Research Article *Open Access*

Journal of CardioVascular Insights

DOI: doi.org/10.63721/25 /JCVI0104

Programmed Ventricular Stimulation In 2025

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Citation: J Nguadi, KBerrag, J Fagouri, H Bouzelmat, A Benyass (2025) Programmed Ventricular Stimulation In 2025. J of Card Vas Insights 1(2), 01-09. WMJ/JCVI-104

Abstract

Background: *Programmed ventricular stimulation is a vital electrophysiological technique used to evaluate ventricular arrhythmia risks and inform therapeutic strategies, particularly for automatic defibrillator implantation.*

Objective: *This article aims to review advancements in programmed ventricular stimulation protocols, explore new applications, and address limitations and future directions for its role in predictive cardiology.*

Method: *The review synthesizes recent literature on programmed ventricular stimulation, focusing on adaptive algorithms, artificial intelligence integration, and its expanded use in non-ischemic cardiomyopathies and ablation therapy evaluation.*

Results: *Advances in programmed ventricular stimulation include adaptive algorithms and artificial intelligence, improving diagnostic accuracy. Its application now extends to non-ischemic cardiomyopathies and ablation guidance, enhancing therapeutic outcomes. However, limitations such as inter-patient variability and risks of inducing unstable arrhythmias persist. Future prospects involve personalizing protocols using genetic biomarkers, developing non-invasive devices, and standardizing procedures to reduce variability.*

Conclusion: *Programmed ventricular stimulation remains a cornerstone in assessing and managing ventricular arrhythmias, with technological advancements broadening its clinical utility. Despite challenges, its integration into predictive cardiology is promising. Multicenter research is essential to standardize protocols and validate innovations for optimized clinical use.*

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Submitted: 28.10.2025 **Accepted**: 05.11.2025 **Published:** 20.11.2025

Keywords: Programmed Ventricular Stimulation, Ventricular Arrhythmias, Implantable Defibrillator, Sudden Death, Electrophysiology, Antiarythmic.

Introduction

Programmed ventricular stimulation (PVS) is an essential technique in cardiac electrophysiology, enabling invasive exploration of ventricular arrhythmias and precise assessment of sudden cardiac death risk. By delivering targeted electrical stimulations, PVS induces arrhythmias in a controlled setting, providing a unique window into arrhythmogenic mechanisms and underlying pathological substrates [1]. This approach plays a critical role in identifying high-risk patients, particularly those with ischemic heart disease or unexplained symptoms such as syncope, and guides crucial therapeutic decisions, such as the implantation of implantable cardioverter-defibrillators (ICDs) or planned catheter ablations . Despite its recognized efficacy in certain contexts, particularly for monomorphic ventricular tachycardias, PVS has limitations in evaluating polymorphic arrhythmias or certain structural and congenital heart diseases, where its sensitivity and specificity may be reduced [2]. In an era marked by rapid advancements in cardiac imaging, electrophysiological mapping, and stimulation technologies, PVS is part of an integrative approach combining standardized protocols, clinical data, and updated guidelines. This article provides an in-depth analysis of the fundamental principles of PVS, its clinical applications, technical protocols, limitations, and future perspectives, aiming to optimize its use to address the complex challenges of contemporary cardiology.

