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EVALUATION OF THE ABLATION INDEX DURING CATHETER TREATMENT WITHOUT FLUOROSCOPY IN PATIENTS WITH CORONARY HEART DISEASE AND SYSTOLIC DYSFUNCTION

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Aim. To study the ablation index (AI) in the context of catheter treatment of ventricular tachycardia (VT) without the use of fluoroscopy in patients with chronic heart failure.

Methods. Catheter ablation of VT was performed in 47 patients with coronary heart disease and chronic heart failure. Intraoperative parameters of the ablation, including the average ablation index, were assessed. The fact of arrhythmia induction after a series of radiofrequency exposures was assessed. Recurrence of VT was also assessed. The observation period was 12 months.

Results. During surgery after ablation exposure, arrhythmia induction was impossible in 100% of patients. After 12 months of observation, freedom from arrhythmia was 84,8%. Patients without recurrence of VT had a statistically significantly higher mean AI (612 [522,5; 683,5]) than with recurrence of VT (7 (15,2%) patients) (438 [416,5; 462]) (p=0,001). The possibility of predicting recurrence of VT depending on the mean AI value was also assessed. It was found that with the mean AI value greater than or equal to 473, the risk of recurrence of VT is lower (p=0,001).

Conclusions. AI can be used as a parameter for monitoring effective ablation exposure in the context of catheter ablation of VT along with other determinants currently used.

Keywords: ventricular tachycardia; ablation index; heart failure; coronary artery disease; catheter ablation

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Ventricular tachycardia (VT) is defined as a tachycardia (rate >100 bpm) consisting of three or more consecutive complexes originating below the bifurcation of the His bundle, arising from the specialised conduction system, the ventricular myocardium, or both tissues, irrespective of atrial or atrioventricular nodal conduction. Such rhythm disturbances may occur in patients with coronary artery disease as well as in those with various cardiomyopathies. The pathophysiology of VT differs across patient populations [1, 2].

In patients with prior myocardial infarction (MI), most sustained monomorphic VTs are caused by a macro-re-entry mechanism involving the scar zone. Experimental studies have shown that the electrophysiological substrate for monomorphic VT gradually forms during the subacute phase of MI, with no difference observed between VTs induced in the subacute versus chronic phases, as both are associated with comparable sites of early presystolic activation. These sites are predominantly located in the border zone, adjacent to

dense scar areas in both phases [3]. Persistent coronary occlusion usually leads to the development of a dense, transmural central scar core within the region supplied by the occluded artery, surrounded by a thin border zone where fibrous tissue and viable myocardial fibres are intricately interwoven. By contrast, early reperfusion may result in a more complex substrate characterised by non-transmural myocardial necrosis, heterogeneous scarring, and multiple channels of viable myocardium interspersed within the scar and border zones [4].

The development of VT significantly worsens both prognosis and quality of life. Implantation of an implantable cardioverter-defibrillator (ICD) can reduce mortality in patients with VT; however, this therapy does not prevent recurrence of arrhythmias. Implantable devices may, however, provide valuable data on the “burden” of ventricular arrhythmias [5, 6]. In 2022, a hypothesis was proposed suggesting a link between VT burden and mortality. Researchers considered two possible scenarios: one in which

progression of underlying heart disease increases both mortality risk and VT recurrence, and another representing a causal relationship, where disease progression increases the frequency of VT recurrence, thus creating a vicious cycle. This cycle of disease progression increasing VT burden, and in turn elevated VT burden increasing mortality risk due to cardiac pathology, illustrates a self-reinforcing loop [7].

VT accounts for approximately 25% of sudden cardiac deaths (SCD) in the general population, and in patients with cardiovascular disease this figure reaches 50% [8]. A distinct subgroup at risk of clinically significant arrhythmias is patients with chronic heart failure (CHF). The occurrence of arrhythmias in this population further aggravates the course of their disease. At the same time, given that CHF is the natural outcome of most cardiovascular pathologies, the development of ventricular arrhythmias in CHF patients is largely predictable [9]. Implantation of ICD for primary or secondary prevention of SCD is included in several contemporary guidelines [10]. However, ICD shocks may contribute to progression of CHF and impair quality of life [11-13].

