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A Review of ICD Anti-Tachycardia Therapy Programming with Generic Programming for Primary and Secondary Prevention

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Abstract

Intracardiac defibrillator plays a pivotal role in preventing sudden cardiac death; however, inappropriate shock delivery remains an important source of morbidity and mortality. Advancements in device technology along with various shock reduction strategies play a key role in reducing inappropriate and unnecessary shocks. Anti-tachycardia pacing (ATP) is the first-line therapy prior to shock delivery. Several trials have validated the efficacy of ATP for both slow and fast ventricular tachycardia without significant increase in occurrence of arrhythmia-related syncope. In addition, trials also support that therapy for non-sustained tachycardia can be prevented by higher programmed zones and prolonged intervals to detect without higher risk of syncope. With this perspective, authors employ a customized programming for both primary and secondary prevention to reduce inappropriate therapies or unnecessary therapies, in particular, progression to shock but allow for spontaneous termination at slower ventricular tachycardia rates. The programming was instituted at the time of device implantation or at follow up.

Keywords: intracardiac defibrillator, anti-tachycardia pacing, inappropriate therapies, shock, customized programming, ICD programming, ICD therapies, reducing ICD therapies, ICD templates

1. Introduction

Implantable cardioverter-defibrillator (ICD) remains the main therapeutic option in reducing sudden cardiac death (SCD). Several randomized trials and registries have shown that ICD extends survival in patients with severe left ventricular function and mild-to-moderate heart failure [1–4]. The shocks delivered whether appropriate, inappropriate or unnecessary

remain an important source of mortality and morbidity from proarrhythmic potential, heart failure, painful delivery of shock causing significant anxiety, depression and post-traumatic stress disorder [5–11].

Inappropriate ICD shocks are those delivered for a condition other than true ventricular arrhythmias, which most commonly include supraventricular arrhythmias with rapid rates, mechanical failure of ICD lead/system like lead conductor fracture resulting in noise detection and non-mechanical issues such as T-wave over sensing resulting in double-counting [3]. Unnecessary or potentially avoidable shocks are those where the ventricular tachycardia (VT) was to terminate spontaneously or could have been interrupted by appropriately timed pacing stimuli.

Table 1 lists the major clinical trials and registries reporting the incidence of inappropriate shocks. We review some of the trials here.

The anti-arrhythmics versus implantable defibrillators (AVID) was a multi-centre trial which patients were randomized to receive ICD or anti-arrhythmic drug therapy; 492 patients were randomized to receive an ICD over a follow-up period of 22 ± 12 months. Inappropriate shocks in this cohort were due to supraventricular tachycardia in 18% and 3% were due to ICD malfunction or inappropriate sensing [12].

The Pain FREE Rx II trial was a prospective randomized control trial consisting of 634 patients with a mean follow up of 11 ± 3 months. All patients received ICDs and were randomized to anti-tachycardia pacing versus shock only programming [13]. There were 4230 spontaneous episodes retrieved from all implanted ICDs, 1837 had complete electrogram data and were included in the analysis. Of these, 491 episodes (27%) were determined to be inappropriately detected supraventricular tachycardia (SVT), and 4 (0.2%) were non-physiological artifact. Sweeny et al. performed a subgroup analysis of the PainFree trial and showed that the proportion of true ventricular detections that resulted in shocks was similar between primary and secondary prevention groups (40% versus 32%, respectively) [14]. The proportion of inappropriate ventricular detections due to SVT that resulted in shocks was also similar between primary and secondary prevention groups (44% versus 42%, respectively).

MIRACLE ICD was a prospective, randomized double-blind trial of 978 patients with a mean 10-month follow-up [15]. This trial evaluated the safety and efficacy of cardiac resynchronization combined defibrillator therapy (CRT-D) versus ICD only therapy in both primary and secondary prevention patients. The reported incidence of inappropriate shocks was 30% in primary prevention patients and 14% in secondary prevention patients.

In the Multicenter Automatic Defibrillator Implantation Trial II (MADIT-II), inappropriate shocks constituted 31.2% (184/590) of the total shock episodes [16]. The most common triggers were atrial fibrillation (44%) and supraventricular tachycardia (36%) with improper discrimination by the ICD device, followed by abnormal sensing (20%). The majority of inappropriate ICD therapy episodes were delivered for rhythms below or equal to 200 bpm; the mean ventricular rate triggering inappropriate shock for atrial fibrillation (AF) or SVT was 174 ± 22 bpm. Patients with inappropriate ICD shocks showed a significantly higher mortality during the follow-up (HR = 2.29, 95% CI: 1.11–4.71, $p = 0.02$) than patients with

Clinical trial	AVID [12]	PainFREE [13]	MIRACLE ICD [15]	MADIT II [16]	SCD-Heft [17]	ALTITUDE ICD [18]	ALTITUDE CRT-D [18]	Leidin [19]
Patient no.	449	582	978	719	811	39,396	29,904	1544
Follow up, months	22	11	10	20	46	28	28	41
Single/dual	Single/dual	Single/dual	Single/dual	Single/dual	Single	Single/dual	Single/dual	Single/dual
Primary/secondary	Secondary	Primary/secondary	Primary/secondary	Primary	Primary	Primary/secondary	Primary/secondary	Primary/secondary
Inappropriate Rx, %	21	15	14–30*	12	17	16	17	18
Inappropriate Rx, (HR, 95% CI, <i>p</i>)	n.a	n.a	n.a	2.29 (1.11–4.71) 0.02	1.98 (1.29–3.05) 0.002	1.84 (1.30–2.61)	1.60 (1.15–2.23)	1.60 (1.10–2.30) <i>p</i> = 0.01
Appropriate Rx, %	68	33	23–31**	21	23	23	23	n.a
Appropriate Rx, (HR, 95% CI, <i>p</i>)	n.a	n.a	n.a	3.36 (2.04–5.55) <0.01	5.68 (3.97–8.12) <0.001	2.05 (1.55–2.71)	2.51 (2.01–3.14)	1.60 (1.20–2.10) <0.01
ATP (yes/no)	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes
Outcome measure	n.a	n.a	n.a	Mortality	Mortality	Mortality	Mortality	Mortality

*14% in secondary prevention and 30% in primary prevention.
**23% in primary prevention and 31% in secondary prevention.

