Review Article

Ventricular Arrhythmias: From the Electrophysiology Laboratory to Clinical Practice

Part I: Malignant Ventricular Arrhythmias

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Ventricular arrhythmias represent a common problem for the clinical cardiologist in everyday practice.[1-5] Apart from being commonly asymptomatic, they may manifest with a variety of clinical symptoms, such as palpitations, "strong" precordial beats, "missing" heart beats, chest pounding, or occasionally episodes of long-lasting tachycardia accompanied with dyspnoea, chest discomfort, hypotension and syncope. However, most concerns are directed towards the risk of sudden cardiac death (SCD) due to the unpredictable occurrence of sustained ventricular tachyarrhythmias.[4] Thus, risk stratification for SCD should always be among the first priorities for all patients presenting with ventricular arrhythmias.[6-7] According to the findings of this risk stratification process and of the clinical presentation, the most appropriate treatment plan will be selected, including simple measures such as regular follow up with psychological support and symptomatic treatment with antiarrhythmic medication, as well as more complex therapeutic interventions, such as endocardial catheter ablation of the arrhythmogenic foci and prophylactic insertion of automatic implantable cardioverter defibrillators (AICD) with adequate treatment of all co-existing haemodynamic or ischaemic abnormalities.[8-26]

The electrophysiology (EP) laboratory provides us with great opportunities in patients with potential life-threatening ventricular arrhythmias who are at risk for SCD, in terms of completing the risk stratification, implementation of all interventional therapies previously mentioned and effective suppression of ventricular arrhythmias in cases where antiarrhythmic pharmacological treatment has failed. On the other hand, the role of the electrophysiological study (EPS) is limited to research purposes when ventricular arrhythmias are considered to be "benign", after non-invasive electrocardiographic and haemodynamic assessment.

Classification of ventricular arrhythmias

Ventricular arrhythmias could be classified into 3 diagnostic groups, depending on their complexity in ambulatory as well as in 12-lead electrocardiography.[3] This classification, however, mainly reflects the needs of the medical community for a common methodological approach to these conditions and consequently the implementation of the best diagnosis and treatment plan. In reality, they represent a

continuous spectrum ranging from the "benign" sporadic ventricular ectopic beats to the malignant ventricular arrhythmias, such as sustained ventricular tachycardia (VT) and ventricular fibrillation (VF). In between there is the group of potentially malignant arrhythmias, such as very frequent premature ventricular contractions, ventricular couplets and episodes of non-sustained VT (i.e. VT lasting less than 30 s, usually in the form of 3 to 10 consecutive ventricular complexes) (Table 1).

The degree of SCD risk depends mainly on the nature of the underlying heart disease, the extent of the ventricular dysfunction, the presence of ischaemia or haemodynamic instability, the functional condition of the autonomic nervous system, and the presence of sites of slow ventricular conduction in the right or left ventricle. Prior to classifying ventricular arrhythmias as "benign", potentially malignant or malignant, a non-invasive electrophysiological assessment should take place, including the patient's medical history, echocardiographic findings, exercise testing, and signal-averaged electrocardiogram (SAECG) results. Thus, even sporadic and rare ventricular ectopic beats without complex morphology should be considered as potentially malignant ventricular arrhythmias in patients with severe ventricular dysfunction, positive late potentials and a history of pre-syncopal and/or syncopal attacks, while on the other hand, the presence of idiopathic sustained monomorphic VT is associated with a relatively low risk of SCD. The clinical value of this complex system of non-invasive assessment and risk stratification is important, thus limiting the invasive electrophysiological diagnostic approach and treatment to those patients with potentially life-threatening or malignant ventricular arrhythmias. Moreover, programmed ventricular stimulation (PVS), aiming to reveal a well-organised electrophysiological substrate responsible for a future episode of sustained ventricular tachyarrhythmia, should be selectively implemented in those patients with ventricular arrhythmias in whom the pharmacological or non-pharmacological antiarrhythmic intervention would probably result in not only a better life expectancy, but also a better quality of life.

In the present paper, the electrophysiological methods for investigating malignant ventricular arrhythmias will be reviewed as they currently apply to the EP laboratory. In the second part of this paper we will present the different electrophysiologically guided approaches to investigate the great variety of ventricular arrhythmias, ranging from the benign to potentially malignant.

Malignant ventricular arrhythmias

Coronary heart disease (CHD) is the commonest cause of sustained ventricular arrhythmias. In most cases, VF or polymorphic VT is the consequence of acute coronary ischaemia, whereas sustained monomorphic VT in patients with structural heart disease is usually due to a myocardial scar resulting from a prior infarct, or other causes of non-ischaemic cardiomyopathies, through re-entry mechanisms.[27]

Sustained monomorphic VT that is not related to structural heart disease can be successfully treated with endocardial ablation of its site of origin through radiofrequency current intracardiac catheters (radiofrequency catheter ablation, RCA).[8,9,19-21] The site of origin is usually located in the right ventricular outflow tract, and occasionally in other locations of the right ventricle (RV) or in the posterior inferior septal wall of the left ventricle (LV). When deriving from the RV, ventricular tachycardia is characterised by a left bundle branch block (LBBB) pattern with a varying axis depending on the site of origin, whereas

Table 1. Classification of ventricular arrhythmias.