Materials and Methods

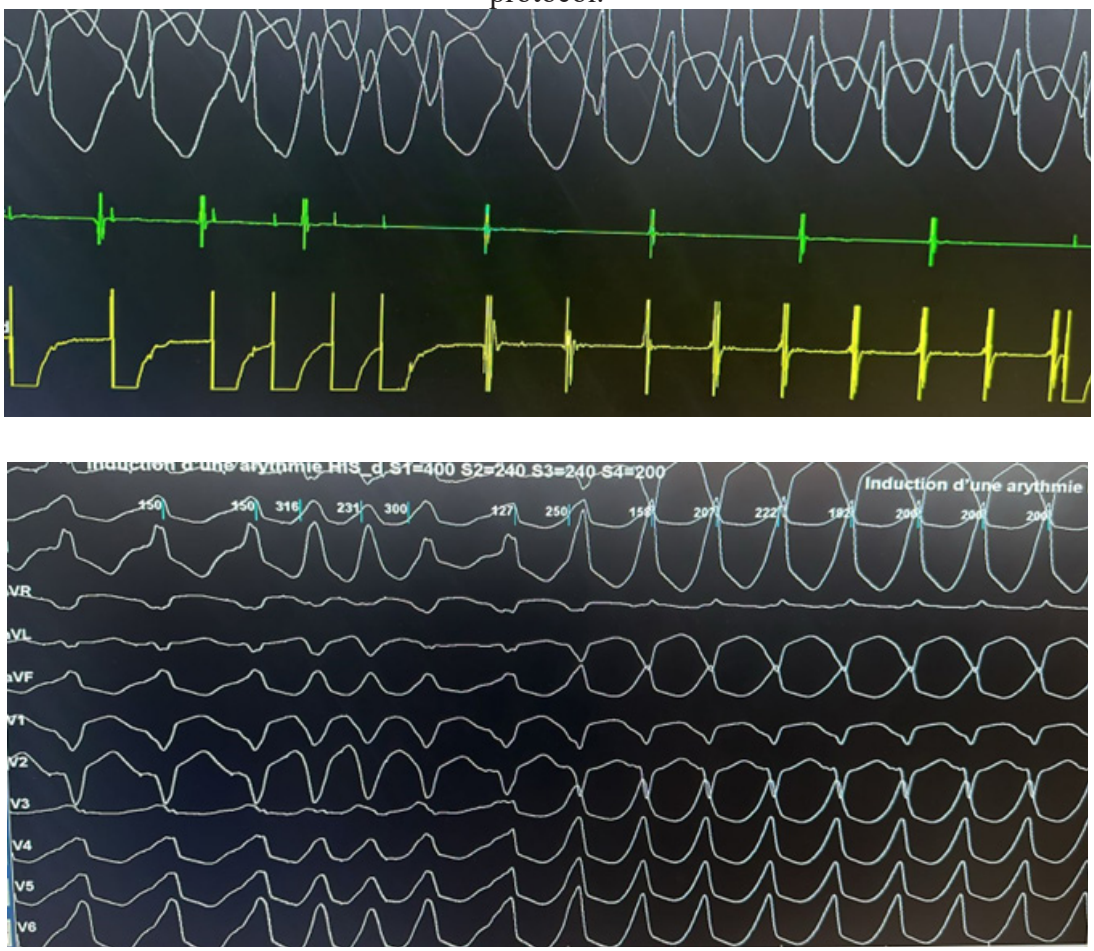
This narrative review synthesizes literature on programmed ventricular stimulation (PVS) up to July 2025, focusing on protocols, clinical applications, advancements, and limitations. A comprehensive search was conducted using PubMed, Embase, Scopus, and Web of Science, combining MeSH terms and keywords like “programmed ventricular stimulation,” “ventricular arrhythmias,” “electrophysiology,” and “artificial intelligence.” Filters included English-language human studies post-1980. Hand-searching of guideline references (ESC 2022, AHA/ACC/HRS 2025) and conference proceedings supplemented the search. Studies on PVS protocols, indications, outcomes, or innovations (e.g., AI, non-invasive methods) were included; non-human studies and small case reports were excluded. Two reviewers screened 512 titles/abstracts, selecting 83 studies after full-text review. Data were extracted on study design, patient characteristics, protocols, and outcomes using a standardized form. Quality was assessed with the Newcastle-Ottawa Scale for cohorts and AMSTAR-2 for reviews. Narrative synthesis integrated findings thematically, summarizing arrhythmia types and clinical implications without meta-analysis due to study heterogeneity.

Programmed Ventricular Stimulation Protocols and Principles

Programmed ventricular stimulation (PVS) is a fundamental invasive electrophysiological technique designed to induce and analyze ventricular arrhythmias by delivering targeted electrical impulses to the myocardium, allowing detailed exploration of the heart’s electrophysiological properties and precise identification of arrhythmogenic substrates, particularly in the context of structural or hereditary heart diseases [3]. Performed using multi-electrode electrophysiology catheters strategically positioned in the right ventricle (often at the apex or outflow tract, but sometimes in the left ventricle or specific sites based on anatomical abnormalities), PVS linked on standardized yet adaptable protocols. These protocols combine fixed stimulation cycles (S1) at predefined frequencies (typically 600, 500, or 400 ms, corresponding to 100–150 beats per minute) to establish a baseline rhythm, followed by the progressive introduction of programmed extrasystoles (S2, S3, or even S4) at decreasing coupling intervals (in steps of 10 to 20 ms, until reaching the ventricular effective refractory period, typically 200–250 ms) [4]. In cases where induction fails, rapid stimulation trains (burst pacing) at cycles of 300 to 200 ms or less are used to stress vulnerable myocardial areas [5]. These stimulations exploit conduction abnormalities, such as reentry circuits in post-infarction scars or repolarization alterations in channelopathies, to provoke monomorphic or polymorphic ventricular tachycardias or ventricular fibrillations, thereby revealing specific pathophysiological mechanisms [6].