Table 1. List of ICD clinical trials and registries with frequency of appropriate and inappropriate therapy and outcome.

appropriate ICD shocks (HR = 3.36, 95% CI: 2.04–5.55, $p < 0.01$). This demonstrated that all shocks, although demonstrated to save lives, also have a detrimental effect and lead to heart failure deterioration and eventual mortality.

Similar data could be extrapolated from the sudden cardiac death in heart failure trial (SCD-HeFT) [17]. In this trial, 2521 patients with primary prevention indication and with mild-to-moderate heart failure were randomized in equal proportions to receive placebo, amiodarone or a single-chamber ICD programmed to shock-only mode. Follow-up was for an average of 46 months. In 811 patients assigned to the ICD arm, the rate of inappropriate shocks was 17% as compared to 22.4% appropriate shocks during a 46-month follow-up. Patients with an inappropriate ICD therapy had a twofold increase in the risk of all-cause mortality (HR = 1.98, 95% CI: 1.29–3.05, $p = 0.002$).

The results from the randomized trials were confirmed in larger registries. The ALTura Impact on the Treatment of Abdominal Aortic Aneurysms Using a Novel D-stent EVAR Design (ALTITUDE) registry involved 39,396 ICD patients and 29,904 patients implanted CRTDs. Patients were implanted for both primary and secondary indications [18]. The 1-year incidence of inappropriate shocks was 8% and 6% and at 5 years increased to 16% and 17% for ICD and CRT-D patients, respectively. The two most common reasons for shock were atrial flutter/atrial fibrillation and sinus tachycardia or supraventricular arrhythmia. Inappropriate shock was due to noise, artifact or over sensing in 3% of the episodes.

The Leiden group published a large scale study in 1544 ICD patients and reported an 18% incidence of inappropriate ICD therapy over 41 months of follow-up. This study also confirmed the increased risk of death for both inappropriate and appropriate ICD therapy (HR1.60 for both, $p = 0.01$ for inappropriate; $p < 0.01$ for appropriate ICD therapy) [19].

2. Possible mechanism of increased risk of death with ICD shocks

It may be hypothesized that there is a direct mechanical or hemodynamic effect of the inappropriate ICD therapies themselves or that some of the inappropriate ICD therapies lead to fatal pro-arrhythmia due to an increase in sympathetic discharge, which in turn leads to rate-related changes in ventricular refractoriness and worsened myocardial ischemia [20].

It is likely that it is not the inappropriate shock *per se* that is detrimental, but the sequelae that hastens the adverse clinical outcome.

It has been suggested that high shock fields are associated with changes in electrophysiological properties of the heart and can be the primary source of activation wave fronts that may give rise to idioventricular rhythms after the shock and perhaps may even perpetuate ventricular fibrillation [21]. The cellular injury from intra-cardiac shock delivery whether it be appropriate or not is reflected in a rise in cardiac troponin I [22]. Though the result may be a rescue from acute ventricular arrhythmia, studies have shown a relationship with increased mortality and morbidity from progression to heart failure as a consequence of the myocardial stunning [5–11].

Minimizing the need for shock delivery overall will, therefore, ultimately prevent the downstream complications.

3. Shock reducing strategies

There have been several technological advancements to improve ICD therapy delivery and to avoid inappropriate and unnecessary ICD therapies. The shock reduction programming strategies may be divided into the following categories:

- Optimizing ventricular tachyarrhythmia discrimination by applying advanced detection algorithms in all therapy zones.
- Delaying onset of anti-tachycardia therapies either by prolonged detection intervals or setting higher tachycardia detection limits.
- Using anti-tachycardia pacing (ATP) as first-line therapy whenever possible prior to shock delivery.

4. Clinical evidence and the rationale to support ATP therapy

Monomorphic VT can be interrupted when an appropriately timed pacing stimulus is delivered into the excitable gap of a re-entrant circuit where a collision with the orthodromic wave front results in termination of the tachycardia.

An alternative explanation is that the paced stimuli result in myocardial depolarization during the relative refractory period. This pre-excites the preceding wave front, thereby altering myocardial excitability and extinguishing the propagation re-entrant VT [23].

The duration of the excitable gap and the conduction time from the pacing stimulus site to the re-entrant circuit are the main factors influencing penetration of the excitable gap and termination of the arrhythmia [24].

Ventricular conduction time is influenced by anatomic and functional barriers as well as the influence of the sympathetic nervous system [25]. The efficacy of the ATP is improved with adequate beta blockade and is not adversely influenced by other anti-arrhythmic drugs as is the case with defibrillation thresholds [26].

5. Customized programming

Several trials have validated the efficacy of ATP in terminating slower cycle length of VT.

The Pain FREE Rx II trial was the first trial that extended the use of ATP for fast VT (FVT) with heart rates of 188–250 beats/min. This study also used longer intervals to detect VT as

compared to the previous conventional programming (**Figure 1**) [13, 27]. The result was that 73% of FVT episodes were successfully terminated by the ATP.

It also showed that FVT made up 76% of all ventricular arrhythmias that would have conventionally been treated by shock alone according to conventional ICD programming.

Acceleration of FVT occurred in patients programmed to receive ATP as well as those in the shock only group: 4/273 monomorphic VT episodes (2%) in the ATP arm versus 2/145 (1%) in the shock arm. There were also three episodes of syncope during treatment for FVT (ATP, $n = 2$; shock, $n = 1$). This study, therefore, established the safety and efficacy of ATP for both slow and FVT as first-line therapy in ICDs with a non-significant occurrence or difference in arrhythmia-related syncope in either therapy arm.