	Malignant ventricular arrhythmias	Potentially malignant ventricular arrhythmias	Benign ventricular arrhythmias
Electrocardiographic characteristics	Sustained VT or VF	Non-sustained VT, frequent (≥ 30/hour) PVCs and ventricular couplets	Sporadic PVCs
Organic heart disease	Usually present	Most commonly present	Absent
Prognosis and risk of sudden cardiac death	Impaired in the presence of organic heart disease	Depending on EPS results and severity of underlying organic heart disease	Benign

VT – ventricular tachycardia; VF – ventricular fibrillation; PVCs – premature ventricular contractions; EPS – electrophysiological study.

when deriving from the LV, it demonstrates a right bundle branch block (RBBB) morphology with left axis. In order for the electrophysiological mapping to be completed, reproducible induction of the arrhythmia with PVS is required. The site of origin of the ventricular arrhythmia is identified by recording presystolic electrograms during the induced tachycardia or by reproducing the 12-lead morphology of the arrhythmia when pacing from the presumptive target site while in sinus rhythm. In cases where the underlying mechanism is ventricular re-entry, determination of the exact site of origin is feasible by meeting the entrainment criteria, an electrophysiological technique that contributes to the safest and most effective ablation by limiting the number of unnecessary lesions applied.[11,17,18] RCA in idiopathic VT is reserved for patients who do not respond to medical treatment and is performed successfully in more than 80% of the cases, with a low rate of complications or future relapses.[27]

The effectiveness of VT RCA in patients with structural heart disease is lower, varying from 50% to 80%.[27] Ablation in disorders other than post-infarction cardiomyopathy is often more difficult and recurrence of VT more frequent.[27] In patients presenting with sustained VT or VF the optimal treatment is AICD implantation.[27-29] However, it is not uncommon for recurrent episodes of VT that are not controlled with antiarrhythmic agents to lead to repeat AICD activation, resulting in a deterioration in the patient's quality of life. Furthermore, AICDs do not provide absolute protection against SCD, with an estimated incidence of non-response of 5%.[30] These patients, in addition to those with incessant VT, could be treated effectively by modification of the arrhythmological substrate by RCA.[10-17,27,31,32] RCA might also be an alternative to AICD implantation in certain population subgroups such as the elderly.[33,34] In a recent trial, patients who received AICD plus VT-ablation, often described as hybrid therapy, demonstrated a lower incidence of appropriate AICD activation.[35] In another recent study, prophylactic RCA before AICD implantation was suggested in post myocardial infarction (MI) patients who manifested VT and a reduced left ventricular ejection fraction (LVEF) ≤50%.[36] In these cases, the underlying structural cardiac disease—which is most usually post-infarction CHD, but not infrequently dilated cardiomyopathy, operated congenital heart disease or arrhythmogenic right ventricular cardiomyopathy/dysplasia—leads to the formation of one or more sites of ventricular re-entry (scar-related VT), from which frequent or even unsuppressed ventricular ectopic activity may originate. The morphology of VT in the 12-lead electrocardiogram (ECG) provides us with important information about the approximate anatomical location of origin of the arrhythmia. In cases where more than one morphology of VT is recognised, it is possible that multiple foci of VT, or alternatively an extended myocardial scar resulting in a re-entry circuit with more than one exit tract, are present (Figure 1).

Reproduction of the 12-lead morphology of the VT during pacing, at the presumptive site of origin, is of great importance for the exact localisation of the target site (Figure 2). Furthermore, detection of pre-systolic or, even better, mid-diastolic electrograms during mapping is of great help in identifying the ideal target site (Figures 3 & 4).[9,10,27] Meeting the entrainment criteria of VT when pacing from the slow conduction area, and specifically from its exit tract site, is reassuring evidence for exact locali-

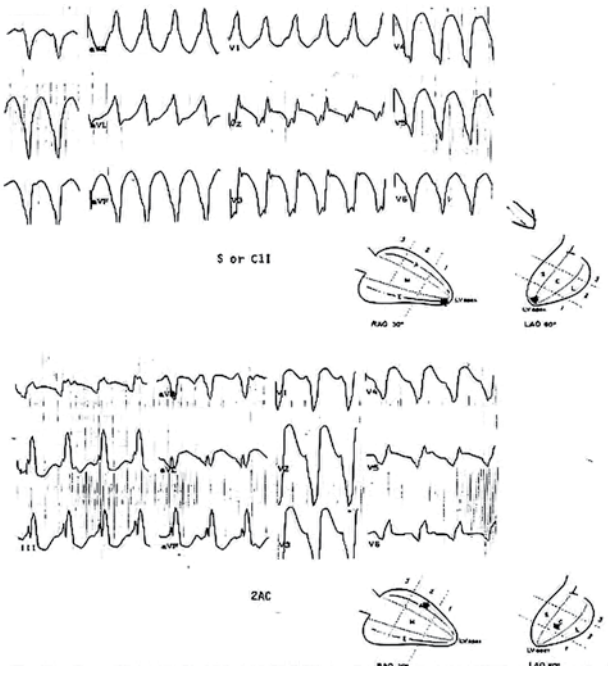


Figure 1. Localisation of the site of origination of sustained ventricular tachycardia (VT) by means of the electrocardiogram (ECG). A 12-lead ECG of a 70-year-old, post-myocardial infarction patient, showing two different types of sustained VT, not suppressed with amiodarone. Based on the electrocardiographic morphology and the axis of the sustained VT, its exit tract sites can be localised at the apex of the left ventricle and the middle section of the intraventricular septum (S or CIL position and 2AC position according to Kuchar and Josephson, respectively).

