sup>18</sup> reports a significant correlation between the initial arrhythmia that prompted ICD implantation and the arrhythmia responsible for ES.

### Triggers and risk factors

Many papers report that in the majority of patients, an evident trigger i.e. a contingent cause that prompts the storm, cannot be identified.<sup>10</sup> Credner<sup>9</sup> underlined the presence of hypokalemia, acute coronary syndrome and worsening heart failure as potential triggers in 26% of the patients in his case-records. Similarly, the SHIELD trial,<sup>17</sup> which evaluated the effect of azimilide on the frequency of defibrillator shock, identified a storm trigger in 13% of patients: worsening heart failure in 9%

and electrolytic imbalance in 4%. By contrast, the papers by Green<sup>14</sup> and Bansch<sup>15</sup> respectively report an identifiable trigger in 71% and 65% of their patients. In both studies, psychological stress seemed to be a trigger, defining 10% of the causes detected by Green<sup>14</sup> and 4% of those reported by Bansch.<sup>15</sup> This finding seems to be determined by adrenergic activation, the impact of which on the genesis of the storm is corroborated by the finding of an increased incidence during the daytime hours, a marked effect of beta-blocker therapy and a reduced sensitivity of baroreceptor reflexes.

Like triggers, the risk factors for ES are also difficult to identify. Severely compromised ventricular function, chronic renal insufficiency and ventricular tachycardia as the onset arrhythmia all seem to correlate significantly with the development of storms.<sup>19</sup> In the MADIT II study,<sup>12</sup> patients with acute coronary syndrome or episodes of tachycardia and/or ventricular fibrillation after enrolling showed a higher incidence of ES.

Although these data on the causes and risk factors are not conclusive, it emerges that ES is the result of multiple interactions between a predisposing electrophysiological substrate and alterations in the autonomous nervous system and cellular milieu.<sup>10</sup> The progression of myocardial disease through fibrosis, ischemia and ventricular remodeling may manifest itself as an isolated tachyarrhythmic episode that is predictive of future ES. The correlation among worsening heart disease, acute disease and emotional stress corroborates the critical role of an increased activation of the sympathetic nervous system in the pathogenesis of ES.<sup>10</sup>

### Prognosis

The immediate clinical consequence of ES is hospitalization, which takes place in 80% of patients, particularly those who have received a shock from the device (100% if >3 shocks are received).<sup>15</sup> Moreover, the electrical instability impairs the patient's quality of life and can induce a state of anxiety, which may have psychological repercussions.<sup>10</sup>

With regard to mortality, it is not surprising that studies conducted on large numbers of patients for a sufficiently long period of follow-up have documented an increase. In the AVID study,<sup>11</sup> patients with ES displayed a 2-fold higher risk of all-cause mortality; this risk was particularly concentrated in the first 3 months following the event. On considering patients with non-ischemic heart disease, Bansch<sup>15</sup> found a higher mortality rate over a follow-up of 3 years in patients with a previous episode of ES; this risk was even greater if the electrical instability had caused acute heart failure. The MADIT II study<sup>12</sup> also reported a higher mortality rate among patients with ES, again concentrated in the first 3 months after the event.

Verma<sup>18</sup> also observed that mortality was higher among patients with ES than among control subjects with ICD. However, this increased mortality occurred much later than the first 3 months reported in the previous studies and proved to correlate with

**Table 2 : Success rate of transcatheter radiofrequency ablation of electrical storm.**

Author	Number of patients	% of acute success in controlling electrical storm
Willems <sup>30</sup>	6	100
Strickbecker <sup>31</sup>	21	76
Sra <sup>32</sup>	19	66
Schreieck <sup>33</sup>	5	100
Silva <sup>34</sup>	15	80
Marrouche <sup>35</sup>	8	100
Bänsch <sup>36</sup>	4	100
Brugada <sup>37</sup>	10	80
Carbucicchio <sup>38</sup>	95	100

the progression of heart failure rather than arrhythmic death. This finding raises the question of how ES contributes to worsening the prognosis of such patients. Undoubtedly, it can be hypothesized that the shocks exert an effect on the myocardial damage, inflammation and even remodeling; however, any such effect should not be overestimated.<sup>21</sup> Indeed, episodes of ventricular fibrillation can lead to an increase in intracellular calcium and the progression of heart failure.<sup>21</sup>

Such mechanisms remain plausible, though unconfirmed. What is clear, however, is that, while immediate mortality linked to ES in ICD patients is not high, the first few months after the episode seem to be critical on account of the increased mortality that is related not so much to the potential arrhythmic instability as to the progression of cardiac dysfunction.<sup>21</sup> This finding suggests that ES is a critical moment in the progression towards irreversible heart failure, of which it may also constitute the initial manifestation.<sup>14</sup>

### Pharmacological therapy

Electrical storm constitutes a critical clinical situation both on account of the difficulty in approaching and managing hemodynamically unstable ventricular arrhythmias and because it is associated with significant pain and anxiety, which further increase the sympathetic tone and facilitate further arrhythmias.<sup>20</sup>

A patient with ES has to be hospitalized and monitored in an intensive care unit. The most urgent evaluation concerns the hemodynamic stability of the arrhythmias and, if they degenerate into acute heart failure, prompt assessment of the complications linked to this (such as pulmonary edema or acute renal insufficiency).

When a trigger can be identified, its correction may reverse the electrical instability of the myocardium. For this reason, thorough clinical and laboratory evaluation is of fundamental importance in order to search for possible pro-arrhythmic triggers, such as hydro-electrolytic imbalance or the intensification of myocardial ischemia.<sup>20</sup> If any of these triggers is detected, it must be promptly treated. In some cases, myocardial revascularization

is necessary; equally often, however, electrical stabilization is required.<sup>20</sup> The intravenous administration of magnesium and potassium may be undertaken in patients with QT lengthening or hypokalemia.<sup>20</sup> With regard to immediate drug therapy, a beneficial effect can be achieved by blocking the sympathetic system through the intravenous administration of beta-blockers combined with sedatives, such as benzodiazepine.<sup>22</sup>

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.<sup>3,5</sup> If the intravenous combination of amiodarone and betablockers proves inefficacious, the addition of lidocaine is a reasonable option.<sup>23</sup>

For what concerns the prevention of ES, interesting results have been yielded by some drugs such as azamildide, a class III antiarrhythmic which blocks the calcium channels and prolongs the refractory period.<sup>17</sup> Nevertheless, the principal factor in the prevention of electrical instability is correct ICD programming.<sup>21</sup> Given that sympathetic hyperactivation is an important trigger, the risk of shock would be minimized, all the more so as anti-tachycardia pacing can successfully terminate a significant percentage of ventricular tachycardias.<sup>21</sup> In some cases, greater electrical stabilization can be achieved by shifting to cardiac resynchronization therapy (CRT) by means of biventricular pacing (which necessitates replacement of the ICD and implantation of a lead in the coronary sinus for left ventricular pacing). This strategy is supported by a very recent study by Nordbeck,<sup>24</sup> in which the incidence of ES was seen to be much lower in CRT patients than in ICD patients undergoing pacing in the left ventricle alone. In addition, a sub-analysis of the Italian Insync ICD registry<sup>25</sup> has revealed that the incidence of ES is lower in patients on CRT and that those who do not respond to CRT are at greater risk.

### Transcatheter radiofrequency ablation therapy

In cases that are refractory to drug therapy, transcatheter radiofrequency ablation of the arrhythmogenic myocardial substrate can be carried out during ES. This was first described in a few case reports<sup>26-29</sup> and subsequently presented in ample case-records (Table 2).