Protocols vary by pathology: in ischemic heart diseases, where reentry circuits predominate, aggressive protocols including up to three or four extrasystoles (S1–S4) are often used to maximize the induction of monomorphic tachycardias, as recommended in recent guidelines for post-infarction patients at risk of sudden death [7]. In contrast, in hereditary arrhythmic syndromes, such as those involving ion channels, less aggressive protocols (limited to S1–S2 or S1–S3) are preferred to minimize non-specific responses, with particular attention to coupling intervals to respect the variable refractory period and avoid inducing irrelevant ventricular fibrillations [8]. In congenital heart diseases, protocols are tailored to anatomical abnormalities, sometimes using leadless pacemakers implanted directly in the right ventricle to facilitate access to specific sites, as noted in studies on advancements in stimulation technology [9]. PVS implementation requires strict control of electrical parameters (intensity of 2 to 10 mA, pulse duration of 1 to 2 ms, bipolar or unipolar polarity) and continuous monitoring of electrocardiographic signals (surface and intracardiac ECG) to detect and interpret induced arrhythmias, often complemented by three-dimensional electroanatomical mapping systems to precisely locate arrhythmogenic zones [10]. This technique demands clinical expertise to adjust protocols to the patient's context, considering limitations such as the risk of inducing clinically irrelevant arrhythmias (particularly in non-ischemic contexts where specificity is lower) or inter-operator variability in result interpretation [11]. Recent recommendations emphasize the importance of integrating PVS into a multidisciplinary evaluation, combining imaging (cardiac MRI to identify scars) and clinical data, to optimize its diagnostic and therapeutic value [12].

Figure 1: Electrophysiology lab images, triggering of monomorphic VT during PVS using a 4-extrasystole protocol.



Programmed ventricular stimulation in 2025

Recent advancements in programmed ventricular stimulation (PVS) are transforming its role in managing ventricular arrhythmias, driven by technological progress and new clinical applications. Modern stimulators incorporate adaptive algorithms that dynamically adjust stimulation parameters (coupling, intensity, drive cycles) based on electrophysiological responses, reducing false positives and the risk of inducing unstable arrhythmias [14]. Artificial intelligence (AI) is revolutionizing PVS by using deep learning models, trained on electrophysiological databases, to predict arrhythmia thresholds with unprecedented accuracy while analyzing intracardiac signals in real-time to optimize protocols [14]. Clinically, PVS is expanding beyond ischemic heart diseases to include non-ischemic cardiomyopathies, such as dilated or hypertrophic cardiomyopathy, where it identifies candidates for implantable cardioverter-defibrillators (ICDs) with increased sensitivity [15]. It also plays a key role in ventricular arrhythmia ablation, reproducing arrhythmic circuits to guide procedures with precision, improving success rates by up to 20% according to recent studies [16]. In research, non-invasive approaches are emerging, using transcutaneous devices to stimulate the myocardium without catheters, reducing complications and expanding PVS access, particularly in less-equipped centers [17]. The integration of advanced imaging, such as late gadolinium enhancement MRI or multislice CT, allows correlation of myocardial fibrosis zones with stimulation responses, offering finer risk stratification [18]. Finally, multicenter studies, such as those supported by the ESC, aim to standardize protocols to reduce inter-patient variability and explore the use of genetic biomarkers to personalize PVS, paving the way for more precise and individualized predictive cardiology [19].