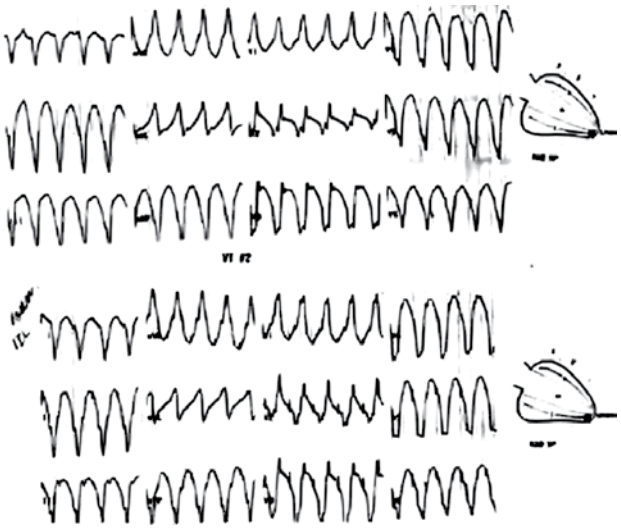


Figure 2. Electrophysiological study. Reproduction of the 12-lead morphology of the ventricular tachycardia (VT) during pacing. Pacing from the apex of the left ventricle, at a cycle length similar to that of the clinical tachycardia, results in reproduction of the 12-lead electrocardiographic morphology of the sustained VT.

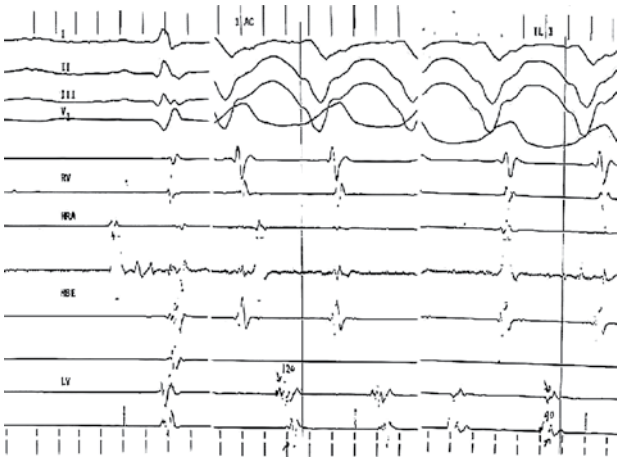


Figure 3. Identification of the arrhythmogenic site with presystolic electrograms during the electrophysiological study. Presystolic electrograms 120 ms and 90 ms before the tachycardia complex are recorded in the two different exit tract sites of the sustained ventricular tachycardia. From top to bottom, surface leads I, II, III and V_1 are shown, followed by the endocardial electrograms of the right ventricle (RV), the high right atrium (HRA), the His bundle (HBE) and the zone surrounding the infarction area of the left ventricle (LV).

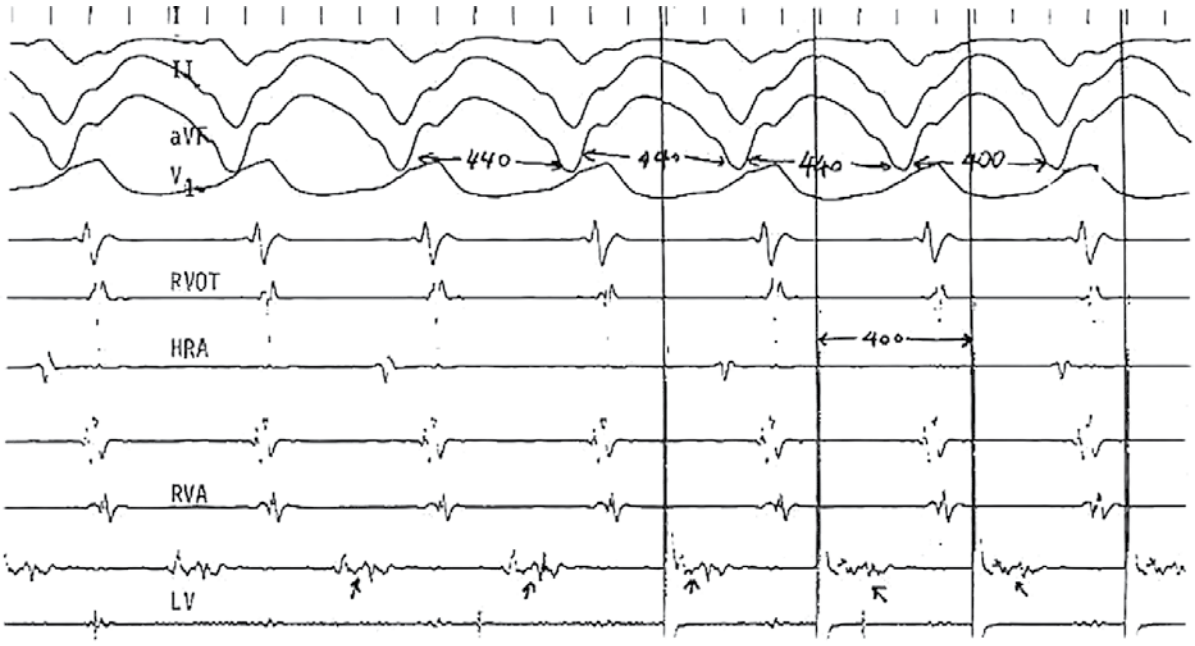


Figure 4. Entrainment of ventricular tachycardia during electrophysiological study. During the sustained ventricular tachycardia (VT), in the slow conduction site (LV) of the zone surrounding the infarction area, low amplitude, long duration multi-fragmented electrical activity is observed in the middle of the cardiac cycle (mid-diastolic electrograms). Pacing from this site, while the patient remains in sustained VT, results in entrainment of the VT at the paced cycle length (acceleration from 440 ms to 400 ms) without any changes in the surface lead morphology. From top to bottom, surface leads I, II, aVF and V_1 are shown, followed by the endocardial electrograms of the right ventricular outflow tract (RVOT), the high right atrium (HRA), the apex of the right ventricle and the zone surrounding the infarction area of the left ventricle (LV).

