Research Square

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Programmed Ventricular Stimulation Induction Threshold Does Not Predict Arrhythmic Burden in Ischemic and Non-Ischemic Cardiomyopathy: A Single- Center Prospective Registry

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Abstract

Background

Programmed ventricular stimulation (PVS) guides implantable cardioverter-defibrillator (ICD) decisions in gray-zone cardiomyopathy patients. Whether the ease of monomorphic ventricular tachycardia (mVT) induction predicts arrhythmic burden is unknown.

Objective

To evaluate the relationship between PVS induction threshold and arrhythmic events recorded by implanted devices over 12 months.

Methods

We enrolled 66 consecutive heart failure patients (mean age 62.7 ± 9.0 years, 89.4% male) referred for PVS. Patients had ischemic (ICM, n = 36) or non-ischemic cardiomyopathy (NICM, n = 30). Among 54 patients (81.8%) with inducible mVT, the threshold was categorized as S2 (single extrastimulus, n = 4), S3 (double, n = 16), or S4+ (triple/burst, n = 34). All underwent 12-month device interrogation.

Results

Events occurred in 9/54 patients (16.7%): 0/4 (0%) in S2, 1/16 (6.2%) in S3, and 8/34 (23.5%) in S4+. Neither the chi-square test (χ^2=3.20, p = 0.20) nor the Cochran–Armitage trend test (r = 0.237, p = 0.084) reached significance. Events comprised 4 sustained VT requiring shock and 5 NSVT/ATP episodes. NICM patients had higher event rates (25.0% vs. 11.8%). Age did not differ between patients with and without events.

Conclusion

PVS inducibility identified meaningful arrhythmic risk (16.7% event rate). The induction threshold did not predict outcomes; patients requiring aggressive protocols had numerically higher event rates. PVS positivity itself, rather than induction ease, appears to be the primary prognostic marker.

INTRODUCTION

Sudden cardiac death (SCD) remains one of the leading causes of cardiovascular mortality, and patients with structural heart disease and reduced left ventricular function bear the highest burden. Over the past three decades, the implantable cardioverter-defibrillator has proven its value in primary prevention, most

convincingly in the MADIT, MADIT-II, and SCD-HeFT trials, which cemented an LVEF of 35% or below as the threshold for device implantation[1–4].

In practice, many patients do not fit comfortably into this framework. Patients with mildly reduced ejection fraction in the 36–50% range, patients with unexplained syncope that appears arrhythmic but remains undocumented, and patients with non-ischemic substrates where LVEF predicts SCD less reliably, often end up in a clinical gray zone. For these patients, programmed ventricular stimulation in the electrophysiology laboratory offers a way to directly probe the myocardial substrate and assess whether a re-entrant circuit can be sustained under provocation [5–8].

The evidence base for PVS-guided management is anchored in several important studies. The MUSTT trial showed that electrophysiology-guided therapy in coronary disease patients with reduced LVEF and non-sustained VT significantly cut arrhythmic death [3]. The PRESERVE EF study expanded this concept by demonstrating that even in post-infarction patients with LVEF above 40%, a two-step screening strategy combining non-invasive risk markers with PVS identified an inducible subgroup whose major arrhythmic event rate reached 22% over 32 months of follow-up, a finding that led to early termination of the trial [9]. More recently, Soris et al reported similar results in 168 patients with myocardial scarring and preserved or mildly reduced LVEF, with 43% of inducible patients experiencing major arrhythmic events over nearly 4 years [10].

The need for better risk stratification tools is especially pressing in non-ischemic cardiomyopathy. The DANISH trial demonstrated that ICD implantation did not reduce all-cause mortality in NICM patients selected solely on the basis of LVEF ≤ 35%, although it did reduce SCD [11]. Post-hoc analyses identified younger patients and those with higher SCD risk as subgroups most likely to benefit, but in practice we lack a reliable substrate-level test for identifying which NICM patients truly need a device. PVS may fill this gap, though the 2022 ESC Guidelines remain more cautious about its use in NICM than in ICM [5, 6].

What remains less clear is whether the aggressiveness of the stimulation protocol carries independent prognostic information. Standard protocols use a hierarchical approach, with single (S2), double (S3), and triple (S4) extrastimuli. Clinicians often have an intuitive sense that patients in whom VT fires off with a single extra beat must harbor a more dangerous substrate than those who require maximal provocation [12, 13]. In Brugada syndrome, this intuition has been validated: pooled data from over 1,300 patients show that induction with fewer extrastimuli predicts worse outcomes [14]. Whether the same principle applies to structural cardiomyopathy, where VT is driven by fixed anatomical re-entry rather than functional conduction abnormalities, has not been tested.

We asked a simple question: in gray-zone cardiomyopathy patients who test positive on PVS and receive an ICD, does the ease of VT induction predict the burden of arrhythmic events their device will record over the following year?

METHODS

Study design and population

This was a prospective, single-center, observational registry conducted at a tertiary electrophysiology center in Sismanogleio General Hospital, Athens, Greece. All heart failure patients were enrolled and referred for PVS between January 2024 and May 2025. The study population included patients with both ICM and NICM whose clinical presentation placed them in a gray zone for primary prevention ICD. This meant that the treating electrophysiologist judged the standard LVEF-based criteria insufficient, on their own, to determine whether a device was warranted.

