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POSSIBILITIES OF STEREOTACTIC BODY RADIOTHERAPY FOR THE TREATMENT OF VENTRICULAR ARRHYTHMIAS. CURRENT STATE OF THE ART

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This review article examines current approaches to the use of Stereotactic Arrhythmia Radioablation (STAR) in patients with sustained ventricular tachycardia refractory to medical therapy and catheter ablation. The biophysical and molecular mechanisms of the therapeutic action of the method, experimental data, principles of target planning and imaging, as well as international and our own clinical experience with the method in patients with ventricular tachycardias are presented. Technical aspects of the method, unresolved issues, limitations, and the potential for implementing STAR into Russian clinical practice are discussed separately.

Key words: stereotactic arrhythmia radioablation; non-invasive ablation; cardiac radiotherapy; stereotactic body radiotherapy; refractory ventricular tachycardia; SBRT; STAR.

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Ventricular tachyarrhythmias (VTs) are lifethreatening cardiac rhythm disorders characterised by pathologically accelerated and, as a rule, irregular electrical activity originating in the ventricular myocardium or the conduction system below the bundle of His. Prolonged persistence of VT may lead to haemodynamic instability and cardiac arrest [1].

Sudden cardiac death (SCD) remains one of the leading causes of mortality in developed countries: according to epidemiological studies in the USA, its annual incidence is estimated at 185-450 thousand cases (7-18% of all deaths) [2]. In the majority of cases, SCD is caused by malignant ventricular arrhythmias - sustained VT or ventricular fibrillation, especially in the presence of structural myocardial damage. According to 2023 data from the Russian Federation, surgical interventions for ventricular arrhythmias were performed in 5,369 patients, including 3,578 cases of endovascular destruction of the arrhythmic substrate and 1,791 patients receiving antiarrhythmic device implants. Repeat catheter procedures were performed in 9.6% of patients [3].

Currently, the main treatment modalities for VT are pharmacotherapy, implantation of cardioverterdefibrillators (ICDs), and catheter ablation. Implantable devices effectively prevent SCD by promptly terminating lifethreatening arrhythmias via antitachycardia pacing or electrical defibrillation. However, multiple ICD activations, particularly with frequent VT episodes, are associated with a marked decline in quality of life, increased anxiety and depression, and worse heart failure prognosis [4].

Catheter ablation of VT can achieve sustained arrhythmia control in a significant proportion of patients. Nevertheless, its success rate is lower in cases of deep intramural or subepicardial foci, extensive scarrelated myocardium, and repeated catheter ablations. According to the literature, VT recurrence after ablation may reach 20-50%, often requiring multiple reinterventions. Moreover, some patients are not candidates for ablation due to severe comorbidities, haemodynamic instability, or low myocardial contractility [5].

A particular challenge is presented by cases where VT is not controlled or recurs even after ablations, or where ablation is difficult due to anatomical features (large post-infarction scar, involvement of intramural and epicardial surfaces, etc.) [6]. In this context, there is growing interest in noninvasive treatment modalities that can address the limitations of traditional approaches [7-9]. There remains a clinical need for alternative therapies for patients with refractory or recurrent VT, in whom standard approaches are ineffective or inapplicable, driving the search for novel techniques [5, 10, 11].

One of the most promising directions is stereotactic arrhythmia radioablation (STAR), which is attracting increasing attention as a potential noninvasive alternative when invasive treatment options are limited. As of today, a PubMed search for "stereotactic arrhythmia radioablation" shows a steady growth in the number of scientific publications: from 24 articles in 2021 to 126 articles in 2025, reflecting the growing research interest in STAR technology.

STAR involves a single session, targeted delivery of high-dose ionising radiation to the arrhythmogenic substrate [12, 13]. This approach is based on the principles of stereotactic body radiation therapy (SBRT), which is widely used in oncology, including the treatment of localised lung tumours, liver tumours, and oligometastases [14-16]. The quality and effectiveness of STAR are determined by consensus technical requirements originally developed for stereotactic radiotherapy in oncology. The accumulated experience of high-dose irradiation of thoracic organs, including cardiac structures, has allowed the adaptation of the technique for arrhythmia treatment and served as the basis for the first experiments in radiation therapy for arrhythmology [17, 18].