Willems et al.<sup>30</sup> were the first to utilize transcatheter radiofrequency delivery in a series of 6 patients with sustained ventricular tachycardia; they identified the site of emergence of the arrhythmia through early activation on mapping during tachycardia and through pace-mapping. In their case-records, acute success was 100%; in all cases, the arrhythmia was interrupted and, at the end of the procedure, could no longer be induced. No major procedural complications were recorded. One patient died of acute heart failure 24 hours after the ablation procedure. However, the rate of recurrence of single episodes of ventricular arrhythmia was 80%.

Strickbecker<sup>31</sup> reported that radiofrequency ablation significantly

reduced ICD interventions in a series of 21 patients who had received frequent defibrillator shocks for sustained ventricular arrhythmias refractory to medical therapy.

Sra<sup>32</sup> was the first to describe the advantage of electroanatomical mapping in the ablation of ES in a series of 19 patients; no recurrence of arrhythmia was recorded in 66% of cases over a 26-week follow-up. In that study, only one case of cardiac tamponade occurred during ablation, and this was successfully drained. No deaths or long-term complications were recorded during the follow-up period considered. Similarly efficacious results were described by Schreieck<sup>33</sup> in 5 patients with ischemic heart disease who underwent transcatheter ablation (during follow-up of 12-30 months, 3 were free from arrhythmia recurrence, while 2 suffered single episodes of VT). No intra- or periprocedural complications arose.

Silva<sup>34</sup> reported the efficacy of transcatheter ablation in a group of 15 patients with ES who were heterogeneous in terms of underlying heart disease (ischemic in 9 cases, idiopathic in 4, arrhythmogenic dysplasia of the right ventricle in 1, and no structural heart disease in 1). Ablation of the clinical tachycardia was achieved in 80% of cases. No intra- or periprocedural complications were recorded and, over a follow-up of 12±17 months, only two patients suffered a single recurrence of VT.

Marrouche<sup>35</sup> reported on 29 patients with ischemic heart disease in whom recurrent VF was triggered by monomorphic ventricular extrasystole originating in the fibrous peri-infarction zone. In 8 patients in whom ES was refractory to drug therapy, ablation of the ventricular extrasystole was successfully performed after mapping, and control of ES was achieved. Over a follow-up period of more than 1 year, a single episode of VF occurred in one patient and an episode of monomorphic VT occurred in another.

Bansch<sup>36</sup> reports the case-records of 4 patients with ES following acute myocardial infarction in whom control of the ventricular tachyarrhythmias was achieved through ablation of the premature ventricular beats underlying the arrhythmia.

In 10 patients with sustained ventricular arrhythmia, Brugada<sup>37</sup> reported that epicardial ablation was able to control ES in 8 cases. No procedural complications were recorded and no deaths occurred during follow-up (18±18 months).

Carbucicchio<sup>38</sup> reports the experience of transcatheter ablation as the emergency therapy of choice in a large heterogeneous population of patients with ES refractory to drug therapy in an important Italian cardiology center. In 95 patients suffering from idiopathic ischemic heart disease and arrhythmogenic dysplasia of the right ventricle, radiofrequency transcatheter ablation was able to suppress ES in the acute phase in all cases and achieved non-inducibility of ventricular arrhythmias at the end of the procedure in 89%. On 2-year follow-up examination, 92% of patients were free from ES recurrence and 66% were free from VT recurrence. No major procedure-related complications were

recorded.

## Conclusions

Electrical storm is a very challenging clinical event that can be considered under two perspectives: the arrhythmic event can constitute the clinical manifestation of worsening heart failure or it might compromise myocardial function, thereby giving rise to the high long-term mortality seen in these patients.

Treatment in the acute phase is off-label and often requires the simultaneous intravenous administration of several antiarrhythmic drugs.

Recent studies have proposed transcatheter ablation in cases of electrical storm that are refractory to drug therapy. In several case-records, this approach proved to be safe and effective in interrupting the rapid sequence of life-threatening ventricular arrhythmias, nevertheless it does not seem to change the long-term prognosis of these patients, who continue to bear an increased burden of mortality.

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## Case Report

# Pseudo-Atrial Dissociation: A Respiratory Artifact

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### Introduction

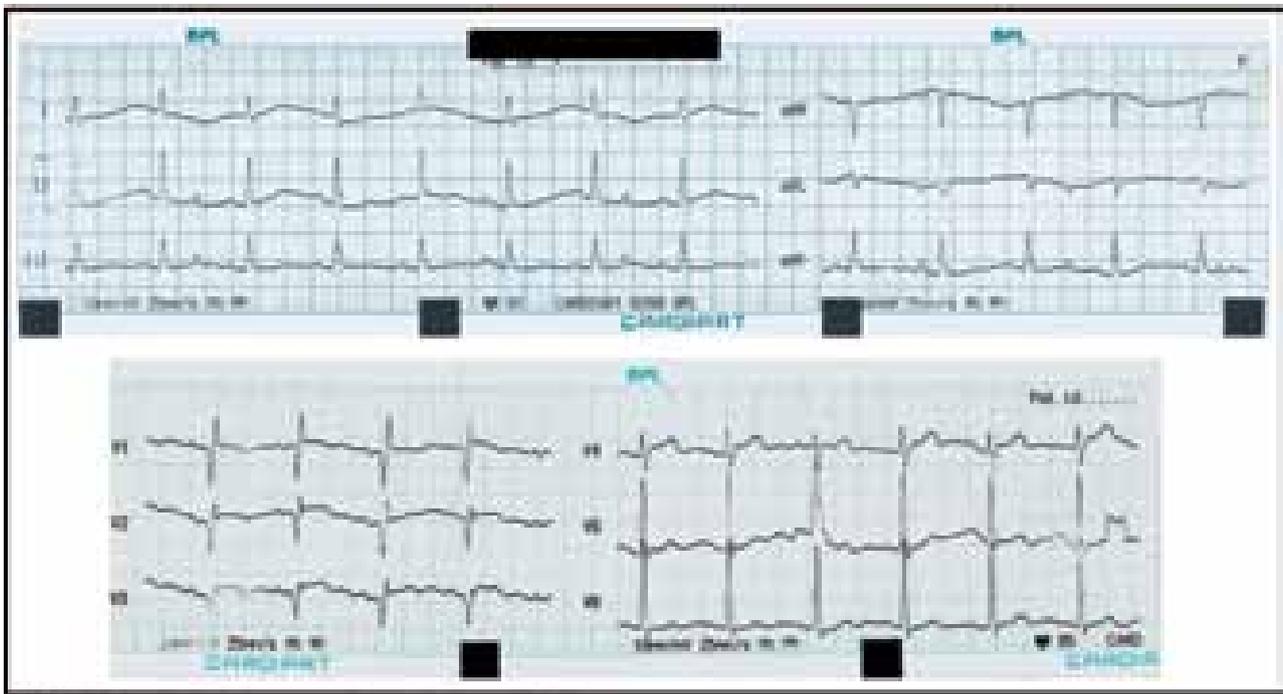
Atrial dissociation is an electrocardiographic phenomenon characterized by the presence of two separate atrial rhythms. One of these rhythms is not conducted to the ventricle, and is independent of the dominant conducted rhythm.<sup>1</sup> The condition is thought to be caused by complete interruption of impulse conduction between the right atrium and the whole (interatrial block) or part (intra-atrial block) of the left atrium, and implies disease of the Bachmann and the other interatrial bundles.<sup>2</sup> Numerous authors have described this phenomenon with different names: atrial dissociation, intra-atrial dissociation, inter-atria dissociation, double command auriculaire, uni-atrial para-arrhythmia and interatrial Block.<sup>1</sup> Bellet, coined the term atrial dissociation and reserved the term inter-atrial block for

the widening of the P wave, as seen in P mitrale.<sup>1</sup>

After experimental demonstration of atrial dissociation by Schref and Siedeck, most authors considered atrial dissociation a true electrical phenomenon.<sup>3,4,5</sup> Several authors have doubted its existence, and suggested relation with respiration.<sup>6-7</sup> The present article reports a case of patient with an assumed atrial dissociation in whom it is suggested that the P' waves correspond to the action potentials of the diaphragmatic muscle.