The next stage in the evolution of device therapy programming was to defer treatment (VT detection) till absolutely necessary. In this regard, the Primary Prevention Parameters Evaluation (PREPARE) trial (**Figure 2**) [28] evaluated a prolonged detection interval duration of 30/40 ventricular beats and an increased tachycardia detection interval (TDI) of 182 beats/min. Supraventricular detection discrimination algorithms and ATP were also optimized in this programming strategy. Arrhythmic syncope was only 1.6% in the test programming strategy. All-cause mortality in the PREPARE study group was also relatively low (Kaplan-Meier estimated 12-month mortality of 4.9%). Thus, the overall safety behind the rationale of this programming was acceptable.

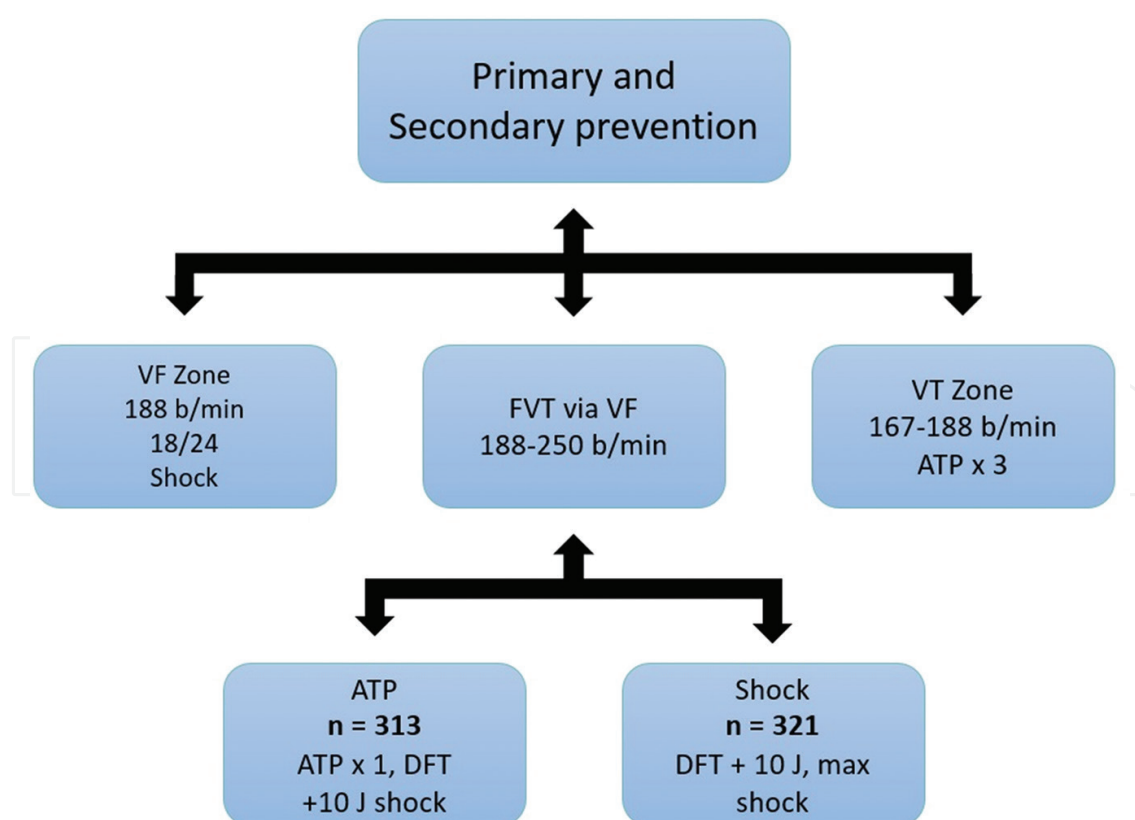


Figure 1. Programming strategy of experimental and control arm in Pain FREE Rx II trial.