Initial clinical results of STAR have been promising, demonstrating a marked reduction in arrhythmia frequency in patients with refractory VTs [19, 20]. A systematic review showed that the technique reduces the number of VT episodes by more than 85% with an acceptable safety profile in more than 40 patients, demonstrating high efficacy in patients with refractory VTs that do not respond to standard therapies [21-23]. These data have driven further development of the method: the number of STAR procedures has increased significantly, and the number of publications, including clinical cases, small series, and prospective studies, has grown. Currently, active clinical trials are ongoing to evaluate the efficacy and tolerability of STAR. However, the biological mechanisms of its therapeutic action remain a subject of scientific debate.

In this review, several terms are used that reflect historically established approaches to naming the method. "Stereotactic arrhythmia radiosurgery" emphasises the single-fraction nature of the intervention and corresponds to the concept of radiosurgery as a single-fraction, high-dose, geometrically precise radiation delivery. "Radioablation" highlights not so much the technical parameters of delivery, but rather the biological goal - the creation of a controlled radiation lesion of the arrhythmogenic substrate. The term SBRT (stereotactic body radiation therapy) originated in oncological radiotherapy and is used for high-precision hypofractionated irradiation. The abbreviation STAR (stereotactic arrhythmia radioablation) was introduced by a group of authors led by P. Cuculich and P. Robinson and it is currently the most widely used specific term in the arrhythmological literature. In this review, the term "stereotactic arrhythmia radioablation (STAR)" is used as the main designation.

The aim of this review is to systematise and analyse current scientific and clinical data on the use of STAR in the treatment of VTs refractory to conventional therapies.

This review is based on a structured analysis of the contemporary literature on the use of STAR in refractory VT. Publications were searched in the PubMed and Scopus databases using the following keywords: "stereotactic arrhythmia radioablation", "STAR", "ventricular tachycardia", "cardiac radioablation", "SBRT", "noninvasive VT ablation". The search identified more than 200 publications.

The analysis included publications from 2020-2025 in English or Russian, including prospective clinical studies, cohort series, systematic reviews and metaanalyses,

preclinical experimental studies, as well as clinical guidelines and consensus documents. Studies in which the population consisted of patients with sustained VT refractory to pharmacotherapy and/or catheter ablation were considered. Clinical outcomes, including VT recurrence rate, safety and complication rate, and overall survival, were evaluated.

Excluded were publications in languages other than English and Russian, conference abstracts without full text, single clinical case reports ($n=1$), and duplicate publications of the same cohorts. Due to the descriptive nature of the review, a formal PRISMA selection scheme was not constructed. Publication selection was performed by two independent authors based on eligibility criteria; disagreements were resolved by discussion. A total of 99 sources were included in the final analysis. The authors' own clinical observations were not part of the literature search and are presented separately as illustrative material.

PATHOPHYSIOLOGY OF RADIATION-INDUCED MYOCARDIAL DAMAGE

The pathophysiological basis of electronbeam myocardial damage in STAR involves several mechanisms. Ionising radiation acting on the arrhythmogenic substrate leads to structural and functional changes that suppress pathological electrical activity. Key mechanisms include radiation-induced fibrosis and molecular remodelling of the conduction system components. Together, these processes create an area of altered electrical conductivity that disrupts reentry circuits and reduces the likelihood of arrhythmia recurrence.

It was initially hypothesised that the antiarrhythmic effect of STAR is mediated by apoptosis induction and subsequent fibrosis in the arrhythmogenic cardiac tissue. However, accumulating preclinical and clinical evidence suggests a more complex, multifactorial myocardial response to ionising radiation. One of the main effects is the development of radiation-induced fibrosis, triggered through oxidative stress, microvascular damage, inflammatory infiltration, and myofibroblast activation [24]. Fibrotic transformation electrically isolates the pathological reentry pathways, analogous to the goal of catheter ablation. However, the clinical effect of STAR sometimes occurs much earlier than mature fibrosis develops (which typically takes ≥ 6 months), indicating the presence of additional nonhistological mechanisms.

The study by D.M. Zhang et al. (2021) expanded understanding of STAR mechanisms, showing that, alongside fibrosis, molecular changes - particularly remodulation of intercellular contacts and increased connexin expression - may play a key role [25]. Irradiation of myocardial areas adjacent to scar tissue was associated with increased expression of the sodium channel Nav1.5 and connexin43 (Cx43) protein. The Notch signalling pathway, activated in response to radiation, is thought to play an important role in Nav1.5 activation. Thus, radiotherapy may induce electrophysiological remodelling of the cardiac conduction system, opening new possibilities for noninvasive targeting of VT zones. The same study presented pathomorphological data from four patients (after heart transplantation or autopsy 17-478 days after irradiation), confirming the

absence of pronounced myocardial fibrosis after a single 25Gy dose. Despite this, these patients showed a significant reduction in VT episodes, highlighting the clinical importance of alternative, nonfibrotic mechanisms [25].