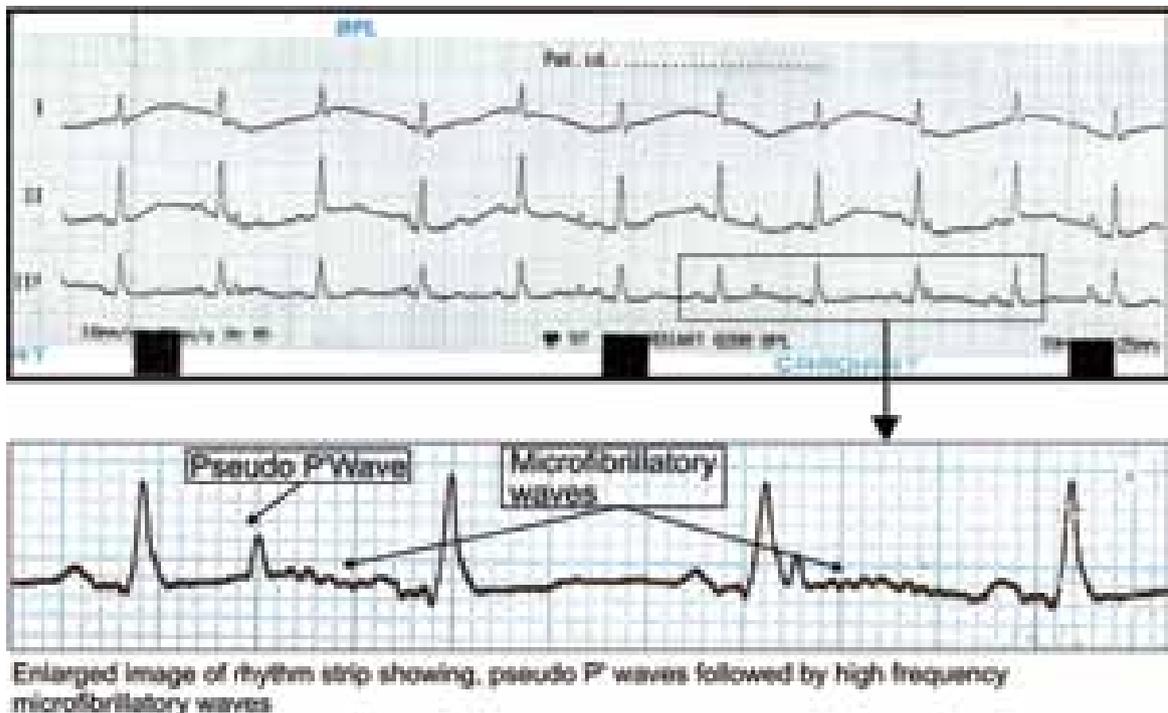
### Case Presentation

A 73 year old lady with anterior wall myocardial infarction reported to the outpatient department with complaints of breathlessness on exertion. She was breathless at rest. She had regular pulse at the rate of 90/minute, with blood pressure of



ECG showing sinus rhythm superimposed by pseudo P' waves.

**Figure 1 :** This ECG shows sinus rhythm with a rate of 88 beats per minute, the PR interval is 0.16 seconds. The duration P wave is 0.10-0.12 seconds best seen in lead II, III, AVF. The basic sinus rhythm is superimposed by pseudo P' waves that are neither related to sinus P nor to the QRS complex. The pseudo P' wave begins more sharply, and rises more steeply to a higher peak. The duration of the P' waves is 0.04 to 0.06 sec and all of them are followed by a run of high frequency waves lasting for 0.30 to 0.55 sec. The P' are registered in all limb leads except I, AVL, & V1-V6. Both, the P & P' waves have vector of +90 degrees. The P' waves are regular, and occur at rate of 53 per minute. Despite the P' waves, the original P waves are unchanged and capture the ventricles consistently with an unchanged PR interval.



**Figure 2 :** Shows enlarged image of rhythm strip clearly showing, pseudo P' waves followed by high frequency microfibrillatory waves.

120/70 mm Hg. She had bilateral basal crepts with respiratory rate of about 50/minute. Echocardiographic study revealed ejection fraction of 40% with regional wall motion abnormality in LAD territory. Her 12 lead ECG was recorded (Figure 1).

There is variable coupling interval between P and pseudo P' waves. This combination of inconstant coupling and related regular interectopic intervals is similar to that of parasystole. The unusual aspect is that, these P' waves are not able to capture ventricles even when falling in the non-refractory period.

The P' waves are not produced by any source of electromagnetic interference. They occurred while no one was touching the patient or the electrodes. The P' waves are not examples of non-conducted atrial premature contractions since there was never any interaction with the native P waves in terms of resetting or delay. Interestingly, the P' waves were followed by microfibrillatory waves, which commonly occur due to artifact arising from diaphragm.<sup>9</sup>

### Discussion

Atrial dissociation is a rare phenomenon which is usually found in patients who are critically ill and often a few hours before death.<sup>5</sup> Complete intra-atrial block has been described to be of following types: sinus rhythm with an ectopic atrial rhythm, sinus rhythm with atrial fibrillation, sinus rhythm with atrial flutter, and a combination of atrial flutter and fibrillation.<sup>8</sup> Cohen and Sherf described the coexistence of sinus rhythm and an ectopic atrial rhythm P' at rate of 28 -56/ minute as the most typical example of complete intra-atrial block.<sup>2</sup> Higgins

et al, doubted the existence of this arrhythmia, particularly due to the low rate of the P' waves, believing, sinus rhythm with interference from respiratory function.<sup>6</sup> He proved that the P' waves and microfibrillation coincided with the beginning of inspiration, attributing such electrical phenomenon to the electromyogram of the accessory respiratory muscles.<sup>6</sup>

It is noteworthy that in present case pseudo P' are followed by the transient microfibrillatory waves which commonly follow artifacts arising from diaphragm.<sup>9</sup> Soler-Soler and Angel-Ferrer, studied seven patients with severe respiratory insufficiency, whose electrocardiograms were suggestive of complete atrial dissociation. The pneumograms proved that the P' waves were temporally associated with inspiration, so that with voluntary apnoea the P' waves disappeared. The use of esophageal leads failed to show P' waves; whereas, they were shown by leads that selectively explored the diaphragmatic region. They also proved that the P' waves were not of atrial origin but represented the action potentials of the diaphragmatic muscle.<sup>9</sup> They further suggested that the diagnosis of complete interatrial block, when the P' waves appear at a slow rate, can only be accepted when it has been shown that the P' waves are not related to respiratory movements.<sup>9</sup> Although in present case the pneumogram was not recorded, the microfibrillatory potentials following pseudo P' suggest diaphragmatic origin and hence diagnostic of pseudo atrial dissociation.<sup>9</sup>

### Conclusion

The following case appears to be a case of pseudo atrial

dissociation, wherein the P' are artifact that occurred due to action potential of diaphragmatic muscle. Diagnosis of atrial dissociation should be doubted when the rate of P' waves lies between 30 - 60/minute. In order to establish the diagnosis of atrial dissociation, one must exclude external interference influencing the patient and demonstrate that the P' waves are not related to respiratory movements.