Inclusion criteria were an established diagnosis of cardiomyopathy with a clinical indication for PVS as determined by the referring electrophysiologist; one or more gray-zone features, including LVEF between 36–50%, unexplained syncope suspicious for arrhythmic etiology, or documented non-sustained ventricular tachycardia; and the ability to provide written informed consent.

Exclusion criteria were: patients with a pre-existing ICD, those referred explicitly for secondary prevention after survived VT/VF arrest, and patients who declined device implantation despite a positive test. The study conformed to the Declaration of Helsinki and received institutional ethics approval.

Programmed ventricular stimulation protocol

PVS was carried out in fasting state. Antiarrhythmic drugs were discontinued for at least five half-lives before the procedure, with the exception of amiodarone, which was withheld for at least seven days when clinically feasible. Stimulation was delivered from the right ventricular apex at twice the diastolic threshold (typically 2.0 mA) with a pulse width of 1.0–2.0 ms. An eight-beat drive train at fixed cycle lengths of 600, 500, and 400 ms was utilized.

The stimulation protocol was hierarchical. In the first step (S2), a single extrastimulus was introduced after the drive train and its coupling interval reduced in 10 ms decrements until ventricular refractoriness was reached. In the second step (S3), S2 was fixed at 10–20 ms above its effective refractory period while a second extrastimulus was decremented in the same fashion. In the third step (S4), S2 and S3 were both fixed and a third extrastimulus introduced. Burst pacing was employed at operator discretion if triple extrastimuli failed to induce tachycardia. A minimum coupling interval of 200 ms was maintained throughout, in accordance with current ESC guidelines [5, 12].

The test was considered positive if sustained monomorphic VT (defined as lasting 30 seconds or longer, or requiring termination because of hemodynamic compromise) was induced. Polymorphic VT or ventricular fibrillation provoked by triple extrastimuli was treated as a non-specific finding and was not, on its own, used to justify ICD implantation [5, 6, 13].

Induction threshold classification

Patients with a positive test were classified according to the least aggressive protocol that achieved mVT induction. Low threshold (S2) denoted induction with a single extrastimulus. Moderate threshold (S3) required two extrastimuli. High threshold (S4+) required three extrastimuli or burst pacing.

Device implantation and follow-up

All patients with positive PVS underwent implantation of an ICD or a cardiac resynchronization therapy–defibrillator system (CRT-D). The specific device was selected according to standard clinical criteria, taking into account QRS duration, LVEF, and NYHA functional class. Device interrogation was performed at 12 months post-implantation by our dedicated outpatient arrhythmia clinic. The primary endpoint was any ventricular arrhythmic episode detected by the implanted device, including sustained VT requiring device therapy (anti-tachycardia pacing or shock) and non-sustained VT.

Statistical analysis

Continuous variables are reported as mean ± standard deviation and were compared using one-way ANOVA or the independent-samples t-test, as appropriate. Categorical variables are expressed as counts and percentages and were compared using the chi-square test or Fisher's exact test. The Cochran–Armitage test was used to evaluate for a linear trend in event rates across ordered threshold categories. A two-sided p-value below 0.05 was considered significant. All analyses were performed using IBM SPSS Statistics (version 29.0, IBM Corp., Armonk, NY, USA).

RESULTS

Baseline characteristics

A total of 66 patients were enrolled. The mean age was 62.7 ± 9.0 years (range 44–78), and 59 patients (89.4%) were male. Thirty-six patients (54.5%) had ICM (Group A) and 30 (45.5%) had NICM (Group B), including dilated and hypertrophic phenotypes. The mean LVEF was 36.8 ± 9.9% overall, and was somewhat lower in the ICM group (34.1 ± 8.3%) than in the NICM group (40.0 ± 10.8%). NYHA functional class distribution reflected a predominantly ambulatory population: Class I in 23 patients (34.8%), Class II in 31 (47.0%), and Class III in 10 (15.2%), with one patient asymptomatic at enrollment. Mean age was similar between groups (ICM 63.4 ± 9.5 vs. NICM 61.9 ± 8.6 years).

Syncope suspicious for an arrhythmic mechanism was reported by 54 patients (81.8%), and 19 (28.8%) had a documented history of sustained VT or VF prior to the index evaluation. The mean QRS duration was 129.4 ± 31.2 ms, and left bundle branch block was present in 11 patients (16.7%). Rates of guideline-directed medical therapy were high overall: beta-blockers in 57 of 60 evaluable patients (95.0%), ACE inhibitor or ARB or ARNI in 47 of 60 (78.3%), SGLT2 inhibitor in 47 of 60 (78.3%), and mineralocorticoid receptor antagonist in 34 of 60 (56.7%). Complete baseline characteristics, stratified by etiology, are presented in Table 1.

Programmed ventricular stimulation

PVS induced sustained monomorphic VT in 54 patients (81.8%). The inducibility rate was significantly higher in ICM (34 of 36, 94.4%) than in NICM (20 of 30, 66.7%). Among the 12 patients with negative PVS, 10 had NICM and only 2 had ICM, a distribution consistent with the known lower sensitivity of PVS in non-ischemic substrates.