Furthermore, studies by M. Amino et al. demonstrated that cardiac irradiation can increase the expression of connexin43, a transmembrane protein that mediates electrical coupling between cardiomyocytes. Increased Cx43 levels were observed both within and outside the irradiated zone; the effect varied depending on the type of radiation (photon, proton, or carbon ions) [26]. It has also been reported that postirradiation reduction in mitochondrial density facilitates more efficient calcium signalling within cardiomyocytes, which may represent a potentially novel mechanism of radiotherapy action [27].

Relationship between VT mechanisms and myocardial scar

VT developing in the context of structural heart disease is predominantly associated with reentry circuits forming in fibrotic myocardial areas. Such changes typically occur after myocardial infarction (MI), inflammatory or infiltrative processes, or cardiac surgery.

In ischaemic cardiomyopathy, scar zones usually follow coronary artery distribution and predominate in the subendocardial layers, sometimes extending through the entire myocardial thickness. In nonischaemic cardiomyopathy, scar transformation may be more heterogeneous - involving endocardial, intramural, or subepicardial layers with focal or diffuse distribution [24, 28]. The most critical areas for initiating and sustaining reentry arrhythmias are zones of slow conduction within scar tissue. These areas serve as the main targets during catheter ablation [29]. The arrhythmogenic substrate may be located on the endocardial or epicardial surface, but often is partially or entirely intramural. This limits mapping to a single side (epi and/or endocardial) and complicates complete anatomical visualisation of the arrhythmic pathway. In patients with scar-related VT, programmed stimulation often induces several morphological variants of sustained monomorphic tachycardia. This may result from multiple reentry circuits, different exit sites, or separate pathways from the same scar zone. Since the scar area is not necessarily the arrhythmia source, accurate localisation and delineation of arrhythmogenic myocardial regions are critical for effective treatment planning.

Animal experimental studies; preclinical studies

The first preclinical study that initiated experimental investigation of the method was the work of A. Sharma et al. (2010). Using a CyberKnife linear accelerator, the authors performed noninvasive STAR and demonstrated the feasibility of delivering precise damage to the electrophysiological arrhythmic substrate [30]. Subsequently, a number of experimental studies investigated the consequences of SBRT in various models: mice, rats, rabbits, pigs, and dogs. The aim was to evaluate morphological and functional cardiac changes after singlefraction irradiation, including in the setting of induced MI. Followup usually lasted up to 8-10 months, with different radiation types used: photon, proton, and heavy ions [31-37].

In most preclinical studies, irradiation targeted either healthy animals or animals after experimentally induced

MI [25, 34, 35]. The main targets were pulmonary veins, the atrioventricular (AV) junction, the cavotricuspid isthmus, and (less frequently) the ventricular myocardium. Typically, irradiation was delivered as a single dose of 25-40 Gy. Information on immediate acute cardiac radiation effects is limited, since significant AV node conduction depression was observed only at extremely high doses (≥ 90 -160 Gy), far exceeding clinical levels [35].

Early radiation effects (two to six weeks)

Signs of local radiation myocardial damage may appear in the first weeks after irradiation. For example, local β irradiation of pulmonary veins (pigs, 60 Gy) at 2-6 weeks histologically showed necrosis of the venous wall and destruction of elastic vascular fibres [38]. In a rabbit MI model, heavyion irradiation (performed on day 14 after MI) increased connexin43 expression in the border zone and remote myocardium as early as 2 weeks after treatment [26]. This was accompanied by a reduction in inducibility of VT and ventricular fibrillation compared with untreated controls [25]. Similarly, in mice after experimental infarction, γ irradiation at 15-25 Gy accelerated conduction in both intact and damaged myocardium; these changes were associated with enhanced expression of sodium channels and Cx43 at 6 weeks after therapy at doses ≥ 15 Gy [25]. Notably, in healthy rabbits, increased Cx43 expression after irradiation persisted up to 1 year, whereas in infarcted animals the effect was less pronounced [26].