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## Case Report

# Inferior Myocardial Infarction with Associated Anterior 'ST' Segment Elevation: A Rare Presentation of Right Ventricular Infarction

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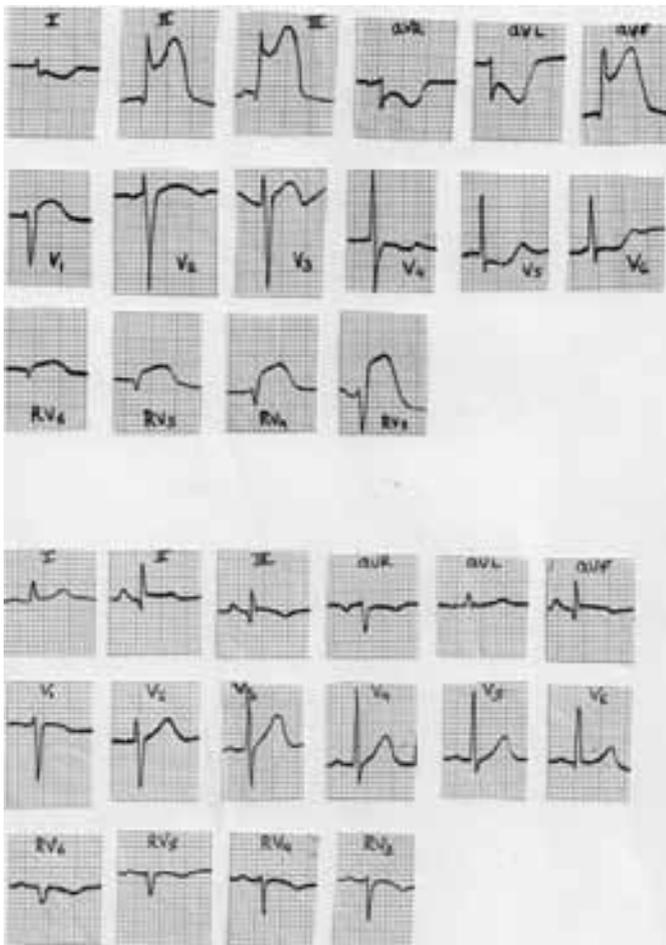
### Abstract

ST segment elevation in the left precordial leads in the setting of an acute inferior myocardial infarction may represent an unusual electrocardiographic pattern of right ventricular infarction. We present herein one such interesting case.

**Key Words :** Electrocardiogram ; Precordial ST elevation; Right ventricle infarction.

### Introduction

Acute right ventricular (RV) myocardial infarction (MI) is

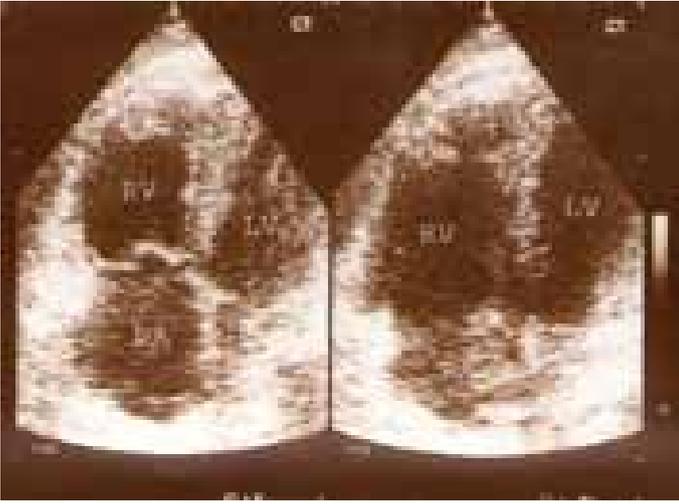


**Figure 1 :** Electrocardiogram showing ST elevation in leads II,III, aVF, RV<sub>6</sub> to RV<sub>3</sub> with ST coved and T wave inversion in leads V<sub>1</sub> to V<sub>3</sub>. Serial ECG showing settling of ST segment and T wave inversion in leads II, III and aVF and RV<sub>6</sub> to RV<sub>3</sub> with no QS in leads V<sub>1</sub> to V<sub>3</sub>

usually associated with inferior left ventricular (LV) MI in 30 to 50% of patients.<sup>1</sup> Earliest recognition of RVMI is important as it defines a significant clinical entity, which is associated with considerable immediate morbidity and mortality and has a well-delineated set of priorities for its clinical management. However, at times ECG changes are inconsistent and bedside diagnosis may be misleading. We describe a patient of inferior MI with concomitant 'ST' elevation in anterior precordial leads suggestive of acute anterior LVMI. These changes were subsequently confirmed to be due to RVMI.

### Case Report

A 43 year old, diabetic male presented to the emergency department with severe, retrosternal chest pain since 1 hour. The pain was radiating to back and to both arms was associated with profuse sweating. On physical examination, his pulse was 60/minute, blood pressure 140/80 mmHg and respiratory rate 20/minute. Jugular venous pressure (JVP) was not elevated. There was no pedal edema, pallor, icterus or lymphadenopathy. Cardiovascular examination was unremarkable. Lungs were clear. Chest skiagram showed mild cardiomegaly. Electrocardiogram revealed ST elevation in leads II,III aVF with ST coved and T wave inversion in leads V<sub>1</sub> to V<sub>3</sub>. A right sided precordial leads showed ST elevation in leads RV<sub>6</sub> to RV<sub>3</sub> (Figure 1). The next serial ECG done 6 hours later showed sagging of ST segment and T wave inversion in leads II, III, and aVF and RV<sub>6</sub> to RV<sub>3</sub> (Figure 1). A transthoracic echocardiography showed normal movements of the LV apex and interventricular septum with LV ejection fraction of 65%. and hypokinetic inferior wall. The right ventricular free wall had a distorted shape with hypokinesia and hyperechogenicity (Figure 2). Finally coronary angiogram was done which revealed normal left coronaries (Figure 3) with 90% stenosis in right coronary artery (Figure 4). Patient underwent percutaneous coronary angioplasty with stenting to RCA followed by heparin infusion, aspirin, and betablockers. With the possibility of an associated additional RVMI, no nitrates were used. The patient



**Figure 2 :** A transthoracic echocardiogram showing hypokinetic right ventricular free wall.



**Figure 3:** Left coronary angiogram (RAO caudal view) showing normal left anterior descending artery and left circumflex artery.

had no recurrence of chest pain. He had an uncomplicated and uneventful hospital stay and was discharged on the 10th day as per our hospital protocol for acute myocardial infarction.