Among the 54 positive patients, the distribution of induction thresholds was: S2 (single extrastimulus) in 4 patients (7.4%), S3 (double) in 16 (29.6%), and S4+ (triple or burst) in 34 (63.0%). Within the ICM group, 4 patients (11.8%) were induced at S2, 12 (35.3%) at S3, and 18 (52.9%) at S4+. Notably, no NICM patient was induced at the S2 level; 4 (20.0%) required S3 and a full 16 (80.0%) required S4 + stimulation. The mean age of patients in the S2 group was 56.2 ± 11.3 years, compared with 62.1 ± 7.2 in S3 and 64.4 ± 9.7 in S4+.

Hemodynamic tolerance of induced VT varied: 6 patients (11.1%) remained stable, 26 (48.1%) developed hypotension, and 22 (40.7%) required cardioversion. The distribution of induced rhythms across all 66 patients was monomorphic VT in 42 (63.6%), polymorphic VT/VF in 1 (1.5%), non-sustained VT in 12 (18.2%), and no inducible arrhythmia in 11 (16.7%). The study flowchart is presented in Fig. 1.

Device implantation

Among positive-PVS patients, 45 received a single-chamber ICD and 7 a CRT-D system. The remaining 2 patients had device implantation deferred or scheduled at an outside institution. The primary clinical indication was primary prevention in 35 patients and secondary prevention in 3.

Twelve-month arrhythmic outcomes

At the 12-month device interrogation, arrhythmic events were detected in 9 of 54 patients with positive PVS, yielding an overall event rate of 16.7%. These events broke down into 4 sustained VT episodes that required ICD shock therapy and 5 episodes of non-sustained VT or VT terminated by anti-tachycardia pacing. The event rates stratified by induction threshold were strikingly graded: 0% (0 of 4) in the S2 group, 6.2% (1 of 16) in the S3 group, and 23.5% (8 of 34) in the S4 + group (Table 2, Fig. 2). The single event in the S3 group was an episode terminated by ATP. The S4 + group accounted for all 4 sustained VT-shock events and 4 of the 5 NSVT/ATP episodes.

When stratified by etiology, the ICM subgroup had an event rate of 11.8% (4 of 34), with all 4 events occurring exclusively in patients with the highest induction threshold: 0 of 4 in S2, 0 of 12 in S3, and 4 of 18 (22.2%) in S4+. The NICM subgroup had a higher overall event rate of 25.0% (5 of 20), distributed as 1 of 4 (25.0%) in S3 and 4 of 16 (25.0%) in S4+ (Table 3, Fig. 3). The mean age of patients who experienced arrhythmic events was 63.2 ± 12.1 years, virtually identical to the 63.1 ± 8.7 years seen in those without events. The mean LVEF among event patients was 32.2 ± 8.3%, compared with 35.4 ± 9.4% in patients without events (Fig. 4).

Statistical analysis

The chi-square test showed no significant association between threshold category and 12-month events ($\chi^2 = 3.20$, df = 2, p = 0.20). The Cochran–Armitage test demonstrated a positive trend that approached but did not reach statistical significance (Pearson r = 0.237, p = 0.084). Given the small cell sizes (particularly the S2 group with only 4 patients) the study was underpowered to detect a moderate effect size with conventional confidence.

DISCUSSION

Principal findings

In this prospective, single center registry of gray-zone cardiomyopathy patients undergoing PVS, three observations emerge. PVS inducibility was high (81.8%), supporting its role as a practical tool for identifying an arrhythmogenic substrate when conventional risk markers are inconclusive. The overall arrhythmic event rate among patients with positive PVS who went on to receive an ICD was 16.7% at 12 months. This rate is comparable to, and in some subgroups approaches, the appropriate therapy rates reported in the landmark primary prevention trials [1–3]. Most unexpectedly, the direction of the threshold-outcome relationship ran opposite to what was hypothesized: event rates were numerically highest in patients who required the most aggressive stimulation to induce VT (S4+: 23.5%), intermediate in those induced with double extrastimuli (S3: 6.2%), and zero in those induced most easily (S2: 0%). Although this did not reach statistical significance (p = 0.084), the pattern deserves attention.

The induction threshold: what does it really measure?

In Brugada syndrome, a channelopathy in which VT and VF arise from functional phase-2 re-entry and conduction abnormalities rather than from fixed anatomical circuits, the number of extrastimuli required for induction has established prognostic value. The pooled analysis by Sroubek et al. of 1,312 patients found that arrhythmia induction with single or double extrastimuli conferred significantly higher risk than induction requiring triple extrastimuli[14]. Makimoto and colleagues reported the same pattern in a smaller Brugada cohort [15]. It is tempting to extrapolate from these data and assume that the same principle should apply to structural heart disease (SHD).

In SHD the mechanism is different. In ICM and NICM, VT is driven by anatomical re-entry around and through regions of scar. As a result, the relationship between stimulation aggressiveness and clinical risk is not straightforward. A patient with a single, well-defined re-entry circuit may be easily inducible in the lab, yet the same circuit may not translate into frequent spontaneous arrhythmias. Conversely, a patient who requires triple extrastimuli may harbor a diffuse, heterogeneous substrate which may be less easily triggered in the laboratory, but may be more prone to spontaneous arrhythmia in real world conditions [7, 15, 16].