Rat studies showed oedema, vacuolisation, and widening of intercalated discs within the first month after irradiation (20-50 Gy). These phenomena intensified at doses ≥ 30 Gy and were accompanied by prolongation of PR and QTc intervals, despite the absence of morphological necrosis, suggesting the involvement of other conductionmodulating mechanisms at early stages [32].

In several largeanimal experiments (minipigs) using STAR with subsequent repeat mapping of targets (25-196 days), a dosedependent reduction in signal amplitude on mapping electrodes was shown, with electrophysiological verification of targets performed after irradiation. The minimum effective dose was ≥ 25 Gy. Bidirectional cavotricuspid isthmus block was achieved 30 days after 40 Gy, and complete AV block after 49 days with 70 Gy [30].

Late effects (3-9 months)

Cardiac morphological changes due to irradiation are strongly dosedependent. For example, β irradiation of the pulmonary vein intima at 60 Gy in pigs caused acute endothelial damage, elastic fibre breakdown, and myocardial necrosis, which in subsequent months were replaced by marked fibrosis (on average at 81 ± 27 days) [38]. Delayed fibrosis is probably explained by a combination of processes, including apoptosis and necrosis, that gradually transform into connective tissue. Some pathological changes (microvascular disturbances, haemorrhages, inflammatory infiltration, fatty degeneration) appear gradually, starting around 3 months after irradiation [25, 31, 32].

For instance, M. Amino et al. showed that after γ irradiation of the left atrium and pulmonary vein ostia, myocardial fibrosis developed at doses > 30 Gy, whereas at 20-27.5 Gy only mild fibrotic changes were observed at 6 months [36, 39]. In an experiment with targeted proton irradiation of the AV node (40-50 Gy) in pigs, AV conduc-

tion slowing was recorded at 10-12 weeks, and histological examination at 3 months revealed areas of fibrosis and necrosis in the node region [33].

Adverse effects of cardiac radiotherapy

Preclinical studies, mainly in pigs, demonstrate dose-dependent cardiotoxicity of stereotactic radiotherapy. At relatively low doses (<35 Gy), only moderate adverse changes - e.g., transient mild reduction in myocardial contractility and limited pericardial effusions - are seen [32].

In contrast, doses >35 Gy are associated with more severe lesions: development of pronounced arrhythmias, acute coronary events, and sudden death in animals, as well as damage to adjacent organs. In one experiment, irradiation of the cardiac region with 37.5 Gy led to the formation of a bronchomediastinal fistula [39]. Additionally, targeted proton irradiation of coronary arteries (up to 40 Gy) caused acute radiation damage to the vessel wall and subsequent luminal occlusion [33].

In addition to foreign data, a Russian research group led by A.Sh. Revishvili and A.V. Golanov conducted an experimental study in domestic pigs to evaluate the safety and efficacy of STAR at different dose levels. The results confirmed high accuracy and conformality of dose delivery and demonstrated a marked dose-dependent therapeutic effect (Fig. 1). Irradiation of the AV node at 40-45 Gy led to complete third-degree AV block (including fatal asystole), whereas 35 Gy did not cause persistent conduction disturbance. Equivalent doses applied to atrial and ventricular myocardium caused transmural changes in the form of focal fibrosis, haemorrhages, and calcification, without involvement of adjacent structures. These results indicate a threshold dose necessary for a reliable electrical effect and simultaneously underscore the need for caution when increasing radiation exposure [40].

Despite encouraging results, questions remain regarding the duration of clinical effect, the sufficiency of the identified mechanisms to explain the early therapeutic response, and the universal applicability of STAR for different aetiologies of tachyarrhythmias. In this regard, a prospective study is underway to evaluate the impact of increasing radiation dose on STAR efficacy and VT recurrence rate [41].

Nevertheless, even with existing uncertainties, in the setting of electrical storm and when no other effective therapeutic options remain, STAR is considered a salvage strategy capable of controlling arrhythmia and significantly improving patient quality of life.

PRINCIPLES OF TARGET PLANNING AND IMAGING IN RADIATION THERAPY

Noninvasive imaging of the treatment substrate

Imaging modalities play a key role in STAR planning, allowing precise identification of arrhythmogenic zones. The most widely used is late gadolinium enhancement cardiac magnetic resonance imaging (LGE-MRI), which depicts areas of macroscopic fibrosis [42-44]. However, the diagnostic yield of MRI is reduced in the presence of implanted ICDs and in nonischemic cardiomyopathies. Comparative studies have shown that LGE-MRI may outperform invasive electroanatomic mapping in identifying intramural and heterogeneous scar areas, although the imaged fibrotic zones may contain viable myocytes [45-47].