The efficacy of catheter ablation (CA) depends on appropriate patient selection, procedural quality, and post-procedural management [14, 15]. Despite the availability of mapping techniques, several critical factors must be met to ensure procedural success. These include achieving transmural and continuity of ablation lines, which requires catheter stability during energy delivery; sufficient application duration; adequate contact force of the ablation catheter with the myocardium; appropriate spacing between adjacent lesions to prevent the formation of conducting channels; and application of radiofrequency (RF) energy with sufficient power and duration. A delicate balance must be maintained, as insufficient RF delivery may result in suboptimal lesion formation, whereas excessive energy increases the risk of complications [16].

METHODS

The study was approved by the Ethics Committee of the National Medical Research Cen-

ter of Cardiology named after Academician E.I. Chazov, Ministry of Health of the Russian Federation (Protocol No. 273 of the Committee meeting, 22 November 2021). A total of 47 patients with coronary artery disease (CAD) and CHF who underwent RFCA for VT were prospectively enrolled. All participants provided written informed consent. The follow-up period was 12 months.

Inclusion criteria were: moderately reduced or severely reduced left ventricular (LV) ejection fraction (EF);

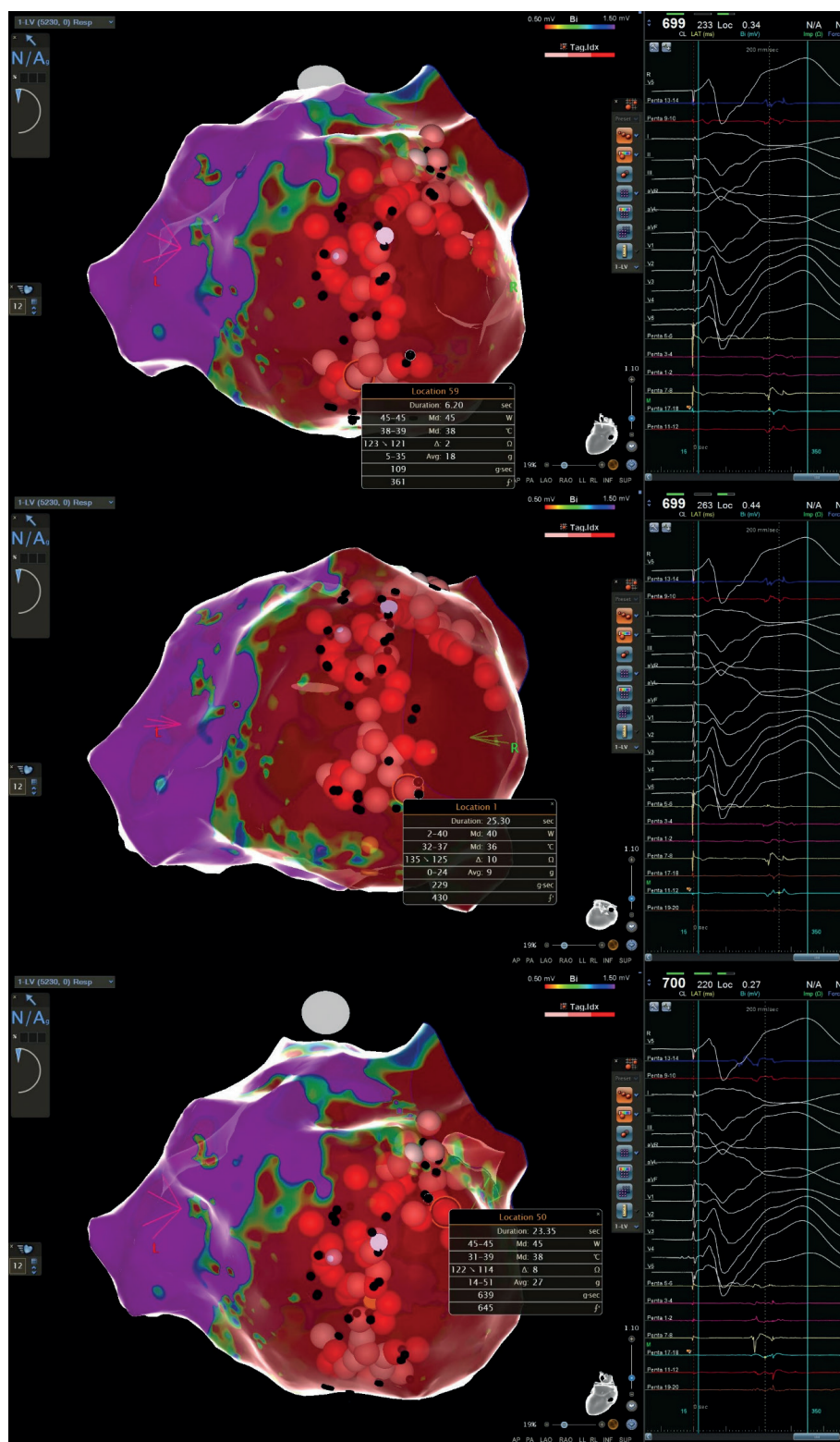


Fig. 1. Three-dimensional electroanatomical map of the left ventricle with volumetric markers indicating RF applications. Colour intensity reflects the ablation index at each site (≤ 400 , white; 400-500, pink; > 500 , red).

New York Heart Association (NYHA) functional class II-III CHF; previous MI; prior revascularisation; and the presence of haemodynamically significant VT. Additional requirements were stable clinical status for at least one

Table 1.

Clinical and demographic characteristics of the patients included in the study

Parameters	Value (n=47)
Male sex, n (%)	41 (87.2)
Age, years	65 [57; 71]
BMI, kg/m ²	28 [26; 30]
NYHA class II CHF, n (%)	28 (59.6)
NYHA class III CHF, n (%)	19 (40.4)
NT-proBNP, pg/mL	756 [493.5; 1106]
Overall LVEF, %	40 [34; 46]
CHFmrEF, n (%)	26 (55.3)
CHFrEF, n (%)	21 (44.7)
Arterial hypertension, n (%)	34 (72.3)