sation and hence for safe and effective ablation (Figure 4).[9-11,16-18] Specifically, when pacing from the slow conducting area of the ventricular VT site of origin, at a cycle length shorter to that of the induced sustained VT, we entrain the VT at the pacing rate without changing its 12-lead ECG morphology and axis, while when the pacing is interrupted the returning cycle length remains identical to that of the induced VT. When ablating at the corresponding entrainment site, there is a higher success rate compared to lesions applied in areas of early pre-systolic activation and pace-mapping reproduction of the 12-lead ECG VT morphology. However, in focal VT demonstrating a point source of endocardial activation, the entrainment criteria cannot be used and distinction from macro–re-entrant VT is important because the ablation site characteristics are very different.[37] On occasion, it is interesting to observe an atrioventricular-node–like behaviour with decremental properties within the slow conduction area of the VT site of origin (Figure 5). Recently, numerous "modern" mapping technologies have been developed, resulting in increased success rates of VT catheter ablation.[38] These techniques are based on colourful electro-anatomical reproduction of the ventricular cavity of interest with either activation and/or voltage mapping

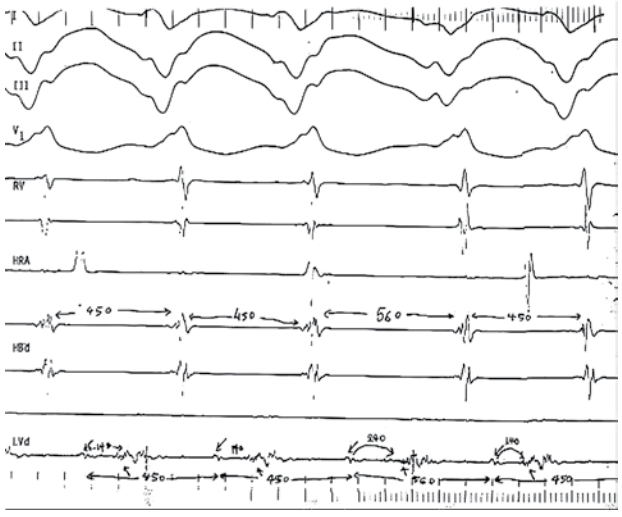
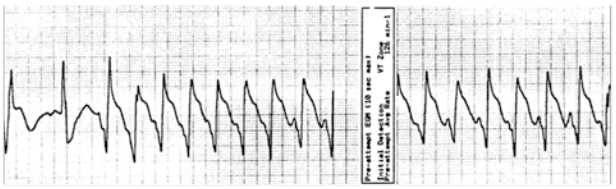


Figure 5. Atrioventricular-node–like behaviour with decremental properties within the slow conduction area of the ventricular tachycardia site of origin. Self-termination of sustained ventricular tachycardia (VT). In the slow-conduction area of the intraventricular re-entry circuit, prolongation of the distance between the mid-diastolic and pre-systolic electrograms is observed occasionally, before the former are blocked in the circuit, in the last sustained VT complex. The sequence of the electrocardiographic leads and electrograms is as in Figure 3.

performed through specially designed recording and ablation catheter systems introduced into the LV or RV cavities. The mapping could be performed either during sinus rhythm or during the induced VT. Thus, areas of slow conduction and low voltage, as well as of late potentials and mid-diastolic activity, can be identified during sinus rhythm, representing abnormal scar tissue sites of interest. Similarly, activation mapping during spontaneous or induced ventricular ectopy may identify early activation sites of interest. All of these sites represent potential ablative lesion areas which could be "modified" by "drawing" lines between them until the target VT is either completely suppressed or more difficult to induce. Apart from achieving higher success rates, such techniques significantly also limit the radiation exposure to both invasive electrophysiologists and patients.