Indications for Programmed Ventricular Stimulation

Programmed ventricular stimulation (PVS) is a central electrophysiological tool in rhythmology, with exhaustively defined diagnostic and prognostic indications by European (ESC 2022, class I to III) and American (AHA/ACC/HRS 2017, revised up to 2025, class I to III) guidelines, covering a wide range of clinical scenarios for assessing ventricular arrhythmias and stratifying sudden cardiac death risk in various contexts, including ischemic, structural, congenital, and genetic heart diseases [20]. For diagnostic purposes, PVS is strongly recommended (class I, level of evidence B) for investigating unexplained syncope in patients with ischemic heart disease, particularly post-myocardial infarction, where it can identify sustained monomorphic ventricular tachycardia (>30 seconds or requiring cardioversion) as a likely cause, especially when syncope occurs during exercise, in a supine position, or is preceded by palpitations, symptoms highly suggestive of an arrhythmic origin [20]. It is also indicated (class IIa, level B) to differentiate ventricular tachycardias from wide QRS supraventricular tachycardias in structural heart diseases such as non-ischemic dilated cardiomyopathy, arrhythmogenic right ventricular dysplasia (ARVD), cardiac sarcoidosis, hypertrophic cardiomyopathy, or cardiac amyloidosis, where induction of monomorphic VT enables precise etiological diagnosis and guides management [21]. In congenital heart diseases, such as tetralogy of Fallot, transposition of the great arteries, atrioventricular canal, or single ventricles, PVS is recommended (class IIa, level C) to assess complex arrhythmias, particularly monomorphic VTs related to surgical scars or anatomical abnormalities, with increased utility due to leadless pacemakers implanted directly in the right ventricle, facilitating evaluation of arrhythmogenic circuits [22]. PVS is crucial (class I, level A) for mapping reentry circuits before radiofrequency ablation, particularly in post-infarction monomorphic VTs, where it enables precise localization of arrhythmogenic foci (e.g., slow conduction zones in infarction scars) to optimize ablation efficacy and reduce recurrences [23]. For prognostic purposes, PVS is strongly recommended (class I, level B) for stratifying sudden cardiac death risk in post-infarction patients with a left ventricular ejection fraction (LVEF) ≤40%, where induction of sustained monomorphic VT is a robust criterion for justifying ICD implantation, given a significantly increased cardiac mortality risk (34% in patients with syncope vs. 13% in asymptomatic patients), although VT inducibility remains similar (28–29%) [20]. In patients with LVEF between 36% and 40% and unexplained syncope, PVS is indicated (class IIa, level B) to guide ICD implantation, particularly if monomorphic VT is induced, reflecting a high arrhythmic risk [24]. In cases of preserved LVEF (>40%) but with arrhythmic symptoms (recurrent syncope, persistent palpitations),

utility (class I, level A) in patients with documented spontaneous VT or high-risk syncope, even without obvious structural heart disease, to guide ICD implantation decisions [12]. In genetic syndromes, such as Brugada syndrome, long QT syndrome, hypertrophic cardiomyopathy with a family history of sudden death, or arrhythmogenic right ventricular cardiomyopathy, PVS may be considered (class IIb, level C) to assess inducible VT risk, but its use is limited by the low specificity of ventricular fibrillation (VF) induction, which does not reliably predict clinical risk, making its application controversial in these contexts [25]. PVS is also used (class IIb, level C) to evaluate the efficacy of antiarrhythmic treatments (e.g., amiodarone, sotalol, mexiletine, or beta-blockers like bisoprolol) or to optimize ablation parameters, although its reliability is reduced due to variability in pharmacological responses and interindividual differences in arrhythmogenic circuits [26]. In patients with ICDs experiencing inappropriate or recurrent shocks, PVS is indicated (class IIb, level C) to identify underlying arrhythmic mechanisms (e.g., monomorphic VT vs. non-specific VF) and adjust ICD detection zones or adjunctive treatments [27]. In idiopathic VT (without structural heart disease), PVS may be considered (class IIb, level C) to confirm ventricular origin (e.g., fascicular VT or right ventricular outflow tract VT) and guide targeted ablation, though its use is rare in the absence of severe symptoms [28]. In less common contexts, such as acute myocarditis, constrictive pericarditis, or channelopathies without suggestive symptoms, PVS is rarely indicated (class IIb, level C) due to limited evidence and a high risk of non-specific results, which could lead to unnecessary interventions [7]. PVS is formally contraindicated (class III, level B) for polymorphic arrhythmias or non-specific VF due to their low predictive value and the risk of false-positive results, which could lead to unwarranted ICD implantations or inappropriate treatments [29]. In non-ischemic heart diseases without syncope or clear symptoms, PVS is also discouraged (class III, level C) except in exceptional cases, such as suspected underlying arrhythmias based on non-invasive tests (e.g., Holter ECG showing frequent ventricular extrasystoles) [15]. Finally, in pediatric or young adult populations with complex congenital heart diseases, PVS is used (class IIa, level C) to assess monomorphic VT risk associated with surgical scars or hemodynamic abnormalities, with particular attention to new stimulation technologies (e.g., leadless devices) [8]. In summary, PVS is an indispensable diagnostic and prognostic tool, strongly recommended (class I–IIa) for unexplained syncope, post-infarction monomorphic VTs, sudden death risk stratification in ischemic, structural, and congenital heart diseases, and ablation optimization, with more nuanced indications (class IIb–III) in genetic syndromes, myocarditis, or idiopathic VTs, where its use must be carefully weighed based on clinical risks and benefits.