Paroxysmal AF, n (%)	24 (51.1)
Permanent AF, n (%)	4 (8.5)
Artificial rhythm, n (%)	3 (6.4)
Diabetes mellitus, n (%)	9 (19.1)
Chronic kidney disease, n (%)	9 (19.1)
History of interventions	
PCI, n (%)	38 (80.8)
CABG/LIMA graft, n (%)	9 (19.2)
ICD implantation, n (%)	26 (55.3)

Note: BMI - body mass index; NYHA class CHF - New York Heart Association functional class of chronic heart failure; CHFmrEF and CHFrEF - chronic heart failure with moderately reduced and reduced left ventricular ejection fraction (LVEF), respectively; AF - atrial fibrillation; PCI - percutaneous coronary intervention; CABG - coronary artery bypass grafting; LIMA graft - left internal mammary artery graft; ICD - implantable cardioverter-defibrillator.

month prior to enrolment and receipt of optimal medical therapy for CHF for at least three months.

Exclusion criteria were: refusal to participate; NYHA functional class IV CHF; CHF decompensation at the time of enrolment; reversible causes of CHF; surgical interventions within the preceding three months; stable angina of functional class III; and non-ischaemic cardiomyopathy as the underlying cause of CHF.

Catheter Ablation Technique

The procedure was performed under endotracheal general anaesthesia. The internal jugular and femoral veins were punctured, and haemostatic introducers were placed. Through a steerable introducer, a diagnostic 10-pole catheter was advanced into the right ventricle. Transseptal puncture of the interatrial septum was performed. A diagnostic PentaRay catheter (Biosense Webster, Johnson & Johnson, USA) was advanced into the LV cavity under intracardiac echocardiographic guidance. In cases where mapping of the LV outflow tract via the transseptal approach was not feasible, retrograde access through femoral artery puncture was employed. Epicardial VT ablation was performed when epicardial arrhythmogenic foci had been confirmed during prior mapping. In these cases, pericardial puncture was carried out via a subxiphoid approach. Coronary angiography was performed intraoperatively to ensure the safety of RF delivery in proximity to coronary arteries. In certain cases, when technical issues with mapping occurred, a combined approach was used—simultaneous transseptal puncture and right femoral artery puncture.