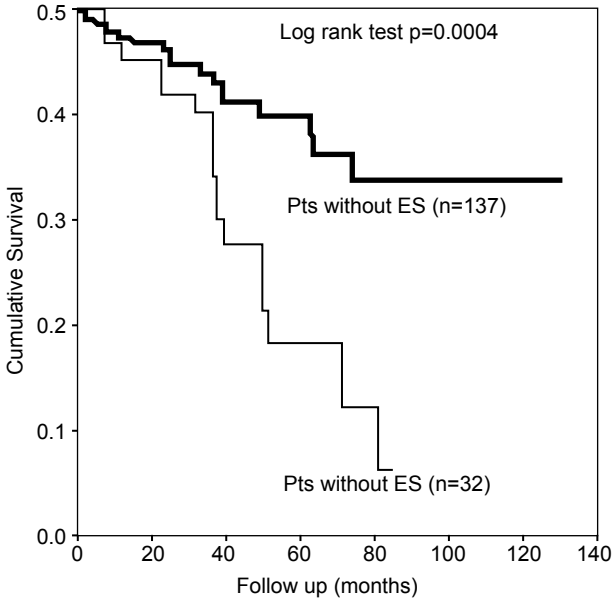
These observations, in addition to corresponding findings from the SAECG supporting the formation of organised areas of slow ventricular conduction, probably explain the occurrence of episodes of electrical storm in patients with a history of malignant ventricular arrhythmias treated with an AICD (Figure 6). Electrical storm, namely the non-predictable occurrence of at least 3 episodes of sustained ventricular tachyarrhythmias in a period less than 24 hours, is a major arrhythmic event presenting as a medical emergency in 1 out of 5 patients receiving an AICD for the secondary prevention of SCD.[38-41] Both short- and long-term prognoses seem to be impaired, although acute management with an antiarrhythmic drug combination regimen is effective in the vast majority of patients affected (Figure 7).[39-50] In fact, an advanced New York Heart Association (NYHA) heart failure stage and the occurrence of electrical storm emerge as the most important independent mortality predictors among patients managed with an AICD.[41,44-46] Some authors, however, argue that electrical storm is frequent but does not increase mortality in AICD recipients.[40,43,48] At the present time, it is difficult to predict the AICD recipient who is going to be affected by electrical storm.[48] Reversible ischaemic, metabolic, haemodynamic or electrolytic abnormalities are not usually detected during the acute event. However the incidence of electrical storm may be higher among AICD patients receiving the device for the secondary prevention of SCD, with severe systolic dysfunction, when the presenting arrhythmia is VT and not primary VF, or with coexisting renal dysfunction.[40,41,43,44,48,51-53] The role of other well accepted risk stratification factors from 12-lead electrocar-



Therapy History — Data Since Counters Cleared on: 06-MAY-1999

Epsd	Date/Time	Stb ms	Onset %	Pre min-1	Therapy			Post min-1
471	13-JAN-2001 20:34	64	25				No Attempt	
470	13-JAN-2001 19:49	57	N/A				No Attempt	
468	13-JAN-2001 19:22	88	28				No Attempt	
467	13-JAN-2001 18:43	14	N/A				No Attempt	
466	13-JAN-2001 18:42	6	N/A				No Attempt	
465	13-JAN-2001 18:41	27	38	129	VT		No Therapy	127
464	13-JAN-2001 18:40	80	0				No Attempt	
463	13-JAN-2001 18:38	10	N/A				No Attempt	
462	13-JAN-2001 18:09	18	41	128	VT		No Therapy	127
461	13-JAN-2001 18:08	31	44	126	VT		No Therapy	119
460	13-JAN-2001 17:56	21	0	118	VT		No Therapy	119
459	13-JAN-2001 17:54	25	41	124	VT		No Therapy	123
458	13-JAN-2001 16:55	6	13	129	VT		No Therapy	130
456	13-JAN-2001 15:13	4	0	122	VT		No Therapy	123
455	13-JAN-2001 15:12	18	34	122	VT		No Therapy	123
453	13-JAN-2001 13:54	25	28	124	VT		No Therapy	122
449	13-JAN-2001 13:19	27	31	120	VT		No Therapy	119
448	13-JAN-2001 13:14	25	47	122	VT		No Therapy	120
447	13-JAN-2001 13:12	23	31	120	VT		No Therapy	120
445	13-JAN-2001 13:02	27	31	121	VT		No Therapy	117
444	13-JAN-2001 10:52	N/A	N/A	115			5 J	80
443	13-JAN-2001 10:50	N/A	N/A	115		1	Burst	113
442	13-JAN-2001 10:50	N/A	N/A	115		1	Scan	113
441	13-JAN-2001 10:49	N/A	N/A	114		2	Ramp/Scan	112
440	13-JAN-2001 10:48	N/A	N/A	115		3	Ramp/Scan	112
439	13-JAN-2001 10:48	N/A	N/A	115		1	Ramp/Scan	113
438	13-JAN-2001 10:47	N/A	N/A	115		1	Ramp/Scan	113
437	13-JAN-2001 10:46	N/A	N/A	115		1	Ramp/Scan	113
436	13-JAN-2001 10:46	N/A	N/A	113		1	Ramp/Scan	113
434	11-JAN-2001 13:36	18	31	119	VT	1	Ramp/Scan	57
433	11-JAN-2001 09:45	14	34	121	VT	1	Ramp/Scan	117
432	11-JAN-2001 05:17	16	41	124	VT	1	Ramp/Scan	60
431	09-JAN-2001 10:10	6	41	146	VT	1	Ramp/Scan	81
430	09-JAN-2001 10:09	10	47	143	VT	2	Ramp/Scan	203
		2	--	205	VF		31 J	108
429	09-JAN-2001 09:39	4	50	147	VT	1	Ramp/Scan	115
428	09-JAN-2001 09:21	10	44	146	VT	2	Ramp/Scan	117
427	09-JAN-2001 09:17	6	34	146	VT	4	Ramp/Scan	77
426	09-JAN-2001 09:15	14	38	143	VT	1	Ramp/Scan	197
		2	--	202	VF		31 J	88
425	08-JAN-2001 09:43	20	41	150	VT	1	Ramp/Scan	92
424	08-JAN-2001 09:43	16	41	151	VT	1	Ramp/Scan	86
423	08-JAN-2001 09:26	10	16	144	VT	1	Ramp/Scan	121
422	08-JAN-2001 01:44	8	38	145	VT	2	Ramp/Scan	82
421	08-JAN-2001 00:15	18	38	146	VT	1	Ramp/Scan	74
420	08-JAN-2001 00:14	8	50	145	VT	1	Ramp/Scan	78
419	06-JAN-2001 08:34	2	0	121	VT	1	Ramp/Scan	120
418	06-JAN-2001 08:14	16	31	138	VT	2	Ramp/Scan	96
417	31-DEC-2000 14:51	18	41	135	VT	1	Ramp/Scan	121
416	31-DEC-2000 10:27	10	38	148	VT	1	Ramp/Scan	104
415	29-DEC-2000 15:28	10	41	156	VT	1	Ramp/Scan	103
414	29-DEC-2000 14:43	10	41	153	VT	1	Ramp/Scan	131
413	29-DEC-2000 10:25	6	47	146	VT	1	Ramp/Scan	92
412	28-DEC-2000 17:14	16	41	138	VT	1	Ramp/Scan	81
411	28-DEC-2000 17:10	16	44	148	VT	2	Ramp/Scan	102
410	28-DEC-2000 09:58	10	44	146	VT	2	Ramp/Scan	103
409	27-DEC-2000 15:36	12	41	150	VT	3	Ramp/Scan	98
408	27-DEC-2000 15:36	14	44	149	VT	2	Ramp/Scan	97
407	27-DEC-2000 15:31	10	38	151	VT	2	Ramp/Scan	95
406	27-DEC-2000 15:29	12	44	146	VT	1	Ramp/Scan	103
405	27-DEC-2000 10:00	14	38	141	VT	4	Ramp/Scan	103
404	26-DEC-2000 13:33	4	47	144	VT	1	Ramp/Scan	84
403	25-DEC-2000 09:43	12	34	145	VT	2	Ramp/Scan	94
402	23-DEC-2000 16:17	14	53	135	VT	1	Ramp/Scan	72
401	22-DEC-2000 09:53	10	47	143	VT	1	Ramp/Scan	86
400	21-DEC-2000 10:45	23	44	138	VT	1	Ramp/Scan	87
399	20-DEC-2000 10:00	10	38	137	VT	1	Ramp/Scan	90
398	19-DEC-2000 13:32	12	41	135	VT	2	Ramp/Scan	134
396	10-DEC-2000 16:55	2	13	144	VT	1	Ramp/Scan	78
395	10-DEC-2000 15:59	6	41	142	VT	1	Ramp/Scan	119
394	10-DEC-2000 15:55	12	47	144	VT	1	Ramp/Scan	75
393	10-DEC-2000 15:53	8	38	142	VT	1	Ramp/Scan	81
392	10-DEC-2000 15:46	6	N/A	144	VT	1	Ramp/Scan	89