Support for this interpretation comes from an entirely different disease population. Khairy and colleagues studied 252 patients with surgically repaired tetralogy of Fallot and found that while PVS inducibility powerfully predicted subsequent VT and sudden death (relative risk 5.0), the specific mode of induction including the number of extrastimuli had no predictive value whatsoever (p = 0.92)[17]. This dissociation between inducibility and threshold in a structural heart disease population closely parallels what we observed in our cardiomyopathy cohort. This concept is also reflected in contemporary guideline interpretations, which emphasize inducibility as a clinically relevant marker while acknowledging the limited prognostic value of specific stimulation protocols[18].

The role of etiology

The interplay between etiology and threshold adds another layer of complexity. All four S2 patients in our cohort had ischemic cardiomyopathy, while the S4 + group was enriched for NICM (16 of 34 patients, 47.1%). The NICM patients had a substantially higher overall event rate (25.0%) than the ICM patients (11.8%), and the different nature of the arrhythmogenic substrate (discrete post-infarction scar versus diffuse interstitial fibrosis) may partly confound the apparent relationship between threshold and events. It is worth noting, however, that within the ICM subgroup alone, the same pattern held: all four ICM events occurred in S4 + patients (4 of 18, 22.2%), with zero events in the S2 (n = 4) and S3 (n = 12) groups.

The 23.5% event rate in our S4 + group is strikingly close to the 22% major arrhythmic event rate reported in the PRESERVE EF study's inducible cohort, despite the fact that PRESERVE EF enrolled exclusively post-infarction patients with preserved ejection fraction[9]. The convergence of these numbers across different LVEF ranges and etiologies suggests that PVS-positive patients, regardless of the specific threshold, constitute a population with a reproducibly high arrhythmic event rate, and that the binary positive result is the clinically actionable signal.

Implications for the NICM-ICD debate

The 25.0% event rate among NICM patients with positive PVS is clinically meaningful. Since the DANISH trial demonstrated no overall mortality benefit from primary prevention ICD in NICM patients selected by LVEF alone [17], there has been an active search for substrate-level markers that could identify the NICM subpopulation most likely to benefit from device therapy [11]. Late gadolinium enhancement on cardiac MRI has emerged as one candidate; our data suggest that PVS may be another. In this context, our findings are clinically relevant. Importantly, among the NICM patients in whom PVS was positive, one in four had a significant arrhythmic event within a single year of follow-up. This is a rate that would justify ICD implantation in clinical practice.

Prior studies have explored the role of PVS in similarly ambiguous populations. In patients with reduced ejection fraction but no documented sustained arrhythmias, PVS has shown to refine risk stratification beyond conventional clinical markers [19, 20]. Earlier work in dilated cardiomyopathy and unexplained syncope also suggested that electrophysiology-guided ICD strategies may identify patients at meaningful risk despite the absence of clear guideline indications [20, 21]. Our findings expand on this

concept, suggesting that once inducibility is established, the degree of stimulation required may be less informative than the presence of inducibility itself.

Soris et al. recently reinforced this perspective, reporting that inducible patients with myocardial scarring and LVEF ≥ 40% had a 4.3% annual incidence of major arrhythmic events, and that VF induced with triple extrastimuli was universally non-specific since none of those patients developed clinical events [10]. Our protocol's exclusion of isolated polymorphic VT/VF as a positive endpoint is consistent with this observation and with the broader literature. The fact that all 9 events in our cohort occurred in patients with induced sustained monomorphic VT reinforces the prognostic specificity of this endpoint.

Age, LVEF, and events

An important finding from a clinical perspective is that age did not discriminate between patients who did and did not experience events (63.2 ± 12.1 vs. 63.1 ± 8.7 years, respectively). This suggests that within a PVS-positive cohort, age is not a useful triage variable, unlike in the DANISH population, where younger patients derived most benefit. The mean LVEF was numerically lower in the event group (32.2% vs. 35.4%), consistent with the known association between lower ejection fraction and arrhythmic risk, though the overlap was substantial. It is also noteworthy that one event patient had an LVEF of 50%, reinforcing the concept that LVEF alone is an imperfect predictor and that substrate-based testing adds genuine clinical value.

Clinical implications

Several practical messages emerge. First, a positive PVS should be considered actionable regardless of how many extrastimuli are required. These findings do not support de-escalation based on induction threshold. If anything, risk appears at least comparable, if not higher. Second, the 16.7% overall event rate validates PVS-guided ICD implantation in this population. Third, PVS may be especially valuable in NICM patients, where conventional LVEF-based criteria have proven insufficient. All in all, the binary result of PVS is more informative than the induction threshold itself. Markman et al. noted that PVS test-retest concordance runs between 70–80% [7], suggesting that the precise S3/S4 + distinction may partly reflect the autonomic milieu at the time of testing rather than a fixed substrate property.

Limitations

Several limitations warrant acknowledgment. The sample of 54 PVS-positive patients with 9 events provides limited power, particularly for the S2 group (n = 4), where the exact binomial 95% confidence interval spans 0–60%. This single-center study may limit generalizability. We did not follow patients with negative PVS, precluding sensitivity/specificity calculations. Grouping triple extrastimuli and burst pacing into S4 + may obscure meaningful differences. Antiarrhythmic drug use and ICD programming during follow-up were not systematically captured. Finally, 12 months may be too short to capture the full arrhythmic trajectory and longer follow-up is needed. The lack of statistical significance should be interpreted in the context of the limited sample size and event rates.