### Discussion

In patients with acute inferior MI, associated RVMI has significant clinical, haemodynamic, and prognostic implications.<sup>2</sup> The classic triad of hypotension, elevated JVP with clear lung fields is highly specific but insensitive for RV infarction.<sup>3</sup> The sensitivity and the specificity of ST elevation in the right-sided precordial leads, especially lead RV<sub>4</sub>, for the diagnosis of RVMI is well over 90%. These ST changes



**Figure 4:** Right coronary angiogram showing stenosis in proximal right coronary artery.

tend to be most prominent in the early hours of acute MI and then dissipate over the subsequent 24 hours.<sup>4</sup> The prevalence of ST elevation in left sided anterior precordial leads in RVMI is rare (<10%).<sup>4</sup> This precordial ST segment elevation can be understood by the following hypothesis: The direction of precordial ST segments changes in acute inferior MI is subjected to two opposing influences: RVMI tends to elevate the ST segment and inferior and posterolateral infarctions tends to produce reciprocal ST depression. Thus, in patients with acute inferior MI with concomitant RVMI, the tendency for RV current of injury to elevate the precordial ST segment would be opposed by the reciprocal effects of inferoposterior current of injury. The left-precordial ST segment elevation would thus occur only when RV involvement predominates and the inferoposterior injury is small.<sup>4</sup>

The following electrocardiographic features help us to differentiate between RVMI and anterior LVMI.

1. When the magnitude of ST segment elevation in V<sub>1</sub> – V<sub>5</sub> decreases from right to left without the development of sequential Q waves, the ECG is suggestive of RVMI<sup>5</sup>
2. When the magnitude of ST elevation in RV<sub>4</sub> is more than the ST elevation in lead V<sub>3</sub> it is RVMI, in contrast to the ST elevation in an anterior MI which is always more in V<sub>3</sub> than the ST elevation in RV<sub>4</sub>.<sup>6</sup>

In our patient, the ST segment elevation decreased progressively from lead V<sub>1</sub> to V<sub>3</sub> and the RV<sub>4</sub> showed a greater ST elevation than the V<sub>3</sub>. In the serial ECGs, there was no development of QS pattern in anterior precordial leads Further no anterior

wall motion abnormality on echocardiography and normal left coronary angiogram ruled out LVMI.

In summary, a diagnosis of RVMI has to be considered when such an ECG pattern is observed because of high in-hospital mortality rates and the therapeutic consequences.

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## A Patient with Regular Narrow and Wide QRS Tachycardia

**P Sushanth, M Ravikumar, S Benjamin Solomon, Sabari Saravanan, Ulhas M Pandurangi**

Madras Medical Mission

A regular narrow QRS tachycardia is almost always a supraventricular tachycardia (SVT). Atrio-Ventricular Nodal Reentrant tachycardia (AVNRT) and Atrio-Ventricular Reentrant tachycardia (AVRT) are the commonest regular narrow QRS tachycardia in the clinical practice. These tachycardia comprise 90-95% of regular narrow QRS tachycardia in adult population. A SVT can also present as regular wide QRS tachycardia if it is associated with intraventricular conduction aberrancy. The underlying pathological or functional intraventricular conduction defects in the form of Right Bundle Branch Block (RBBB), Left Bundle Branch Block (LBBB) or Fascicular block may result in wide QRS during SVT.<sup>1</sup>

In addition to pathological causes of conduction defects (Degenerative changes, Hypertrophy, Infarction, etc) which are permanent, reversible (functional) causes of aberrant conduction include rapid rate of tachycardia, changes in the autonomic tone and effects of antiarrhythmic drugs. Under the influence of such reversible causes a SVT may exhibit wide QRS morphology even though the QRS is narrow during sinus rhythm. These

reversible causes can also convert an ongoing SVT with narrow QRS morphology into wide QRS tachycardia spontaneously. Conversely an ongoing SVT with wide QRS morphology may convert into a narrow QRS when the functional aberrant conduction disappears.<sup>2</sup>

Analysis of a regular wide QRS tachycardia should always include the analysis of morphology of QRS present during the sinus rhythm. If the morphology of the wide QRS tachycardia is similar to that of during sinus rhythm, the diagnosis is almost always SVT with aberrancy.<sup>3</sup>

It is rare for a patient to have both SVT and VT. Hence a patient with regular wide QRS tachycardia who has had a documented regular narrow QRS tachycardia or vice-versa should be considered to have SVT. The occurrence of wide QRS tachycardia should be considered as the result of reversible cause of intraventricular conduction aberrancy.

Figure 1 represents conversion of a regular narrow QRS tachycardia into a wide QRS regular tachycardia. The first part of the trace is a SVT (AVNRT) without RBBB and the later part is SVT with RBBB.

Figure 2 represents a wide QRS regular tachycardia converting into a regular narrow QRS tachycardia. The first half of the trace is SVT (AVNRT) with RBBB and the later part is without RBBB.

Figure 3 represents conversion of a regular narrow QRS tachycardia into a wide QRS regular tachycardia. The first part of the trace is SVT (AVNRT) without LBBB and the later part is with LBBB.

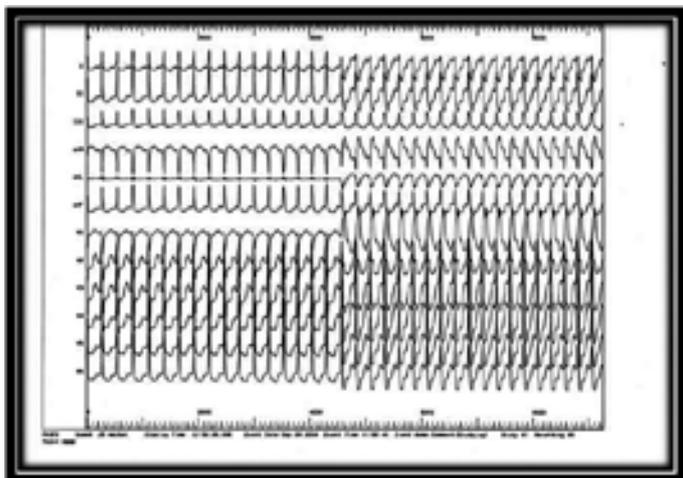


Figure 1 :



Figure 2

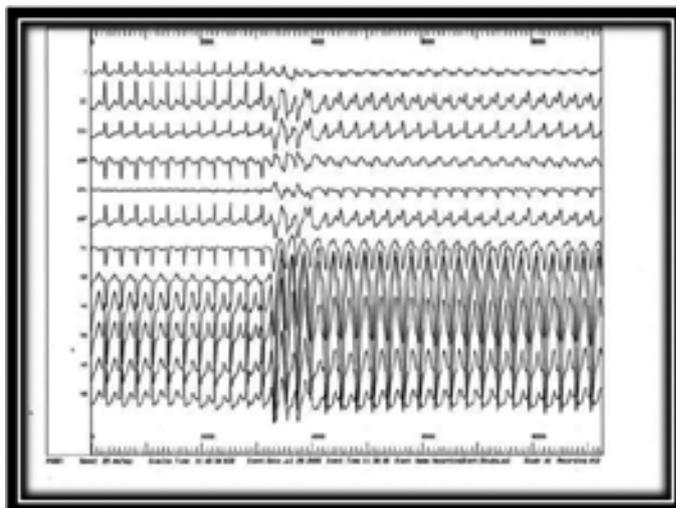


Figure 3 :

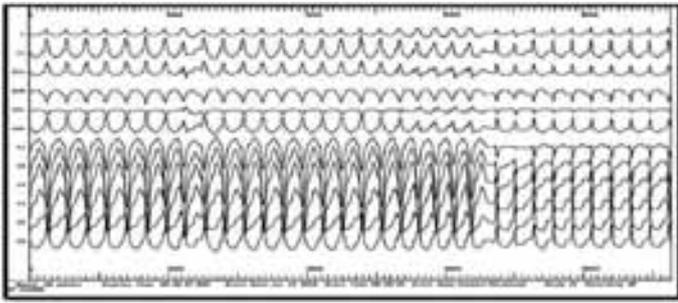


Figure 4 :