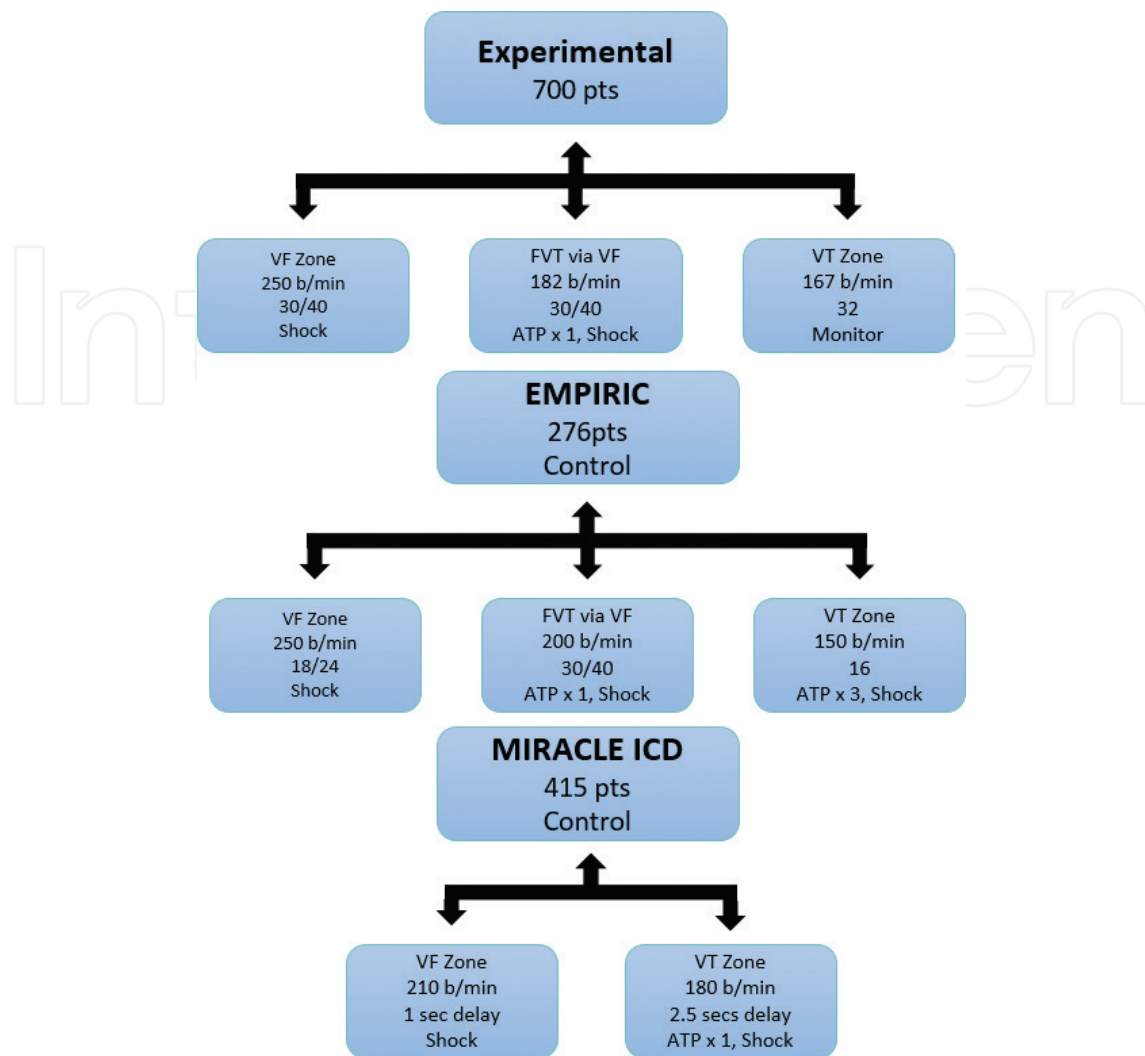


Figure 2. Programming strategy of experimental arm in PREPARE trial with controls from EMPIRIC and MIRACLE ICD patients.

The multicenter automatic defibrillator implantation trial–reduce inappropriate therapy (MADIT-RIT) trial compared three arms: (a) conventional ICD detection of VT with a (b) prolonged detection interval and (c) a higher tachycardia detection interval (**Figure 3**) [29].

The results validated the safety of both the active test arms. There was a 79% reduction in the incidence of therapy in the high-rate group than in the conventional therapy group and delayed therapy (longer detection interval programming) was associated with a 76% reduction in overall therapy delivery. Mortality was reduced by 55% in the high-rate group ($p = 0.01$) and by 44% in the delayed-therapy group ($p = 0.06$). Despite withholding therapies till absolutely needed, there was a mortality improvement in these active programming options.

The incidence of syncope was similar between the groups and was not clinically significant: high rate strategy and delayed therapy versus conventional arm ($p = 0.39$, $p = 0.80$).

Both delayed therapy and higher rate programming were shown to be safe and efficacious.

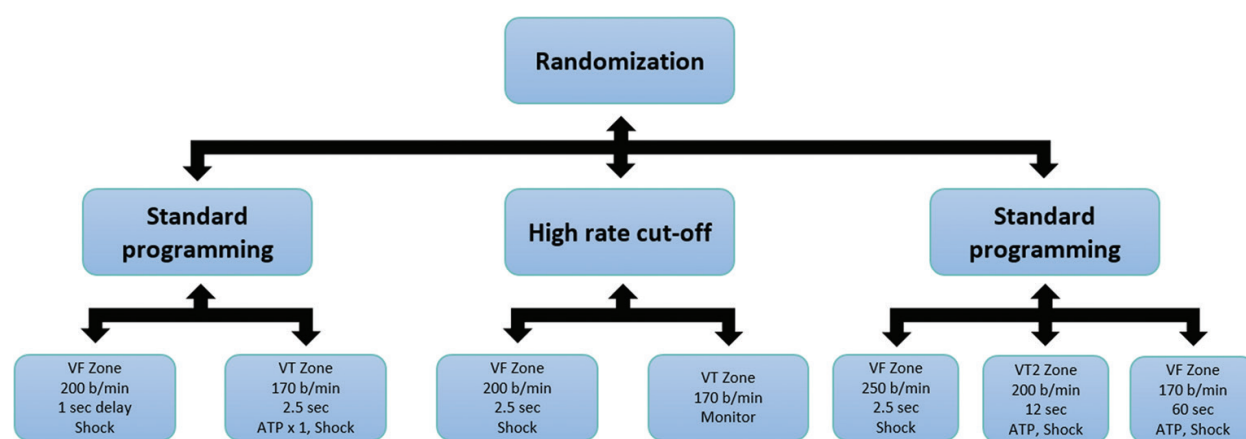


Figure 3. Programming strategy of MADIT-RIT patients.

The ADVANCE III (Avoid Delivering Therapies for Nonsustained Arrhythmias in ICD Patients III) [30] reinforced the findings from the MADIT-RIT trial and included both primary and secondary prevention patients, with or without atrial fibrillation, in whom single-, dual- and triple-chamber ICD were implanted [30]. Thus, this randomized control trial applied an extended detection interval strategy to a heterogeneous cohort of ICD recipients and more likely to resemble a real world setting (**Figure 4**).

This delayed arrhythmia detection strategy resulted in a reduction in the combined end point of all ICD therapies (ATPs and shocks) with 346 delivered therapies (42 therapies per 100 person-years) in test group (extended-detection interval) versus 557 in the control group (standard-detection interval) (67 therapies per 100 person-years); $p < 0.001$.

The incidence of arrhythmic syncope was low in both groups and did not differ significantly with rates of 3.1 versus 1.9 per 100 patient-years ($p = 0.220$ in the extended detection and standard detection groups, respectively). The syncopal episodes were not associated with any additional adverse outcomes. The mortality rates were 5.5 versus 6.3 per 100 patient-years ($p = 0.50$) in extended detection and standard detection, respectively. Both were low and comparable to what was reported in the MADIT-RIT trial.