CONCLUSIONS

In this prospective single-center registry of gray-zone cardiomyopathy patients, PVS identified a population at genuine and clinically meaningful arrhythmic risk: nearly one in six patients with a positive test experienced a ventricular arrhythmic event within 12 months of ICD implantation. The induction threshold did not correlate with subsequent outcomes. A non-significant trend toward higher event rates with more aggressive induction protocols was observed, consistent with data from other structural heart disease populations. Our findings support the clinical value of the binary PVS result, positive or negative, as the primary risk stratification signal, and they argue against using the specific induction threshold to modulate clinical decision-making. In this setting, inducibility appears to matter more than the inducibility threshold. Larger, multicenter studies with extended follow-up are needed to confirm these observations and to refine the role of PVS in the evolving landscape of arrhythmic risk assessment.

Abbreviations

ATP
anti-tachycardia pacing
CRT-D
cardiac resynchronization therapy–defibrillator
EPL
electrophysiology laboratory
ICD
implantable cardioverter-defibrillator
ICM
ischemic cardiomyopathy
LBBB
left bundle branch block
LVEF
left ventricular ejection fraction
mVT
monomorphic ventricular tachycardia
NICM
non-ischemic cardiomyopathy
NSVT
non-sustained ventricular tachycardia
NYHA
New York Heart Association
PVS
programmed ventricular stimulation
SCD
sudden cardiac death

SMVT
sustained monomorphic ventricular tachycardia
VF
ventricular fibrillation
VT
ventricular tachycardia

Declarations

Conflicts of interest:

None declared.

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None.

Author Contribution

G.T. and N.T. conceived and designed the study. S.C.K., G.T., and N.T. performed the programmed ventricular stimulation procedures and acquired the clinical and device-interrogation data. F.T., C.K., I.M., D.K., and A.K. contributed to patient enrolment, data collection, and follow-up. S.C.K. performed the statistical analysis and had full access to the data. S.C.K., G.T., and N.T. wrote the main manuscript text. N.T. prepared Figures 1–4 and the tables. All authors critically reviewed the manuscript for important intellectual content and approved the final version. S.C.K. is the guarantor of the work.

Data Availability

All data supporting the findings of this study are available within the paper and its Supplementary Information.

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Tables

Table 1. Baseline Characteristics

Variable	All (N=66)	ICM (n=36)	NICM (n=30)	p
Age, years	62.7±9.0	63.4±9.5	61.9±8.6	0.50
Male sex, n (%)	59 (89.4)	35 (97.2)	24 (80.0)	0.03
LVEF, %	36.8±9.9	34.1±8.3	40.0±10.8	0.01
NYHA I, n (%)	23 (34.8)	9 (25.0)	14 (46.7)	
NYHA II, n (%)	31 (47.0)	18 (50.0)	13 (43.3)	
NYHA III, n (%)	10 (15.2)	8 (22.2)	2 (6.7)	
QRS, ms	129.4±31.2	133.4±31.6	122.3±30.5	0.33
LBBB, n (%)	11 (16.7)	8 (22.2)	3 (10.0)	0.19
Syncope, n (%)	54 (81.8)	31 (86.1)	23 (76.7)	0.33
Prior VT/VF, n (%)	19 (28.8)	9 (25.0)	10 (33.3)	0.45
Beta-blocker*	57/60 (95.0)	33/34 (97.1)	24/26 (92.3)	0.57
ACEi/ARB/ARNI*	47/60 (78.3)	29/34 (85.3)	18/26 (69.2)	0.13
MRA*	34/60 (56.7)	21/34 (61.8)	13/26 (50.0)	0.36
SGLT2i*	47/60 (78.3)	29/34 (85.3)	18/26 (69.2)	0.13
PVS positive, n (%)	54 (81.8)	34 (94.4)	20 (66.7)	0.003

*Values are mean ± SD or n (%). *Medication data available for 60 patients. p-values by t-test (continuous) or Fisher's exact test (categorical).*

Table 2. Arrhythmic Event Rates by Induction Threshold at 12-Month Follow-Up

Threshold	N	Age, y	Events	Rate (%)	VT-SHOCK	NSVT/ATP
S2 (Single)	4	56.2±11.3	0	0.0	0	0
S3 (Double)	16	62.1±7.2	1	6.2	0	1
S4+ (Triple/Burst)	34	64.4±9.7	8	23.5	4	4
Total	54	63.1±9.2	9	16.7	4	5

Chi-square: $\chi^2 = 3.20$, df = 2, p = 0.20. Cochran–Armitage: r = 0.237, p = 0.084.

Table 3. Arrhythmic Events by Etiology and Threshold

Threshold	ICM Events/N (%)	NICM Events/N (%)	Combined (%)
S2 (Single)	0/4 (0.0)	— (n = 0)	0/4 (0.0)
S3 (Double)	0/12 (0.0)	1/4 (25.0)	1/16 (6.2)
S4+ (Triple/Burst)	4/18 (22.2)	4/16 (25.0)	8/34 (23.5)
Total	4/34 (11.8)	5/20 (25.0)	9/54 (16.7)

No NICM patients were induced at S2.