Figure 6. Electrical storm revealed during interrogation of an automatic implantable cardioverter defibrillator (AICD) in a patient with coronary heart disease. Interrogation of the device in a 65-year-old, post myocardial infarction patient, with a history of sustained ventricular tachycardia (VT) and an AICD implantation presenting four years later with multiple episodes of sustained VT. The interrogation revealed multiple VT episodes within a few days, successfully interrupted with anti-tachycardia pacing or defibrillation shocks. Electrical storm was successfully treated by means of a triple antiarrhythmic drug combination (amiodarone, metoprolol and mexiletine).



Patients at risk	0 mo	20 mo	40 mo	60 mo	80 mo
No ES	137	78	39	24	8
ES	32	27	12	5	2

Figure 7. Survival curve in electrical storm. Probability of survival of automatic implantable cardioverter defibrillator (AICD) patients according to the presence of electrical storm. (From: Gatzoulis KA, Andrikopoulos GK, Apostolopoulos T, et al. Electrical storm is an independent predictor of adverse long-term outcome in the era of implantable defibrillator therapy. Europace 2005; 7: 184-192. Reproduced with permission from Oxford University Press.)

diography, ambulatory electrocardiography, SAECG or T-wave alternans has not been studied. The combination of a lower LVEF with the presence of late potentials is associated with a higher rate of both AICD activation and cardiac mortality.[54]

The role of RCA in electrical storm, aiming towards a favourable modification of the arrhythmological substrate, remains unclear. However, a number of studies have suggested that it not only results in a better quality of life but may also improve the impaired prognosis.[55-58] On the other hand, in patients with dilated cardiomyopathy, RCA was less effective compared with patients with CHD and arrhythmogenic right ventricular cardiomyopathy/dysplasia.[58] Recent data suggest a reduction in the incidence of AICD activation including electrical storm recurrence, favourably affecting mortality, among AICD recipients on optimal antiarrhythmic medication, when they undergo RCA.[58] Such a reduced incidence of AICD activation can also be achieved with antiarrhythmic drugs such as b-blockers, sotalol, and amiodarone, especially when b-blockers are combined with amiodarone.[27]

The increased incidence of AICD activation, with the associated depression, may lead to worsening of quality of life. Thus, every effort to reduce it is highly desirable. Such efforts may also include the appropriate anti-tachycardia pacing capabilities of the device

programming in order to interrupt silently even fast episodes of VT.[59] In these cases, as well as in patients with idiopathic VT in whom it is not always possible to induce sustained ventricular tachycardia, invasive electrophysiology, using novel electro-anatomical mapping techniques seems to be promising (Figure 8).[42,57,58,60] Thus, short-duration episodes of VT or even sporadic ventricular ectopic beats can be tracked and ablated.[22] The effectiveness and safety of these techniques of endocardial ablation are currently under investigation, not only expanding the treating options, but also limiting the radiation exposure for patients and invasive electrophysiologists.