Programmed Ventricular Stimulation: Results And Interpretations

Criterion	Sustained Monomorphic VT	Non-Sustained Monomorphic VT	Polymorphic VT	Ventricular Fibrillation (VF)
Definition	Ventricular arrhythmia with constant QRS morphology, lasting >30 s or requiring intervention (e.g., cardioversion) due to hemodynamic instability [20].	Burst of VT with constant QRS morphology, lasting <30 s, stopping spontaneously without intervention [15].	Ventricular arrhythmia with variable QRS morphology, variable duration (sustained or non-sustained), potentially progressing to VF [29].	Chaotic, disorganized electrical activity on ECG, leading to loss of effective cardiac contraction, requiring immediate defibrillation [30].
Mechanism	Stable reentry circuit around a myocardial scar (e.g., post-infarction) or structural abnormality (e.g., ARVD, tetralogy of Fallot) [6].	Similar but unstable reentry circuit, preventing arrhythmia persistence [15].	Diffuse electrophysiological abnormalities (e.g., acute ischemia, channelopathies, electrolyte imbalances) or aggressive stimulation, without a stable circuit [29].	Diffuse electrical destabilization, often related to aggressive stimulation or pathological conditions (e.g., ischemia, myocarditis) [30].
Diagnostic Specificity	High (class I, level B): Indicates a stable arrhythmogenic substrate, strongly correlated with arrhythmic syncope or sudden death risk [20].	Moderate (class IIb, level C): Suggests a potential arrhythmogenic substrate, but less correlated with high clinical risk [15].	Low (class III, level B): Non-specific, can be induced in patients without pathological substrate [29].	Low (class III, level B): Non-specific, can occur without significant clinical risk, especially with aggressive stimulation [30].
Prognostic Implications	High risk of sudden death, especially in post-infarction patients (cardiac mortality 34% with syncope vs. 13% without, class I, level B) [20].	Moderate risk, depending on associated factors (e.g., LVEF ≤40%, syncope). Less predictive of sudden death [15].	Low to moderate risk, not predictive of sudden death except in specific contexts (e.g., genetic syndromes, class IIb, level C) [29].	Non-predictive risk, except in specific contexts (e.g., Brugada syndrome with family history, class IIb, level C) [30].
Indications for ICD	Strongly recommended (class I, level A) to prevent sudden death, especially with LVEF ≤40% or unexplained syncope [20].	Not systematic (class IIb, level C), considered if risk factors present (e.g., LVEF ≤40%, syncope, spontaneous VT) [15].	Not recommended (class III, level B), except with additional risk factors (e.g., syncope, family history, class IIb, level C) [29].	Not recommended (class III, level B), except in cases of spontaneous VF or high-risk factors (class IIb, level C) [30].
Indications for Antiarrhythmics	Indicated (class IIa, level B): Amiodarone, sotalol, or beta-blockers (e.g., bisoprolol) to reduce arrhythmia	Considered (class IIb, level C): Beta-blockers or amiodarone for symptomatic repetitive bursts, but	Considered (class IIb, level C): To treat underlying causes (e.g., ischemia, hypokalemia), but limited role without clear substrate [29].	Considered (class IIb, level C): For reversible causes (e.g., ischemia, electrolytes), but limited role without clear

	frequency or ICD shocks [26].	lower priority [15].		substrate [30].
Role in Ablation	Crucial (class I, level A): Mapping reentry circuits for radiofrequency ablation, reduces reliance on ICDs and antiarrhythmics if successful [23].	Possible (class IIb, level C): Considered for frequent, symptomatic bursts, but less effective than for sustained VT [15].	Not indicated (class III, level C): Lack of stable circuit, ablation technically challenging and less effective [29].	Not applicable (class III, level C): No defined circuit [30].
Typical Clinical Context	Ischemic heart disease post-infarction, ARVD, dilated cardiomyopathy, congenital heart diseases with surgical scars [6].	Structural heart disease with moderate risk, documented non-sustained spontaneous VT [15].	Acute ischemia, channelopathies (e.g., long QT), electrolyte imbalances, aggressive stimulation [29].	Acute ischemia, myocarditis, channelopathies, or non-specific aggressive stimulation [30].