Similarly the PROVIDE (Programming Implantable Cardioverter Defibrillators in Patients with Primary Prevention Indication to Prolong Time to First Shock) trial was a programming strategy with combination of higher detection rates, prolonged detection intervals, optimized SVT discriminators and empiric ATP therapy compared to conventional parameters in patients receiving ICDs for primary prevention (**Figure 5**) [31]. The primary end point was time to first shock delivery. The median time to first shock was significantly longer at 13.1 months in experimental group versus 7.8 months in the control group. In addition, the 2-year shock rate was 12.4% in the experimental group compared to 19.4% in the control group. An overall reduction in both appropriate and inappropriate shock and ATP was observed. The decrease in ICD therapies was associated with a 30% relative reduction in all-cause mortality.

The incidence of arrhythmic syncope was not significantly different between the two groups with overall incidence of 1.7% over 2 years of follow-up.

With this perspective, we can summarize the overall current trends in ICD programming:

1. Higher zone thresholds
2. Prolonged detection duration or detection intervals
3. Use of advanced discriminators in all zones
4. Use of tiered therapies with ATP as first line therapy before shock delivery.

The aim of these strategies is to reduce inappropriate therapies (ITS) particularly progression to shock and not to over treat ventricular arrhythmias but to allow for spontaneous termination at ventricular rates that are safe to do so.

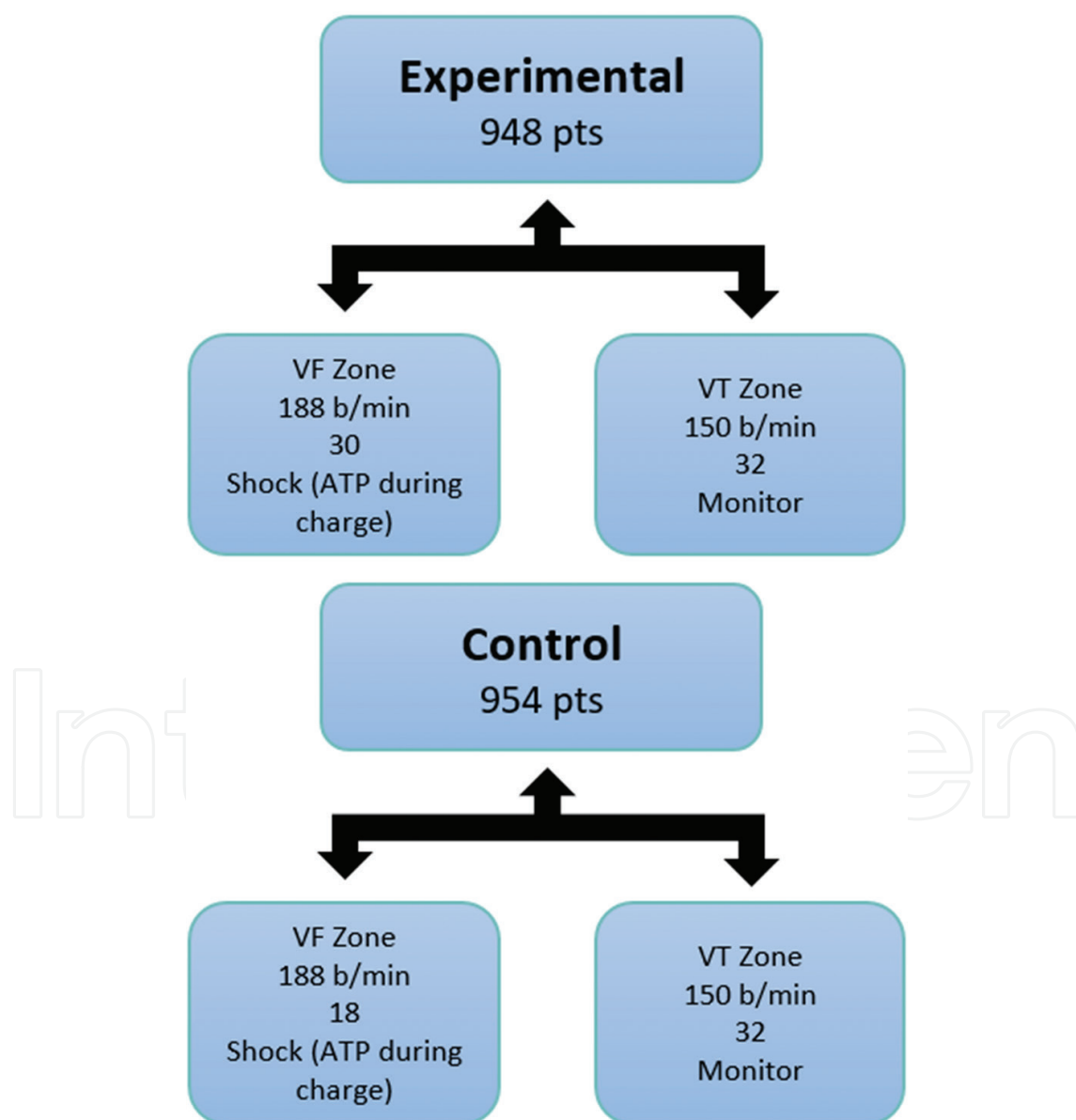


Figure 4. Programming strategy in ADVANCE III trial.