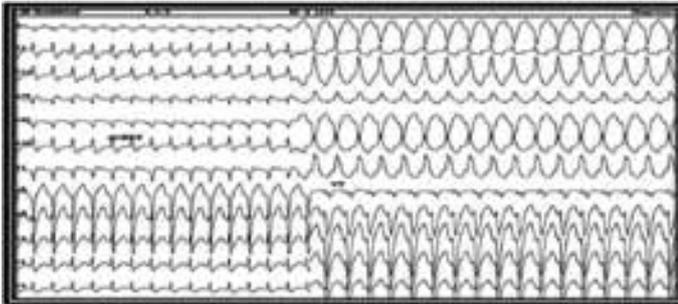


Figure 5 :

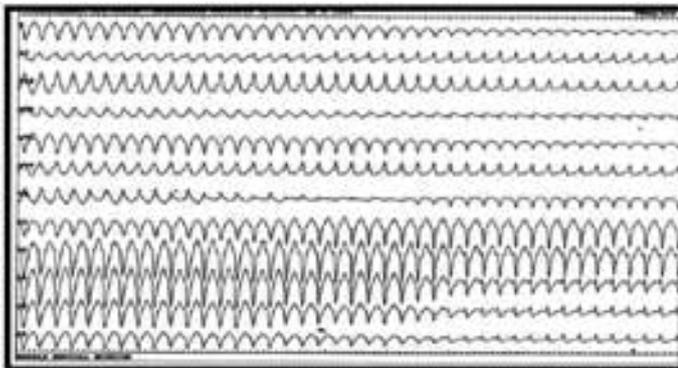


Figure 6 :

Figure 4 represents a wide QRS regular tachycardia converting into a regular narrow QRS tachycardia. The first half of the trace is SVT (AVNRT) with LBBB and the later part is without LBBB.

*The traces above are the representatives of the following rule-of-thumb of electrocardiology:*

***“Occurrence of a regular narrow QRS tachycardia and a regular wide QRS tachycardia in a same patient is almost always diagnostic of supraventricular tachycardia”***

The premises on which the above rule-of-thumb is formulated are:

1. Wide QRS morphology can be the result of reversible causes – rate, autonomic tone and antiarrhythmic drugs.
2. It is rare for a patient to have both SVT (narrow QRS tachycardia) and VT (wide QRS tachycardia).

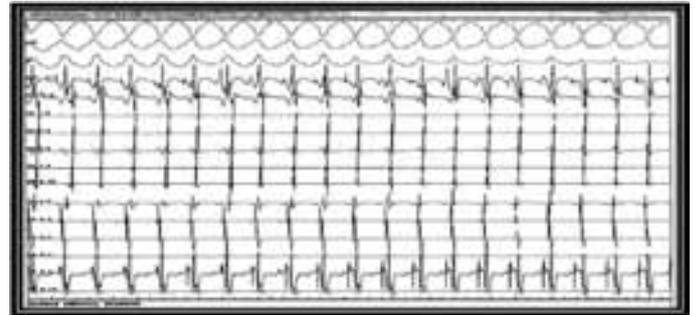


Figure 7 :

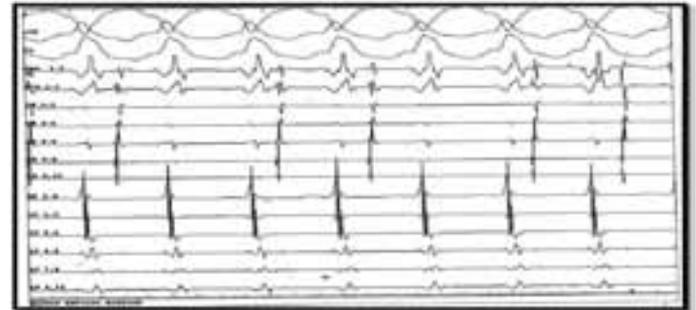


Figure 8 :

Exceptionally however a patient may have both SVT and VT. Even rarer is the occasion when a SVT with a narrow QRS morphology converts into a VT with a wide QRS morphology.<sup>4,5</sup> The following traces (Figure 5 to Figure 8) illustrate one such case.

Figure 5 represents in the first half a regular narrow QRS tachycardia during AVNRT getting converting into regular wide QRS tachycardia (VT) in the later half.

Figure 6 represents in the first half a regular wide QRS tachycardia (VT) getting converted into a regular narrow QRS tachycardia (AVNRT)

In the first half of the intracardiac trace (Figure 7) the electrograms show V-A dissociation during wide QRS tachycardia, which is diagnostic of VT. In the later half of the trace, simultaneous atrial and ventricular activation during narrow QRS tachycardia is seen, which is diagnostic of typical AVNRT.

In Figure 8 the conspicuous V-A dissociation during wide QRS tachycardia is seen from the intracardiac electrograms, which is the hallmark of VT.

**A regular narrow QRS tachycardia in an adult is most often either AVNRT or AVRT. If the morphology of the wide QRS tachycardia is similar to that during sinus rhythm, the tachycardia is almost always SVT with aberrancy. Occurrence of a regular narrow QRS tachycardia as well as a regular wide QRS tachycardia in a patient is almost always diagnostic of SVT. However extremely rarely a patient may have both SVT and VT.**

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# Inappropriate Implantable Cardioverter Defibrillator Shock in the Swimming Pool

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## Abstract

We describe the case of a 41 year old woman with an implantable cardioverter defibrillator (ICD) who received an inappropriate shock while getting out of a swimming pool. The device interrogation demonstrated evidence of electromechanical interference (EMI) corresponding to 60 cycle alternating current as the cause of the shock. EMI is a well recognized cause of inappropriate ICD shocks. Preventive measures must be taken to avoid exposure to potential sources of EMI.

## Introduction

ICD shocks have become a contemporary clinical problem in cardiology and are associated with higher morbidity and mortality as well as increased psychological stress.<sup>1-4</sup> Shocks may be inappropriate in 32% of patients receiving ICD shocks.<sup>2</sup> Significant efforts have been made to reduce the magnitude

of this problem.<sup>5-7</sup> Electromechanical interference (EMI) is a relatively common cause for inappropriate ICD shocks. It consists of external electrical signals generated outside the patient and unrelated to the implanted hardware that are sensed by the ICD as if they were physiological signals. Depending on the context and characteristics of the signals, EMI may be innocuous or result in inappropriate mode switch, inhibition of pacing and ICD shock. EMI has been reported as the cause of inappropriate ICD shocks in different settings, involving home appliances, personal care devices and entertainment machines among others.<sup>8-16</sup>

## Case

We report the case of a 44 year old woman who received an ICD shock while exiting the swimming pool. The patient had history of coronary artery disease, prior myocardial infarction and sustained ventricular tachycardia. She had NYHA class I symptoms, ejection fraction 45% and had received a Medtronic 7227 Gem single chamber implantable cardioverter defibrillator for secondary prevention. The patient described swimming in a local pool and after grabbing the stepladder hand rail to exit, she experienced an electrical sensation in her arm followed by an ICD shock. Device interrogation demonstrated evidence of electromechanical interference (EMI) corresponding to 60 cycle alternating current as the cause of the ICD shock (Figure). The intermittent pattern of the signals is generated by an automatic sensitivity adjustment built in the device software<sup>17</sup> which decreases ventricular sensitivity right after a sensed event