Radionuclide techniques, such as positron emission tomography (PET) and perfusion singlephoton emission computed tomography (SPECT), provide functional assessment of perfusion and metabolic activity and may be useful for refining target boundaries in STAR planning, though they have not yet entered routine navigation practice. Recent studies show that the use of ^{99m}Tc SPECT and ^{82}Rb or ^{13}N ammoniaPET allows additional characterisation of ischemic and functional myocardial features [48, 49]. Despite lower spatial resolution compared with CT and MRI, fusion of CT anatomical data with radionuclide images improves target localisation and enhances interdisciplinary planning consistency.

For quantitative assessment and localisation of target areas, the AHA17 segment model (2017) is used, taking into account dominant coronary blood supply [50]. The radiopharmaceutical ^{123}I metaiodobenzylguanidine (^{123}I IMIBG), a norepinephrine analogue, is used to assess myocardial sympathetic innervation with SPECT and scintigraphy in heart failure patients. Although studies are limited, an association between regional innervation defects

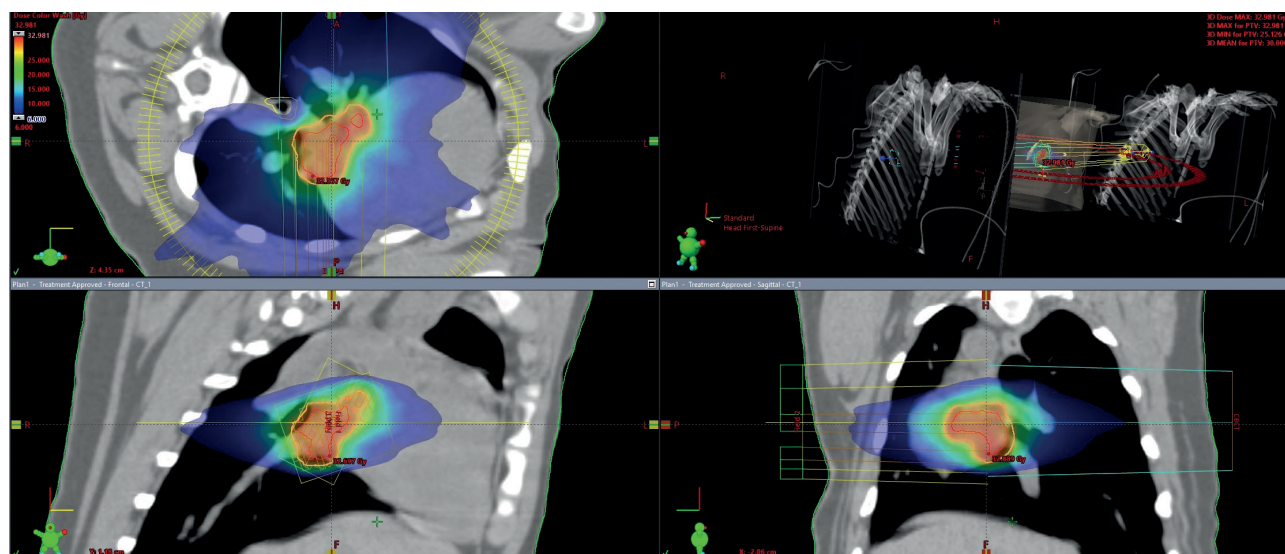


Fig. 1. Dose distribution visualisation, projection of the target area at the left ventricular summit onto native cardiac CT of a pig in the Precision treatment planning system.

and clinical outcomes in VT has been noted [51]. While some data indicate no significant changes in MIBG imaging after STAR [52], others suggest that a reduction in perfusion MIBG mismatch may correlate with successful ablation in ischaemic patients [53]. ^{11}C hydroxyephedrine, a more accurate method for sympathetic imaging, is a promising direction. Radionuclide imaging is noninvasive, allows disease tracking, and may in the future contribute to standardisation of target selection for STAR therapy.