Figures

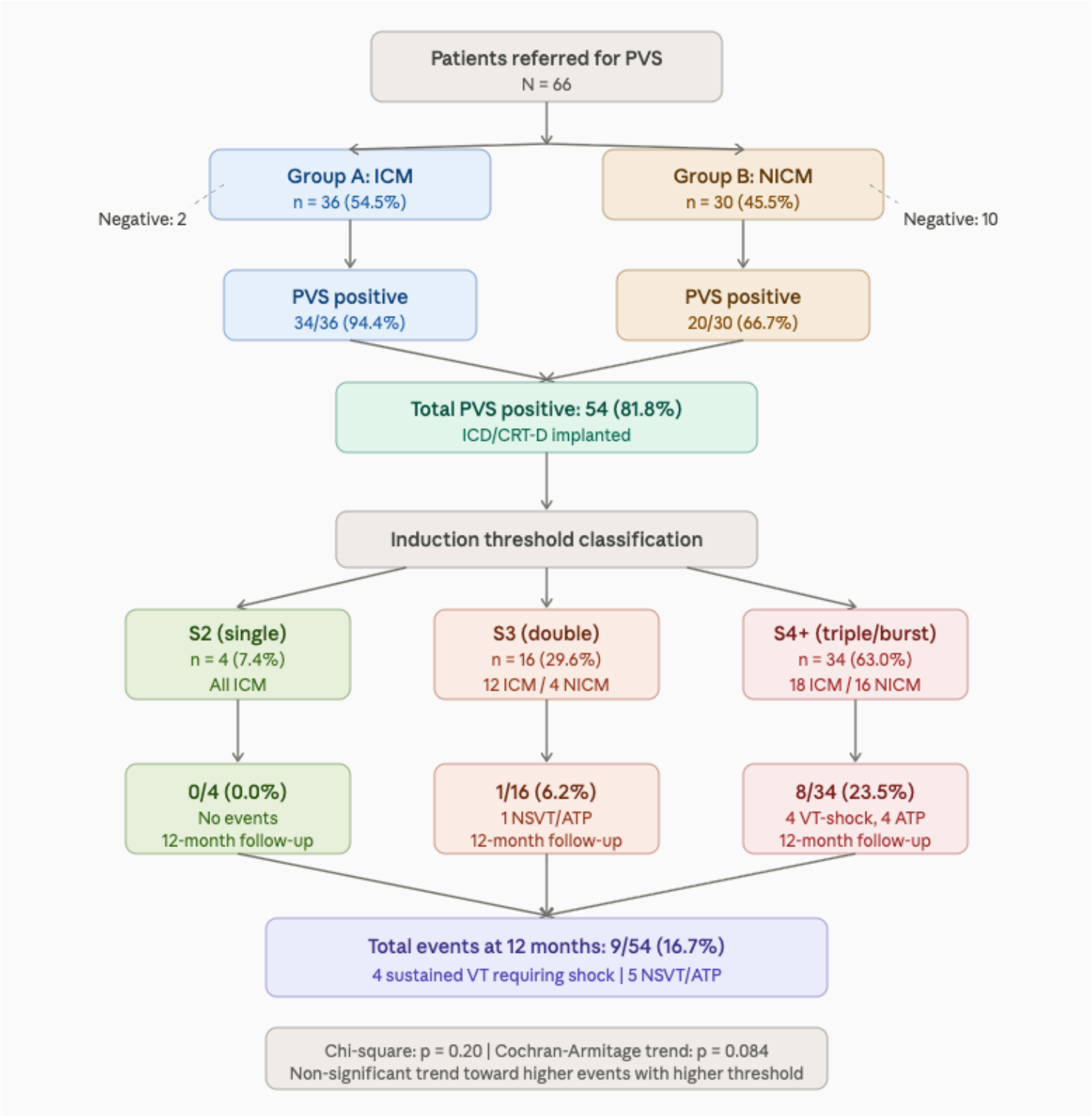


Figure 1

Study flowchart. Of 66 patients referred for PVS, 36 had ischemic (ICM) and 30 had non-ischemic cardiomyopathy (NICM). PVS was positive in 54 patients (81.8%). Among positive patients, induction threshold was classified as S2 (n=4), S3 (n=16), or S4+ (n=34). At 12-month device interrogation, arrhythmic events occurred in 9 patients (16.7%), with higher rates across ascending threshold categories.