Apart from RCA of the VT site of origin, in the EP laboratory we safely and effectively perform AICD implantations in high-risk cardiac patients who have had a previous spontaneous sustained ventricular tachyarrhythmia event (secondary prevention of SCD), or whenever the risk stratification process defines such a risk in the near future (primary prevention of SCD). Detailed reviews regarding the clinical and laboratory indications for the implantation of an AICD in high-risk patients, the implantation techniques and the long term follow up of these patients have been published in the past.[23-27,29,61,62] Recent technological improvements in the field of endocardial defibrillation, the expanding experience of the implantation centres, as well as the well-documented positive impact of AICDs on survival in patients with structural heart disease and a history of spontaneous or/and induced malignant ventricular arrhythmias, have resulted in a tremendous increase in the number of AICD implantations worldwide, although there are still striking differences between the two sides of the Atlantic.[63]

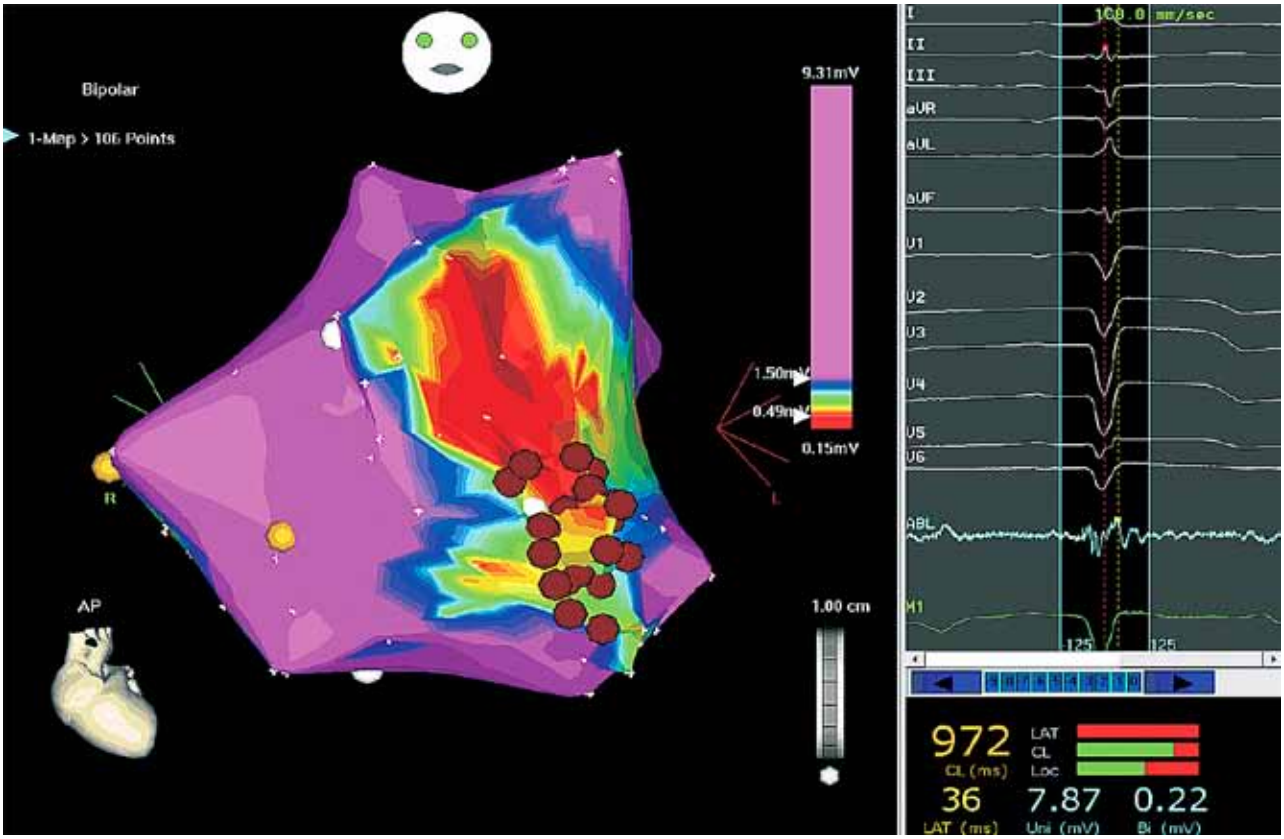


Figure 8. Electroanatomical mapping in a patient with electrical storm. A 65-year-old patient with coronary heart disease presented with repeat episodes of sustained ventricular tachycardia (electrical storm) prior to automatic implantable cardioverter defibrillator (AICD) implantation. The episodes were treated with triple antiarrhythmic medication and subsequent ablation/modification of the arrhythmogenic substrate using a three-dimensional colour electro-anatomical mapping system (potential map). Five years later the patient remains in stage II heart failure while the AICD has been successfully activated with anti-tachycardia pacing only once, three months after the ablation and AICD implantation (Modified from: Gatzoulis KA, Sideris SK, Kallikazaros IE, Stefanadis CI. Electrical storm: a new challenge in the age of implantable defibrillators. Hellenic J Cardiol. 2008; 49: 86-91.)

The role of electrophysiological intervention, however, is not limited only to the diagnosis and treatment of high-risk patients. Occasionally, patients with a history of malignant ventricular arrhythmias are treated with non-antiarrhythmic or even antiarrhythmic surgery, aiming at mechanical restoration of the detected ischaemic or haemodynamic dysfunctions and modification of the arrhythmia substrate. Patients with CHD, aneurysm of the left ventricle, valvular heart disease or idiopathic hypertrophic obstructive cardiomyopathy should be reassessed in the postoperative period with PVS.[64-67] It is not unusual for a patient with CHD and a history of cardiac arrest and induced ventricular fibrillation on PVS to remain electrically stable after revascularisation, especially when there is no severe left ventricular dysfunction or presence of late potentials. Furthermore, aneurysmectomy of the left ventricle with disappearance of the pre-existing late potentials may be associated with an inability to re-provoke a previously easily triggered VT on PVS (Figure 9).[64,65] It is unlikely, however, that this would occur in a patient with a history of sustained monomorphic VT when severe dysfunction of the left ventricle and the presence of late potentials are still present postoperatively (Figure 10).