Note on Non-Inducibility Non-inducibility during PVS (absence of VT or VF) has a high negative predictive value (class IIa, level B), significantly reducing the likelihood of an arrhythmic cause for syncope or sudden death risk [14]. However, it does not equate to zero arrhythmic risk, as clinically significant arrhythmias may persist without being inducible, particularly in: non-ischemic heart diseases (e.g., dilated cardiomyopathy, sarcoidosis) [7], genetic syndromes (e.g., Brugada, long QT), where external triggers (fever, medications) are not reproduced [25], and congenital heart diseases, where anatomical variations limit inducibility [8].

Management: No systematic indication for ICD or antiarrhythmics (class III, level C), but close follow-up (e.g., implantable ECG monitor, class IIa, level B) is recommended if risk factors persist (e.g., LVEF ≤35%, unexplained syncope) [12]. In summary, sustained monomorphic VT is the most significant result, guiding toward ICD (class I) and antiarrhythmics (class IIa), while non-sustained VT, polymorphic VT, and VF have limited value, and non-inducibility, while reassuring, requires comprehensive clinical evaluation to rule out residual risk.

Limitations of Programmed Ventricular Stimulation

Despite its established role in evaluating ventricular arrhythmias, programmed ventricular stimulation (PVS) has several limitations that restrict its use and interpretation. Inter-patient variability in electrophysiological responses, related to factors such as myocardial fibrosis or ionic alterations, can compromise result reproducibility, making reliable arrhythmia risk prediction challenging in some cases [11]. Additionally, PVS carries a risk of inducing unstable, potentially fatal arrhythmias, requiring rigorous monitoring and technical expertise in the electrophysiology lab, which limits its accessibility in less-specialized centers [5]. The lack of universally standardized protocols leads to variations in practice, affecting data comparability between studies and institutions [76]. In low-risk patients, the utility of PVS remains controversial, as its specificity is reduced, increasing the risk of false-positive results that may lead to unnecessary interventions, such as ICD implantation [15]. Finally, compared to non-invasive methods like microvolt T-wave alternans analysis or advanced imaging, PVS may be less discriminative in certain populations, highlighting the need for an integrated approach to optimize arrhythmic risk stratification [18].

Future Perspectives of Programmed Ventricular Stimulation

The future of programmed ventricular stimulation (PVS) is oriented toward increased personalization and advanced technological integration to optimize its role in managing ventricular arrhythmias. Incorporating genetic and electrophysiological biomarkers,

such as genetic variants associated with ion channels or myocardial fibrosis profiles detected by MRI, will enable tailored PVS protocols, enhancing precision in identifying patients at risk of sudden death [19]. Emerging technologies, such as implantable devices with autonomous stimulation capabilities, could reduce reliance on invasive procedures, while non-invasive approaches using transcutaneous stimulators promise to broaden PVS access in less-specialized settings [17]. The integration of artificial intelligence, combined with advanced imaging (4D MRI, high-resolution CT), aims to predict arrhythmic responses with unmatched precision, facilitating a predictive and preventive approach [13]. However, these advancements require global protocol standardization to harmonize practices and multicenter studies to validate their efficacy [7]. On an ethical level, balancing clinical benefits with potential risks, particularly in vulnerable populations, must be carefully evaluated to ensure responsible adoption of these innovations [12].

Conclusion

Programmed ventricular stimulation (PVS) remains a cornerstone of cardiac electrophysiology, offering a robust approach to diagnosing and stratifying ventricular arrhythmia risk while guiding therapeutic decisions such as ICD implantation and ablations. Recent advancements, including the integration of artificial intelligence, adaptive algorithms, and advanced imaging techniques, enhance its potential by expanding its clinical applications and improving its accuracy. However, PVS limitations, such as inter-patient variability and risks associated with its invasive nature, underscore the importance of targeted use and a multidisciplinary approach. Future perspectives, focused on personalization and non-invasive technologies, promise to make PVS an even more precise and accessible tool, addressing the needs of predictive and individualized cardiology. To maximize its impact, concerted efforts are needed to standardize protocols and validate innovations through multicenter research, while ensuring ethical and responsible adoption to optimize care for patients with complex arrhythmic disorders.

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