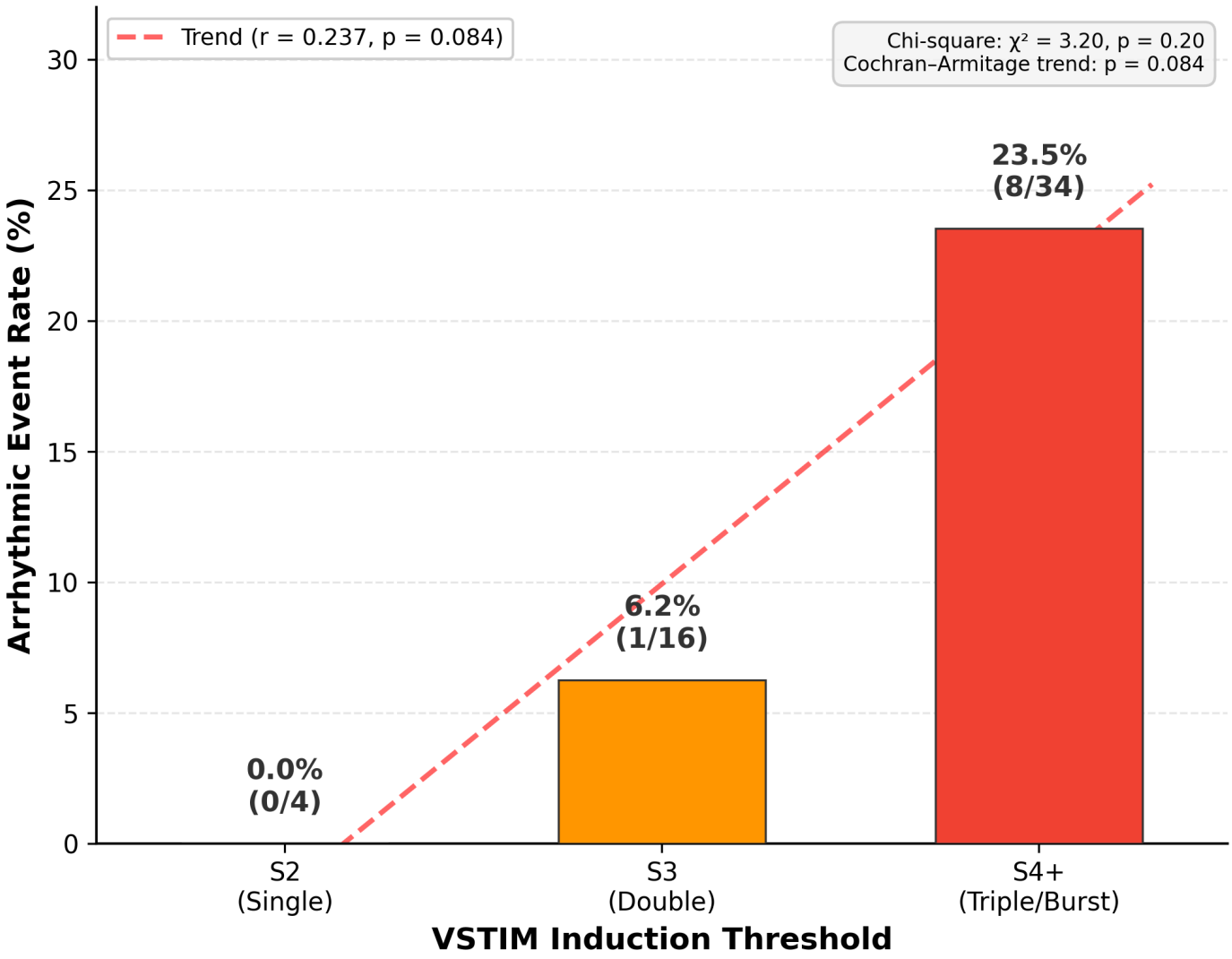


Figure 2

Arrhythmic event rates by PVS induction threshold at 12-month follow-up. Dashed line, Cochran–Armitage trend (r = 0.237, p = 0.084). Inset, chi-square and trend test statistics.