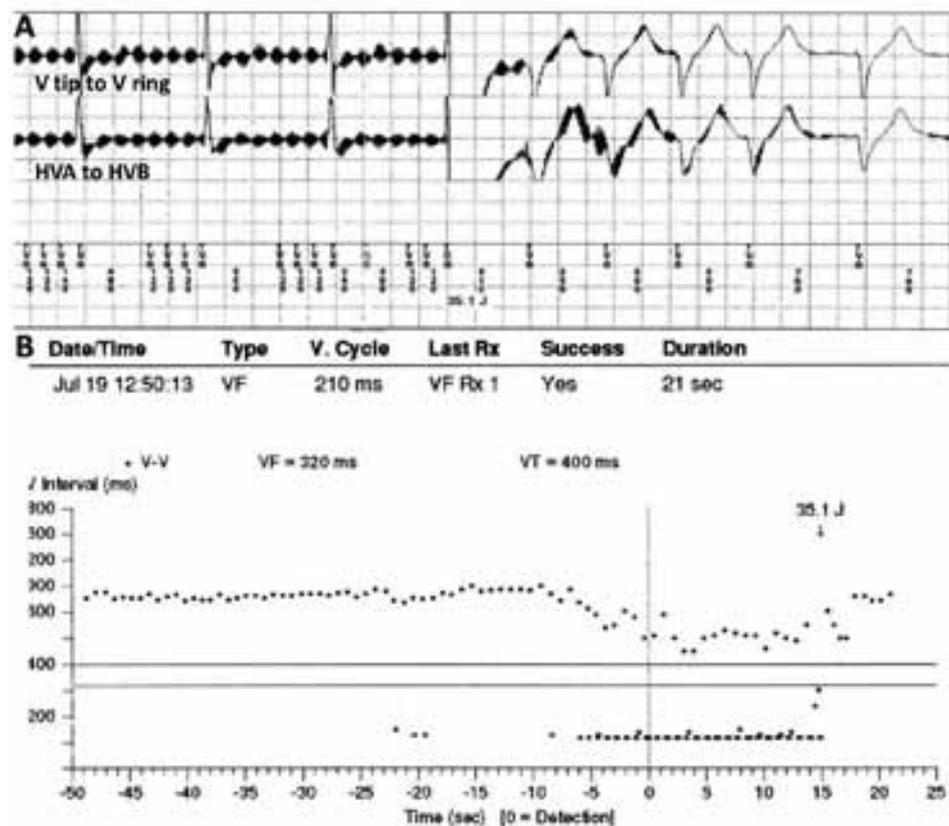


Figure : Panel A shows the electrogram recordings stored in the Medtronic 7227 Gem ICD at the time of the shock. Rapid non-physiologic signals corresponding to 60 cycle alternating current (EMI) are sensed at a CL 210 ms in groups of 4 right before every physiologic ventricular signal. Automatic sensitivity adjustment prevents the detection of the lower amplitude noise right after each sensed physiologic ventricular signal. A 35.1 Joules shock occurs and the EMI terminates after the patient releases the stepladder. Few beats of presumed sinus tachycardia follow the shock. Panel B depicts the R-R interval plot corresponding to the event.

as a function of its amplitude. Immediately after the sensed physiologic R waves the 60 cycle signals are temporarily not sensed (Figure) until sensitivity returns to the programmed threshold, giving origin to the pattern recorded.

The patient had no other demonstrable consequences from the shock and was instructed to avoid the pool until testing and corrections were made to assure lack of current leaks and appropriate grounding of the electrical equipment surrounding it.

## Discussion

EMI is a well recognized cause of inappropriate ICD shocks. After an ICD implant, we routinely educate patients regarding avoidance of sources of EMI including precautions when working with electrical outlets and equipment, avoidance of welding, large generators and industrial warehouses as well as keeping distance from running engines. Exposure to electricity has been recognized as a domestic and work environment hazard and engineering norms and codes include safety measures that prevent routine exposure to electricity. Swimming pools represent a particular challenge given the large body of water interacting with electrical equipment. While electrical devices with ground faults or shorts are a hazard in any environment, the risk increases in the pool area because the water in the pool, on surrounding surfaces and human skin itself provides a conductor for electricity to travel between the faulty equipment through the human body to ground. Defective equipment may be the underwater lights, poolside music players, water heaters or pumps. In the case presented we suspect the patient was exposed to a current leak in the water due to poor grounding and she established a connection to ground through her body as she grabbed the stepladder.

## Conclusion

Patients with ICDs and pacemakers are exposed to the hazards of EMI that could result not only in inappropriate shocks but in inhibition of pacing. Adequate patient education and preventive measures must be taken to avoid potential sources of EMI.

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# ECG Quiz

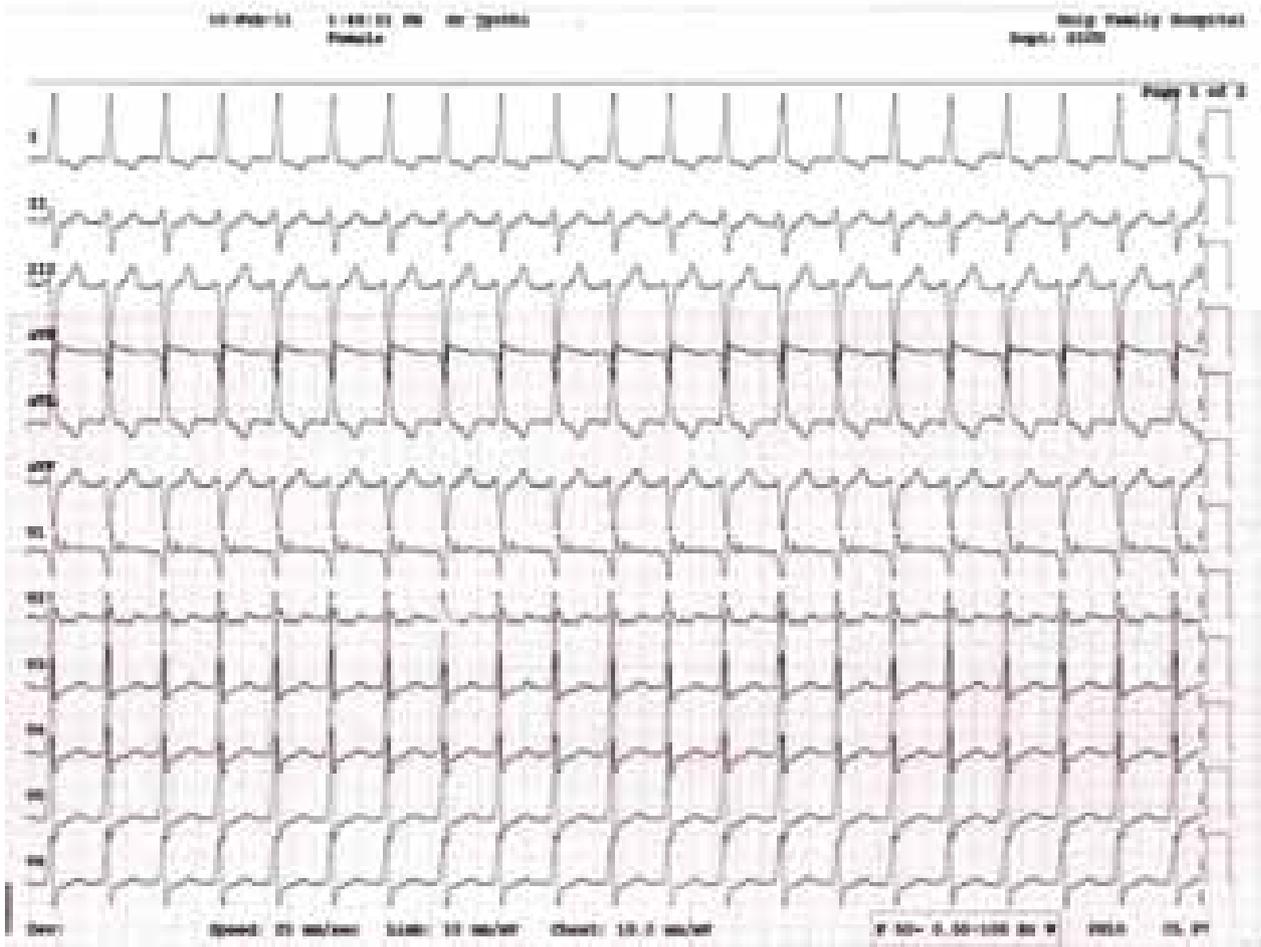
**Yash Lokhandwala\*, Gopi Krishna Panicker\*\***