Among the promising and already clinically implemented methods is the noninvasive cardiac activation mapping system AMICARD. Surface ECG mapping involves simultaneous recording of unipolar ECGs from multiple points on the chest, visual representation of results as isopotential and other isoparametric maps, and their diagnostic interpretation with display on an anatomical heart model. Surface ECG mapping is increasingly used for diagnosing cardiac rhythm disorders and predicting life-threatening ventricular tachyarrhythmias. This method identifies the localisation of critical reentry sites, including epicardial arrhythmogenic substrate zones, and defines the concept of noninvasive arrhythmia treatment - from topical diagnosis to therapy.

Invasive electroanatomical mapping (EAM)

Invasive electroanatomical mapping currently remains the primary method for localising the arrhythmogenic substrate in STAR preparation and is considered the gold standard in this field [54, 55]. When standard approaches are insufficient, additional techniques such as noninvasive ECG mapping, epicardial mapping during EPS, and various imaging methods are applied.

A key preparatory step is the integration of anatomical and electrophysiological data (EAM, CT, MRI) to create a unified 3D cardiac model, which serves as the basis for radiation treatment planning [56-58]. In several studies, the irradiation volume was defined by merging EAM data with CT and, when necessary, contrast-enhanced MRI. However, the multicentre RAVENTA study identified discrepancies in clinical target/tumour volume (CTV) determination between EAM and CT, highlighting existing standardisation difficulties [59].

Particular difficulties arise in patients with hypertrophied myocardium, e.g., hypertrophic cardiomyopathy - in such cases, volumes determined by EAM and CT may not match. Additionally, saline infusion during catheter ablation may distort CT visualisation of left ventricular volume.

Artifacts and technical solutions

The use of delayed enhancement CT and MRI is hampered by ICD-related artifacts and high cardiac mobility, which can reduce dosimetric planning accuracy [60, 61]. Various technical solutions are used to minimise these limitations: single-energy metal artifact reduction CT, second-generation cardiac motion compensation algorithms, neural network-based image reconstruction, automated segmentation of anatomical structures, and photon counting CT [62-70].

Target volume and dosimetric approaches in STAR

In the context of STAR, there is active accumulation of evidence on imaging and electrophysiological criteria

used to delineate arrhythmogenic zones, as well as technical treatment parameters - such as irradiated volumes, dosing regimens, and constraints on critical structures. However, current data indicate substantial heterogeneity in approaches to target volume definition, methodologies used, and completeness of preparation descriptions [71].

In clinical practice, the main contours in STAR planning are the clinical target/tumour volume (CTV), which includes the presumed VT substrate (e.g., fibrosis zones, slow activation areas), and the planning target/tumour volume (PTV), formed by expanding the CTV to account for possible positioning errors, respiratory, and cardiac motion. Some protocols also use an internal target/tumour volume (ITV) - CTV with a margin for motion amplitude determined from 4DCT. The PTV is typically obtained by adding 5-10 mm to the ITV, depending on the technique and guidance system.

According to data from several studies, PTV volumes in STAR ranged from 3.5 to 345 mL (median 81.2 mL), CTV from 6.75 to 108.7 mL (median 34.6 mL), while GTV was reported less frequently (median 25.1 mL) and lacks standardised delineation criteria in the context of VT treatment [72]. This is due to the absence of an imaging method that can accurately encompass the entire arrhythmogenic substrate without including viable myocardium, hence safety concerns dominate CTV definition.

The standard STAR dosing scheme is a single fraction of 25 Gy, with at least 95% of PTV volume covered by this dose. At the same time, dose constraints for critical anatomical structures, especially myocardium and coronary arteries, are extremely important. Historically, dose limits were based on cardiotoxicity data from chemoradiotherapy for mediastinal tumours. According to AAPM TG101 recommendations, the heart volume receiving ≥ 16 Gy (V16) should not exceed 15 cm³, and the maximum point dose (Dmax) to the heart should not exceed, by various estimates, 22 Gy [17, 73, 74] (Fig. 2).

Sources of uncertainty in targeting

STAR planning involves several sources of uncertainty that can affect dose delivery accuracy. The main ones include:

- Accuracy of arrhythmogenic substrate localisation. Electroanatomical mapping (EAM) is a key targeting component, but its informative value depends on point density and spatial resolution. It should be noted that the scar zone does not always fully correspond to the VT substrate.
- Integration of multimodal data. Fusion of EAM with CT, MRI, or PET requires precise registration of anatomical landmarks. Registration errors - including rotational and translational - have been shown to occur more frequently when targets are located on the lateral wall or in the interventricular septum [71].
- Transfer of data to the planning system. Manual contour transfer has limited accuracy and may introduce geometric errors. Direct integration of registered datasets improves reproducibility but requires specialised software.
- Cardiorespiratory motion. Cardiac motion and respiratory excursions create overall positional uncertainty, hindering conformal dose distribution.