Mapping was performed using the CARTO 3 system (Biosense Webster, Johnson & Johnson, USA) with a 22-pole diagnostic PentaRay mapping catheter (Biosense Webster, Johnson & Johnson, USA) and a Thermocool SmartTouch ablation catheter (Biosense Webster, Johnson & Johnson, USA; curve D/F) equipped with contact force sensing and ablation index (AI) measurement. Voltage thresholds for distinguishing scar tissue from viable myocardium were set at 0.3-0.8 mV. During activation mapping, the QRS morphology of clinically significant VT recorded on 12-lead ECG played a key role. In voltage mapping, late potentials (extending beyond the QRS complex) were identified on the navigation model during ventricular pacing when clinically feasible (stable haemodynamics, absence of haemodynamically unstable VT induction); otherwise, mapping was performed during sinus rhythm.

Table 2.

Comparison of baseline echocardiographic parameters between patients with and without ventricular tachycardia recurrence

Parameter	Patients with VT recurrence (n=7)	Patients without VT recurrence (n=39)	p
LVEF, %	35 [25; 40]	42 [34; 46]	0,07
LV EDV, mL	170 [125; 280]	167 [135; 201]	0,6
LV ESV, mL	80 [55; 203]	79 [48; 120]	0,4
LV EDD, mm	62 [51; 75]	62 [58; 68]	0,9
LV ESD, mm	48 [38; 63]	47 [39; 53]	0,4
LV aneurysm, n (%)	1 (14,2)	7 (17,9)	0,8
LVPW thickness, mm	0,9 [0,8; 1,0]	1 [0,7; 1,0]	0,6
NT-proBNP, pg/mL	809 [647; 1566]	722 [385; 1012]	0,2

Note: LV - left ventricle; EDV - end-diastolic volume; ESV - end-systolic volume; EDD - end-diastolic diameter; ESD - end-systolic diameter; PW - posterior wall thickness.

The target AI in areas of thinned myocardium (4 mm, LV aneurysmal regions) was ≤ 550 , whereas in regions with myocardial thickness > 5 mm it ranged from 700 to 800. In isthmus ablation for VT, RF delivery was applied until VT termination, followed by consolidation lesions within the target area until the endpoint of non-inducibility was achieved. For RF ablation of

substrate zones with late potentials, RF applications were delivered across all identified sites, with subsequent electrophysiological testing until the endpoint of non-inducibility was confirmed (Fig. 1).

Statistical Analysis

Statistical analysis was performed using Excel 2010 and STATISTICA 10 (StatSoft Inc., USA). Categorical variables are presented as absolute numbers and percentages. The following statistical methods were used: the Mann-Whitney U test. Sample parameters presented in the tables are expressed as M (SD) and Me [Lq; Uq], where M - mean, SD - standard deviation, Me - median, Lq; Uq - interquartile range. A p -value <0.05 was considered statistically significant; values of $0.05 < p < 0.10$ were interpreted as a trend.

Sensitivity and specificity of the studied parameters were also assessed using receiver operating characteristic (ROC) curve analysis. Quantitative assessment of the ROC curve was performed by calculating the area under the curve (AUC). The following scale was applied to interpret AUC values as a measure of diagnostic test performance: AUC = 0.9-1.0, excellent; 0.8-0.9, high; 0.7-0.8, good; 0.6-0.7, moderate; 0.5-0.6, poor.

RESULTS

Among the 47 patients included in the study, 87.2% were male, and the median age was 65 [57; 71] years. The CHF phenotype was predominantly CHF with moderately reduced LVEF (55.3%), although 44.7% of patients had reduced LVEF. Prior to RFCA, 26 patients underwent ICD implantation. Baseline patient characteristics are summarised in Table 1.

All patients were receiving optimal medical therapy for CHF. As antiarrhythmic therapy, all patients were prescribed amiodarone (100%). At the time of enrolment, 5 patients (10.6%) had a VT history of 6 months, 17 patients (36.2%) between 6 and 12 months, and 25 patients (53.2%) ≥ 12 months. Thus, the median duration of VT prior to hospitalisation was 12 months.