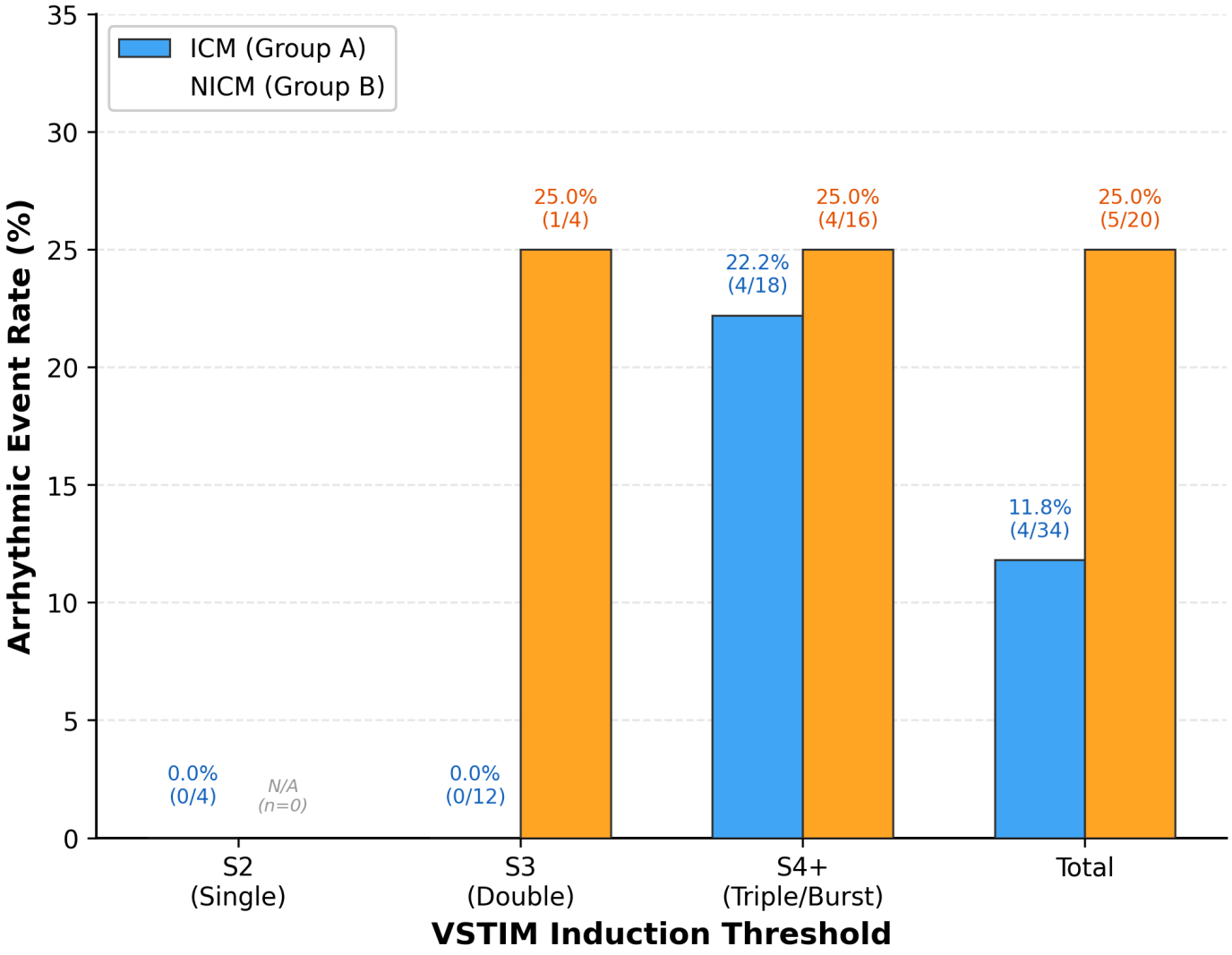


Figure 3

Arrhythmic event rates stratified by cardiomyopathy etiology and induction threshold. Blue, ICM; orange, NICM. No NICM patients were induced at S2.

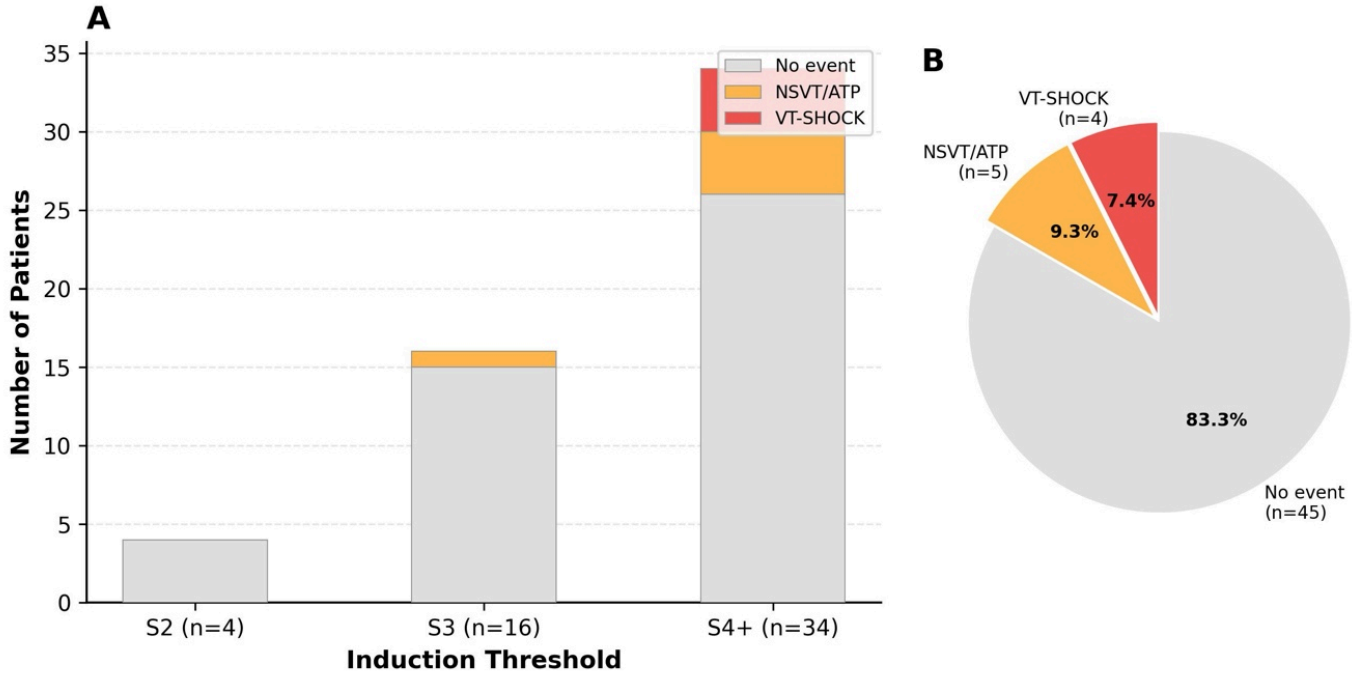


Figure 4

Event type distribution. (A) Stacked bar chart showing outcomes by threshold group. (B) Overall distribution of event types among all 54 PVS-positive patients. ATP = anti-tachycardia pacing; NSVT = non-sustained ventricular tachycardia.