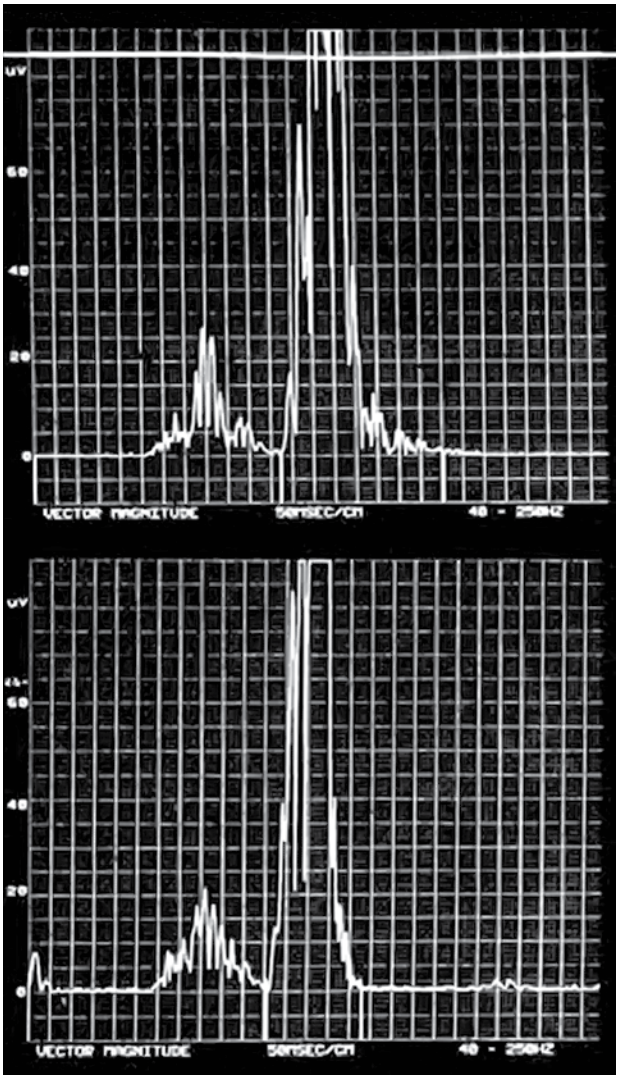


Figure 9. Elimination of late potentials in the signal averaged electrocardiogram (SAECG) after aneurysmectomy. SAECG performed in a 64-year-old post myocardial infarction patient with left ventricular aneurysm before (fQRS = 191 ms, RMS-40 = 1 μV, LAS = 104 ms) and after (fQRS = 113 ms, RMS-40 = 16 μV, LAS = 35 ms) aneurysmectomy of the left ventricle showing disappearance of the pre-existing late potentials. The presenting sustained ventricular tachycardia (VT) (both spontaneous and induced preoperatively) was not induced postoperatively.

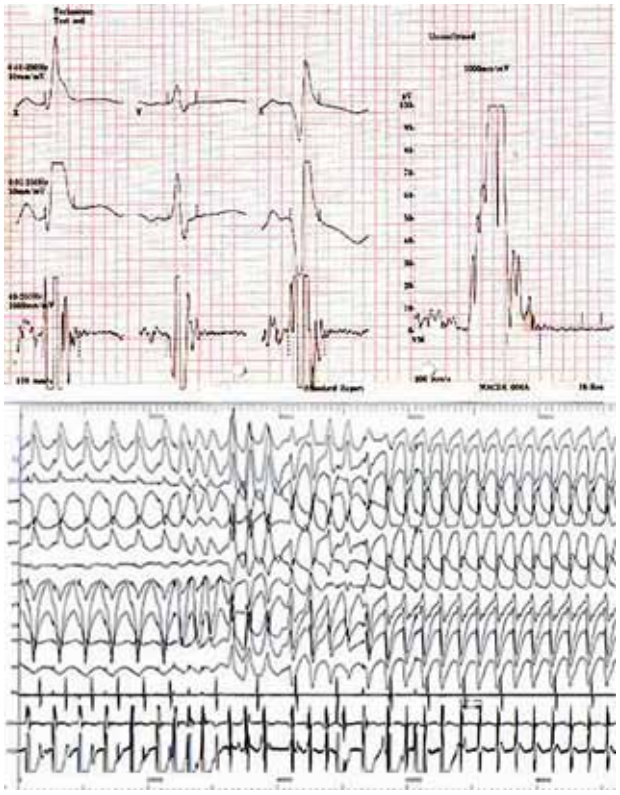


Figure 10. A signal averaged electrocardiogram (SAECG) and electrophysiological study (EPS) in a patient with coronary heart disease. An SAECG and EPS in a middle-aged post myocardial infarction patient who presented with sustained ventricular tachycardia (VT) and underwent a triple coronary artery bypass surgery. Postoperatively, pre-existent positive late potentials persisted and additionally sustained monomorphic VT was induced. The patient remains alive and in good clinical condition (no angina, left ventricular ejection fraction 40%, New York Heart Association class II) 9 years later after the second automatic implantable cardioverter defibrillator (AICD) replacement, with 6 episodes of sustained VT interrupted by the device.

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