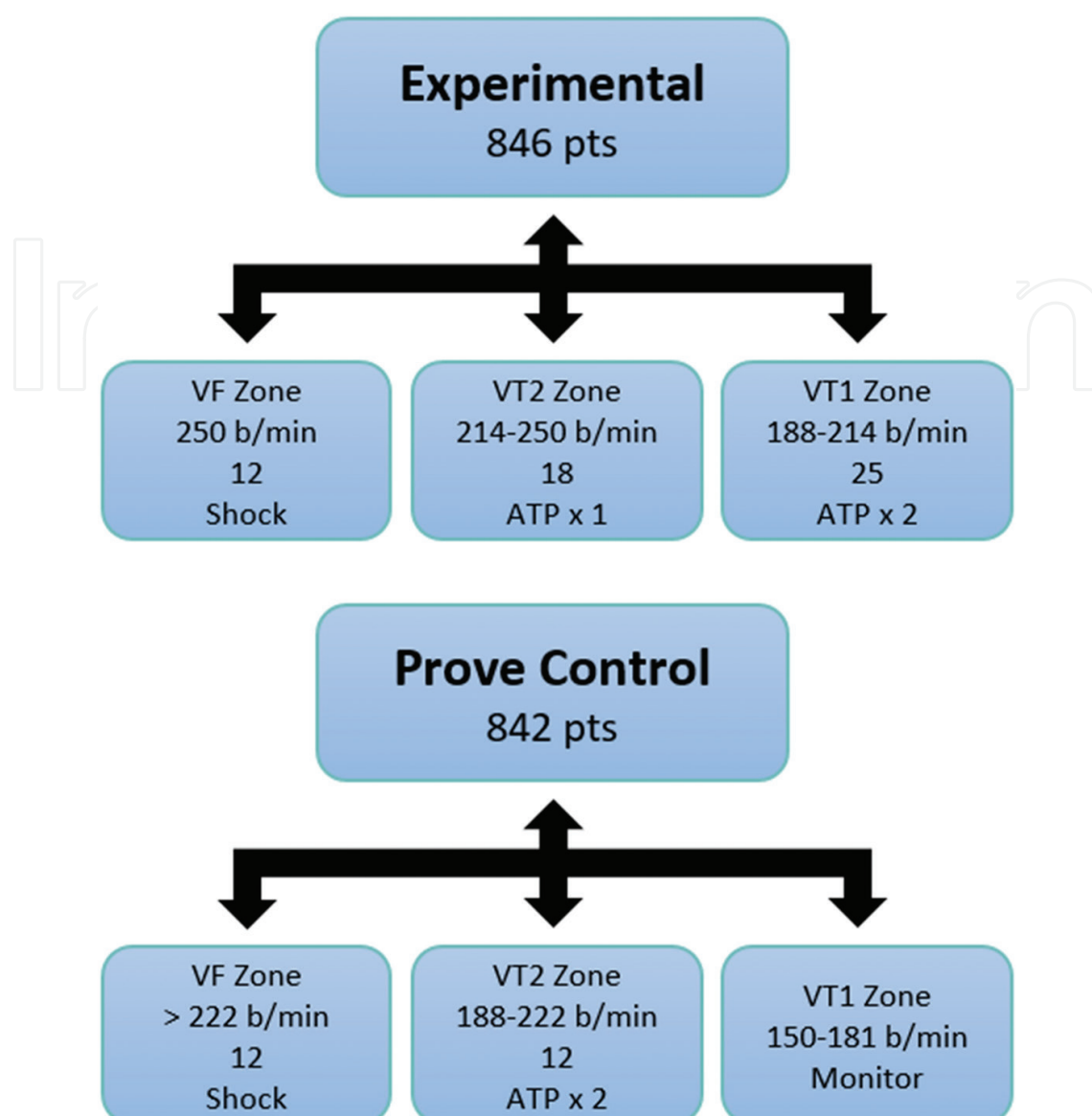


Figure 5. Programming strategy of experimental arm in PROVIDE trial with control population from the PROVE trial.

There has been no data to suggest that implanting dual chamber ICDs is more advantageous in ventricular arrhythmia over single chamber devices in any of the studies we have mentioned. However, there are some practical reasons where one may choose to implant an atrial lead as well to enhance discrimination algorithms [32].

- In patients who require pacing for bradycardia, AV sequential pacing would be preferable.
- In patients with bradycardia-induced or pause-dependent ventricular tachyarrhythmia (such as patients with long QT syndrome and torsades de pointes).
- In patients with documented history of paroxysmal atrial arrhythmias (atrial EGMS will help distinguish the chamber of onset of the tachycardia).

- In patients with hypertrophic cardiomyopathy as they are prone to atrial arrhythmias but may also require pacing.

With this in mind, we employ the following customized ICD programming (**Tables 2 and 3**) for both primary and secondary prevention of SCD in our patients.

	Medtronic	St. Jude medical	Biotronik	Boston scientific	Sorin
VF (VF + FVT) rate in bpm	230–240	250	250	220	200–240
NID	30	30	12/16	2.5 s initial duration	6 cycles
Therapy	ATP during charge	ATP during charge	ATP: Burst 1	ATP: Burst 1	ATP: Burst 1
	Shock × 6	Shock × 6	Shock × 6	Shock × 6	Shock × 6
VT2 (FVT via VF) CL	250	200	200	200	200
NID	30	30	28 (RD-14)	10 s initial duration	6 cycles
Therapy	ATP: Burst 1	ATP: Burst 1	ATP: Burst 1	ATP: Burst 1	ATP: Burst 1
	Shock × 5	Shocks × 4	Shocks × 5	Shocks × 5	Shocks × 5
VT1 CL in bpm	171	171	171	170	170
NID	28	30	30 (RD-16)	30 s initial duration	12 cycles
Therapy	ATP: Burst 1 + Burst 2	ATP: Burst 1 + Burst 2	ATP: Burst 1 + Burst 2	ATP: Burst 1 + Burst 2	ATP: Burst 1 + Burst 2
	Shocks × 4	Shocks × 3	Shocks × 4	Shocks × 4	Shocks × 4
Monitor CL in bpm	150				150
NID	32				12 cycles
Therapy	None				None
ATP programming	Medtronic	St. Jude	Biotronik	Boston	Sorin
Burst 1	8/88/3	8/88/3	8/85/3	8/88/3	8/85/3
Burst 2	8/84/3	8/84/3	8/85/3	8/84/3	8/85/3
Ramp	8/91	8/91	8/90	8/91	8/90

Time out: OFF (Boston Scientific); ATP smart mode: OFF (Medtronic); progressive therapy: ON (Medtronic >2 active zone); ATP optimization: ON (Biotronik); upper rate ATP cut off 260 beats/min (St. Jude); readaptive: ON (St. Jude); Ramp OFF unless specified.

NID: Numbers of interval to detect.

Table 2. Suggested customized ICD programming strategy proposed by the authors for primary prevention.