\*Arrhythmia Associates; \*\*Quintiles Cardiac Safety Services, Mumbai

**The answers and explanations are  
on the reverse side of the page.**

**ECG - 1**

A 60 yr old woman with moderate LV dysfunction and past H/O syncope presented with rapid palpitations and uneasiness. The ECG on admission is shown below:



**The diagnosis is:**

- a. SVT
- b. Junctional tachycardia
- c. VT

For correct answer see overleaf

**ECG - 1**

The correct answer is 'c' – VT

The ventricular rate is 130 bpm. The QRS complex appears narrow at first sight. However, the widest QRS complex measured in leads V1 is 124 ms. Considering the arrow in lead V1 indicative of the second notch, the width would increase to 176 ms (see Figure below).



The QRS axis is  $-45^\circ$ . There is a QR morphology in lead V1. Wide QRS complexes with a QR morphology (in leads other than aVR and aVL) are invariably ventricular in origin. P waves are not clearly seen. There is a suggestion however that retrograde P waves are present immediately after the QRS complex in the inferior leads. If this is so, then the second notch in lead V1 ( $\rightarrow$ ) should be a retrograde P wave.

## ECG - 2

She was administered 80 mg of lignocaine intravenously.

The ECG after this is given below:



**This ECG shows:**

- Sinus tachycardia with pre-excitation
- Sinus tachycardia with IVCD
- Sinus tachycardia with VT
- Sinus tachycardia with junctional tachycardia

For correct answer see overleaf

**ECG - 2**

**The correct answer is ‘c’ – Sinus tachycardia with VT**

TA P wave precedes each QRS complex @ 120 bpm. The P waves are positive in I, II, aVF and V4-V6; hence these are normal P waves. Also note that P waves are peaked in lead V1. The QRS complex measures 140 ms in lead V2. The QRS axis is the same as before. The QRS morphology however, is different in leads V1-V3. The R waves are attenuated and the secondary notch in lead V1 is absent. The QR pattern persists in lead V1. Therefore these QRS complexes are also likely to be ventricular in origin. Crucially, the long lead II shows the PR interval to be gradually shortening. The first PR interval (F) is clearly much longer than the last PR interval (L).

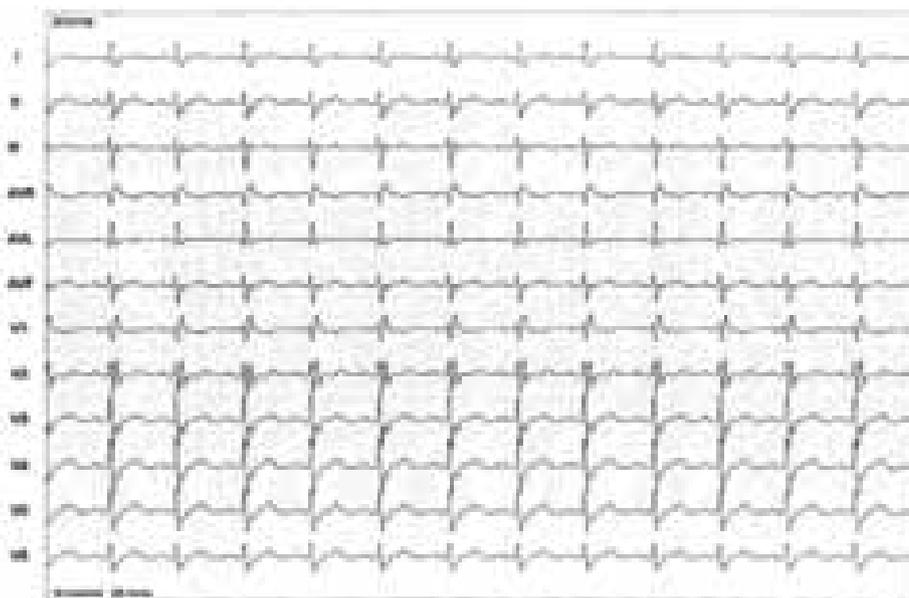


The QRS complexes and the RR intervals remain unchanged. Hence, there is isorhythmic A-V dissociation between sinus tachycardia with VT.

The secondary notch in lead V1 is absent. Retrospectively, therefore, the secondary notch in the first ECG was a P wave, most likely retrograde.

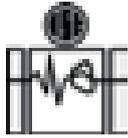
**Later the patient reverted to sinus rhythm.**

**The ECG is shown below:**



This ECG shows underlying RBBB with LAD and the QRS width is 160 ms in lead V2. This completely different QRS morphology confirms the previous ECG as VT. It also elegantly demonstrates the following principle:

“If a QRS complex during tachycardia is *‘narrower’* than in sinus rhythm, it is always VT”.



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Thanking you,

Yours sincerely,

Signature of the Applicant

Proposed by (the Member of the Society)

Name \_\_\_\_\_

Address \_\_\_\_\_

Signature \_\_\_\_\_

**FOR OFFICE USE ONLY**

**Recommendations of the  
Executive Body /  
Credential Committee**

Accepted / Not Accepted

**Life Membership No.**

**Hon. Secretary, ISE**

## **RULES/REGULATIONS OF THE SOCIETY REGARDING ADMISSION OF LIFE MEMBERS/FELLOWSHIP**

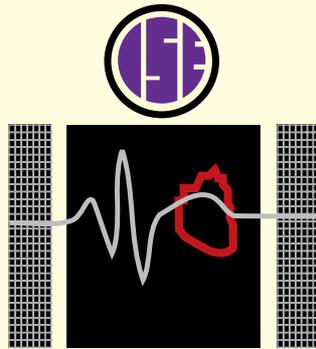
- \*Life Members :**
1. Person should be a Post-Graduate in Medicine/ Pediatrics/Anaesthesia/ Physiology or other allied subjects from an University recognised by Medical Council of India, with interest in Cardiology / Electrocardiology.
  2. Candidates are requested to submit **Xerox** copies of the PG Certificate and Medical Council of India Registration Certificate alongwith Application Form.

- \*\*Fellowship:**
1. Person should be a Member of the Society.
  2. He/She should be of atleast 7 years of standing after Post-Graduation.
  3. He/She should have minimum 3 publications In Cardiology In Indexed Journals (Not Abstracts)
  4. List of Publications to be submitted for the Fellowship.
  5. Fellowship Fees: Rs.2,000/- (+Rs.100/- for outstation cheque) only. Incase, fellowship not approved by the Credential Committee, the cheque / draft will be returned.

\*Subject to approval of the Executive Body of the Society

\*\*Subject to the approval of the Credential Committee of the Society.





SECRETARIAT  
**S. B. GUPTA**  
VICE PRESIDENT

**Indian Society of Electrocardiology**

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