All procedures were performed without fluoroscopy, under intracardiac echocardiographic guidance. Intraoperative mapping was performed in all patients: stimulation mapping in 51% ($n=24$), activation mapping in 38.4% ($n=18$), and scar homogenisation in 10.6% ($n=5$). Access was retrograde in 8 cases (17%), transseptal in 33 (70.2%), epicardial in 4 (8.5%), and combined in 2 (4.2%). The median procedure duration was 190 [150; 227] minutes. VT was inducible in all patients at the start of the procedure. Following RF ablation, VT was rendered non-inducible in 100% of patients. Standard RF parameters were applied: RF power 40-50 W, contact force 10-25 g/cm², application time 31 ± 10.4 - 57.65 ± 24.3 s, and temperature 43-46 °C.

Special attention was given to analysis of the AI. During RFCA of VT in areas of thinned myocardium (post-infarction zones or border zones between viable myocardium and post-infarction scar), AI values were deliberately limited to 400-500 to minimise complications, whereas in regions of "thicker" myocardium, AI was increased to 700-800 to improve efficacy.

At 12-month follow-up, ICD interrogation and Holter monitoring were performed. VT recurrence was

documented in 7 patients (15.2%), corresponding to an arrhythmia-free survival of 84.8% following RFCA. Importantly, no VT recurrences were observed in the first 6 months post-ablation.

Baseline echocardiographic parameters were compared between patients with and without VT recurrence. Overall, the groups were comparable; however, there was a trend toward lower LVEF in the recurrence group (Table 2).

AI was analysed in all patients. Those without VT recurrence (39 patients, 84.8%) had a significantly higher median AI (612.0 [522.5; 683.5]) compared with those with recurrence (7 patients, 15.2%) (438 [416.5; 462]); $p=0.001$ (Fig. 2). VT recurrence risk was further assessed as a function of mean AI using ROC analysis. The area under the ROC curve (AUC) for prediction of VT recurrence by mean AI was 0.886 ± 0.049 (95% CI: 0.791-0.982) (Fig. 2). This model was statistically significant ($p=0.001$). The cut-off value for mean AI was 473. Mean AI ≤ 473 predicted a high risk of VT recurrence. Sensitivity and specificity were

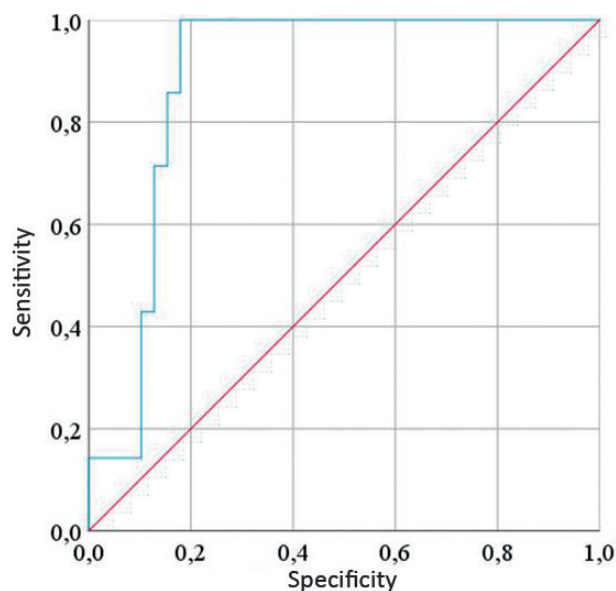


Fig. 3. ROC curve illustrating the relationship between ventricular tachycardia recurrence and mean ablation index.

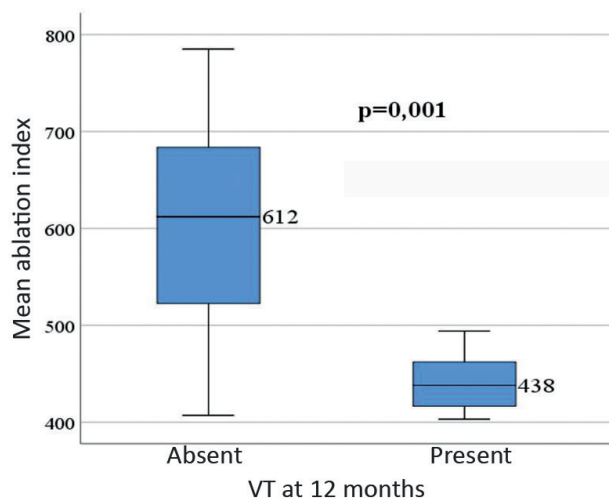


Fig. 2. Mean ablation index values in patients with and without arrhythmia recurrence during follow-up.