	Medtronic	St. Jude medical	Biotronik	Boston Scientific	Sorin
VF (VF + FVT) rate in bpm	200	250	250	220	200–240
NID	30	30	12/16	2.5 s initial duration	6 cycles
Therapy	ATP during charge Shock × 6	ATP during charge Shock × 6	ATP: 1 Burst Shock × 6	ATP: Burst 1 Shock × 6	Shock × 6
VT2 (FVT via VF) rate in bpm	250	200	200	200	200
NID	30	30	28 (RD-14)	10 s initial duration	6 cycles
Therapy	ATP: Burst 1 Shock × 5	ATP: Burst 1 Shocks × 4	ATP: 1 Burst Shocks × 5	ATP: Burst 1 Shocks × 5	ATP: Burst 1 Shocks × 5
VT1 rate in bpm	171/VT-20	171/VT-20	171/VT-20	170/VT-20	170/VT-20
NID	28	30	30 (RD-16)	30 s initial duration	12 cycles
Therapy	ATP: Burst 1 + Burst 2 Shocks ×4	ATP: Burst 1 + Burst 2 Shocks ×3	ATP: Burst 1 + Burst 2 Shocks ×4	ATP: Burst 1 + Burst 2 Shocks ×4	ATP: Burst 1 + Burst 2 Shocks ×4
Monitor rate in bpm	150/VT-30				150/VT-30
NID	32				12 cycles
Therapy	None				None
ATP programming					
	Medtronic	St. Jude	Biotronik	Boston	Sorin
Burst 1	8/88/3	8/88/3	8/85/3	8/88/3	8/85/3
Burst 2	8/84/3	8/84/3	8/85/3	8/84/3	8/85/3
Ramp	8/91	8/91	8/90	8/91	8/90
Time out: OFF (Boston Scientific); ATP smart mode: OFF (Medtronic); progressive therapy: ON (Medtronic >2 active zone); ATP optimization: ON (Biotronik); upper rate ATP cut off 260 beats/min (St. Jude); readaptive: ON (St. Jude); Ramp OFF unless specified.					

Table 3. Suggested customized ICD programming strategy proposed by the authors for secondary prevention.

The programming is instituted at the time of device implantation and refined (if needed) at follow-up in the device clinic.

The programming covers all manufacturers' ICDs that we commonly implant. The custom sets are preloaded onto our programmers, thus minimizing the need for tedious reprogramming and only need to be refined if the case warrants this.

In doing so, the work flow both in the implant suite and at the follow-up device visit is facilitated and the programming can be delegated to our allied professional nurses and cardiac technicians.

6. Conclusion

With the complexity and sophistication of ICD algorithms, the programming of these cardiac devices has become a discipline and challenge in its own right. We have found that it has been difficult to maintain predictability and consistency in the programming of ICDs in our centre, hence a need arose to develop a programming template. This was derived from current trends in programming and is mentioned here in an extensive review of the literature. There is an obvious limitation in which each of the studies has been manufacturer specific. We have tried to identify the principles on which the programming was based and then developed a generic template. This was however still done with due consultation with the manufacturers to ensure applicability and safety with the specific algorithms. There have also been very few studies that deal with programming of secondary prevention of ICDs. We have strived to maintain a compromise between all manufacturers to reach a consensus on programming for primary prevention of ICDs.

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References

- [1] Moss AJ, Zareba W, Hall WJ, et al. Multicenter automatic defibrillator implantation trial II investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. The New England Journal of Medicine. 2002;**346**:877-883
- [2] Bardy GH, Lee KL, Mark DB, et al. Amiodarone or an implantable cardioverter-defibrillator for congestive heart failure. The New England Journal of Medicine. 2005;**352**:225-237
- [3] Kadish A, Dyer A, Daubert JP, et al. Prophylactic defibrillator implantation in patients with non-ischemic dilated cardiomyopathy. New England Journal of Medicine. 2004;**350**:2151-2158

- [4] A comparison of antiarrhythmic-drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. The New England Journal of Medicine. 1997;**337**:1576-1583
- [5] Sood N, Ruwald AC, Solomon S, et al. Association between myocardial substrate, implantable cardioverter defibrillator shocks and mortality in MADIT-CRT. European Heart Journal. 2014;**35**:106-115
- [6] Powell BD, Saxon LA, Boehmer JP, et al. Survival after shock therapy in implantable cardioverter-defibrillator and cardiac resynchronization therapy-defibrillator recipients according to rhythm shocked. The ALTITUDE survival by rhythm study. Journal of the American College of Cardiology. 2013;**62**:1647-1649
- [7] Sweeney MO, Sherfese L, DeGroot PJ, Wathen MS, Wilkoff BL. Differences in electrical therapy type for ventricular arrhythmias on mortality in implantable cardioverter-defibrillator patients. Heart Rhythm. 2010;**7**:353-360
- [8] Poole Je, Johnson GW, Hellkamp AS, et al. Prognostic importance of defibrillator shocks in patients with heart failure. The New England Journal of Medicine. 2008;**4**:1009-1017
- [9] Sears SF, Todaro JF, Lewis TS, Sotile W, Conti JB. Examining the psychosocial impact of implantable cardioverter defibrillators: A literature review. Clinical Cardiology. 1999;**22**:481-489
- [10] Sears SF, Matchett M, Conti JB. Effective management of ICD patient psychosocial issues and patient critical events. Journal of Cardiovascular Electrophysiology. 2009;**20**:1297-1304
- [11] Magyar-Russell G, Thombs BD, Cai J X, Baveja T, Kuhl EA, Singh PP, Montenegro Braga Barroso M, Arthurs E, Roseman M, Amin N, Marine JE, Ziegelstein RC. The prevalence of anxiety and depression in adults with implantable cardioverter defibrillators systematic review. Journal of Psychosomatic Research. 2011;**71**:223-231
- [12] Klein RC, Raitt MH, Wilkoff BL, Beckman KJ, Coromilas J, Wyse DG, et al. Analysis of implantable cardioverter defibrillator therapy in the antiarrhythmics versus implantable defibrillators (AVID) trial. Journal of Cardiovascular Electrophysiology. 2003;**14**:940-948
- [13] Wathen MS, DeGroot PJ, Sweeney MO, Stark AJ, Otterness MF, Adkisson WO, et al. Pain FREE Rx II Investigators. Prospective randomized multicenter trial of empirical anti tachycardia pacing versus shocks for spontaneous rapid ventricular tachycardia in patients with implantable cardioverter-defibrillators: Pacing fast ventricular tachycardia reduces shock therapies (Pain FREE Rx II) trial results. Circulation. 2004;**110**(17):2591-2596
- [14] Sweeney MO, Wathen MS, Volosin K, Abdalla I, DeGroot PJ, Otterness MF, Stark AJ. Appropriate and inappropriate ventricular therapies, quality of life, and mortality among primary and secondary prevention implantable cardioverter defibrillator patients: Results from the pacing fast VT reduces shock therapies (Pain FREE Rx II) trial. Circulation. 2005;**111**(22):2898-2905

- [15] Young JB, Abraham WT, Smith AL, Leon AR, Lieberman R, Wilkoff B, et al. Combined cardiac resynchronization and implantable cardioversion defibrillation in advanced chronic heart failure: The miracle ICD trial. *JAMA*. 2003;**289**:2685-2694
- [16] Daubert JP, Zareba W, Cannom DS, McNitt S, Rosero SZ, Wang P, et al. Inappropriate implantable cardioverter-defibrillator shocks in MADIT II: Frequency, mechanisms, predictors, and survival impact. *Journal of the American College of Cardiology*. 2008;**51**:1357-1365
- [17] Poole JE, Johnson GW, Hellkamp AS, Anderson J, Callans DJ, Raitt MH, et al. Prognostic importance of defibrillator shocks in patients with heart failure. *The New England Journal of Medicine*. 2008;**359**:1009-1017
- [18] Saxon LA, Hayes DL, Gilliam FR, Heidenreich PA, Day J, Seth M, et al. Long-term outcome after ICD and CRT implantation and influence of remote device follow-up: The altitude survival study. *Circulation*. 2010;**122**:2359-2367
- [19] van Rees JB, Borleffs CJ, de Bie MK, Stijnen T, van Erven L, Bax JJ, et al. Inappropriate implantable cardioverter-defibrillator shocks: Incidence, predictors, and impact on mortality. *Journal of the American College of Cardiology*. 2011;**57**:556-562
- [20] Pinski SL, Fahy GJ. The proarrhythmic potential of implantable cardioverter-defibrillators. *Circulation* 1995;**92**:1651-1664
- [21] Yabe S, Smith WM, Daubert JP, Wolf PD, Rollins DL, Ideker RE. Conduction disturbances caused by high current density electric fields. *Circulation Research*. 1990;**66**(5):1190-1203
- [22] Hurst TM, Hinrichs M, Breidenbach C, Katz N, Waldecker B. Detection of myocardial injury during transvenous implantation of automatic cardioverter-defibrillators. *Journal of the American College of Cardiology*. 1999;**34**(2):402-408
- [23] Sweeney MO. Antitachycardia pacing for ventricular tachycardia using implantable cardioverter defibrillators. *PACE*. 2004;**27**:1292-1305. 18
- [24] Josephson ME, Almendral JM, Buxton AE, Marchlinski FE. Mechanisms of ventricular tachycardia. *Circulation*. 1987;**75**:41-47
- [25] Fisher JD. Ventricular tachycardia: Practical and provocative electrophysiology. *Circulation*. 1978;**58**:1000-1001
- [26] Jimenez-Candil J, Hernandez J, Martin A, Ruiz-Olgado M, Herrero J, Ledesma C, Morinigo J, Martin-Luengo C. Influence of beta-blocker therapy on anti-tachycardia pacing effectiveness for Monomorphic ventricular tachycardia occurring in implantable cardioverter-defibrillator patients: A dose dependent effect. *Europace*. 2010;**12**:1231-1238
- [27] Wathen MS, Sweeney MO, DeGroot PJ, et al. Shock reduction using anti tachycardia pacing for spontaneous rapid ventricular tachycardia in patients with coronary artery disease. *Circulation*. 2001;**104**:796-801
- [28] Wilkoff BL, Williamson BD, Stern RS, Moore SL, Lu F, Lee SW, et al. PREPARE study investigators. Strategic programming of detection and therapy parameters in implantable

cardioverter-defibrillators reduces shocks in primary prevention patients: Results from the PREPARE (Primary Prevention Parameters Evaluation) study. *Journal of the American College of Cardiology*. 2008;**52**(7):541-550

- [29] Moss AJ, Schuger C, Beck CA, Brown MW, Cannom DS, Daubert JP, et al. MADIT-RIT trial investigators. Reduction in inappropriate therapy and mortality through ICD programming. *The New England Journal of Medicine*. 2012;**367**(24):2275-2283
- [30] Gasparini M, Proclemer A, Klersy C, Kloppe A, Lunati M, Ferrer JB, et al. Effect of long-detection interval vs standard-detection interval for implantable cardioverter-defibrillators on anti-tachycardia pacing and shock delivery: The ADVANCE III randomized clinical trial. *JAMA*. 2013;**309**(18):1903-1911
- [31] Saeed M, Hanna I, Robotis D, Styperek R, Polosajian L, Khan A, et al. Programming implantable cardioverter-defibrillators in patients with primary prevention indication to prolong time to first shock: Results from the PROVIDE study. *Journal of Cardiovascular Electrophysiology*. 2014;**25**(1):52-59
- [32] Kusumoto FM, Calkins H, Boehmer J, et al. Heart rhythm society; American college of cardiology; American Heart Association. HRS/ACC/AHA expert consensus statement on the use of implantable cardioverter-defibrillator therapy in patients who are not included or not well represented in clinical trials. *Journal of the American College of Cardiology*. 2014;**64**(11):1143-1177