85.7% and 84.6%, respectively (Fig. 3). The odds of VT recurrence in patients with mean AI >473 were reduced by 1.125 (95% CI: 0.221-5.714).

DISCUSSION

The main factors contributing to improved efficacy of ablation are accurate identification of the arrhythmogenic substrate and adequate energy delivery to achieve transmural ablation [22]. At the preoperative stage, magnetic resonance imaging (MRI) with contrast enhancement may be performed. Fibrosis/scar zones identified by MRI are, in most cases, the sources of VT. Moreover, MRI can provide information not only on localisation of scar tissue but also on its density and transmural extent. However, MRI has limitations, particularly in patients with implanted ICDs, where artefacts preclude adequate interpretation [23].

During the procedure itself, invasive mapping of the arrhythmogenic substrate is performed to identify zones of slow conduction. RF applications are then delivered to these regions. The primary objective of activation mapping is to identify the critical isthmus, which plays a key role in sustaining tachycardia. However, this approach has certain limitations. Successful mapping requires stable activation sequences and good arrhythmia tolerance, making activation mapping challenging in haemodynamically unstable arrhythmias, tachycardias with variable morphology, or those with complex or non-reproducible induction [24]. Pace-mapping can aid localisation of the VT source by endocardial stimulation at various sites, followed by comparison of the morphology of paced QRS complexes with tachycardia QRS complexes [24]. Together, these mapping methods, combined with preoperative imaging data, allow accurate localisation of the arrhythmogenic substrate for targeted RF delivery.

Several determinants of RF lesion size must also be considered intraoperatively, such as RF power and application duration. Domestic literature describes a study comparing high-power ablation (50 W) with standard-power ablation (45 W), using a standard application time of 60 s. The procedural endpoint of VT non-inducibility was achieved in 100% of patients. At 12 months, arrhythmia-free survival was 82.6% in the high-power group versus 76.2% in the standard-power group ($p=0.0286$). Importantly, this was the first study to evaluate the ablation index (AI) in VT ablation. The mean AI was significantly higher in the high-power group (475.4) than in the standard-power group (461.1), with borderline statistical significance ($p=0.0549$) [25].

In addition to conventional procedural parameters, the authors assessed mean AI, which had previously only been studied in AF ablation. They later published a follow-up article further evaluating the role of mean AI in VT treatment. This study included 63 patients with CAD referred for RFCA of VT. All patients underwent high-power ablation (50 W). Postoperatively, VT was absent in 52 patients (82.6%). The mean AI was significantly higher in the group without VT recurrence (494.9 ± 73.3) compared with those with recurrence (383.2 ± 44.3 ; $p<0.0001$). Each 10-unit increase in mean AI was associated with a 1.37-fold reduction in VT recurrence risk ($p=0.0025$; 95% CI: 1.16-1.77) [26].

Our findings were consistent with these results, demonstrating that higher mean AI values (612 [522.5; 683.5]) were associated with freedom from VT recurrence. Given the absence of reference values for AI in VT ablation, operators referred to AI thresholds established for left atrial ablation. According to the CLOSE protocol, target AI values for the left atrium are ≥ 550 for the anterior wall, ≥ 400 for the posterior wall, and ≥ 300 when chest pain or oesophageal temperature elevation occurs [27]. It is well known that the anterior wall of the left atrium is thicker than the posterior wall, explaining these differences in AI thresholds. These data were extrapolated to VT ablation in our study. When comparing patients with and without VT recurrence, there was a trend toward more advanced CHF in the recurrence group, which may have contributed to the use of lower AI values in this subgroup of patients.

CONCLUSION

Improving the efficacy of RFCA for VT remains an important objective in contemporary arrhythmology. The AI has already demonstrated its value in atrial fibrillation ablation alongside other determinants of lesion formation. Further studies are required to investigate AI in the context of ventricular arrhythmia ablation in order to establish reference values for different anatomical locations, to refine VT ablation protocols, and thereby to enhance procedural efficacy.

The application of higher AI values during RFCA of VT appears reasonable; however, more definitive conclusions cannot yet be drawn due to the limited number of studies available. The role of AI in ventricular arrhythmia ablation warrants further investigation with larger sample sizes and diverse study designs.

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