Accurate targeting and uniform dose delivery require consideration of both respiratory and cardiac motion. Car-

diac pulsations, unlike respiration, are usually incorporated into the ITV or PTV.

Management of respiratory motion

The following strategies are used to compensate for respiratory motion: forming ITV based on 4DCT or multiphase CT encompassing all predicted target positions; gating - synchronising the beam with a specific respiratory phase (e.g., expiration) using external respiratory monitors; tracking - robotic realtime target position tracking (e.g., CyberKnife matches LED markers on the patient's chest with target position); breathhold - irradiation during a fixed respiratory phase. VMAT allows fast procedure execution and may be preferred in unstable patients, whereas CyberKnife permits more precise dose limitation to critical structures, including heart valves [72].

Accounting for cardiac motion

Cardiac motion is an independent source of uncertainty. Unlike respiratory motion, it is related to cardiac cycle phases and is not eliminated by immobilisation methods. The following approaches have been described in the literature:

ECGgated gating - irradiation activated in a specific cardiac phase (usually diastole) using realtime ECG signal; experimental studies on dynamic phantoms demonstrated improved dose delivery accuracy compared with nongated mode [75].

Use of intracardiac landmarks for tracking - ICD or pacemaker leads, which move synchronously with the myocardium, can serve as radiopaque markers on robotic radiosurgery systems [76].

ECGsynchronised CT - multiphase cardiacgated CT allows quantification of cardiac displacement amplitude and its incorporation into target volume.

Formation of cardiac ITV - in the absence of gating or tracking systems, the range of cardiac motion is included in the ITV based on multiphase or 4DCT; in clinical practice, this remains the most practically feasible approach. Currently, commercial full cardiac motion tracking systems on standard linear accelerators are not available.

One promising direction is performing radioablation during a stable paced rhythm with cardiacphase synchroni-

sation, potentially increasing target motion predictability. C.Q.M.Reis et al. (2025) demonstrated the feasibility of this approach at heart rates up to 120 bpm and confirmed dose delivery stability under highfrequency gating [75].

Thus, STAR planning requires a balanced approach, ensuring optimal coverage of the arrhythmogenic zone while strictly adhering to dose constraints. Advances in imaging, motion management algorithms, and planning technologies allow more precise definition of CTV, ITV, and PTV, enhancing both safety and efficacy.

INTERNATIONAL EXPERIENCE WITH STAR

The clinical evidence base for STAR in VT is currently under development. Most published data come from small singlecentre series and prospective cohort studies with limited followup. Randomised comparative studies are few, and their results are pending. Therefore, the data below are structured by increasing level of evidence - from initial clinical series to prospective studies, multicentre initiatives, and ongoing randomised projects.

Clinical series

The first clinical study of STAR for VT was published in 2017 by P. Cuculich and colleagues, including five patients with ischaemic and nonischaemic cardiomyopathy [19]. The work demonstrated the fundamental feasibility of noninvasive targeting of the arrhythmogenic substrate and launched the clinical development of the method.

The largest singlecentre experience to date is a series of 36 patients treated in the Czech Republic since 2014; all cases received a single 25Gy dose [20].

The first Asian clinical experience of STAR for refractory VT was reported from Taiwan [77]. Planning used 3D electroanatomical mapping, delayedenhancement MRI, and dualenergy CT to refine arrhythmogenic substrate localisation. Irradiation was delivered with a single 25Gy fraction using the Varian TrueBeam/Edge system. The study (20192023) included 11 patients with VT refractory to pharmacotherapy and at least one catheter ablation. At a median followup of 53 months, the reduction in VT episodes in the first 6 months was 88%; some patients experienced late recurrences requiring repeat catheter ablation. Oneyear survival was 83%.

At the early stage of clinical implementation, the evidence base was limited: studies included small numbers of patients, no control groups, and relatively short followup. Nevertheless, the initial series demonstrated clinical feasibility and served as a starting point for subsequent transition to prospective studies and international multicentre initiatives.

Prospective studies

Clinical application of STAR began in 2018 and rapidly spread internationally. A key milestone was the phase I/II prospective trial ENCOREVT (ElectrophysiologyGuided Noninvasive Cardiac Radioablation

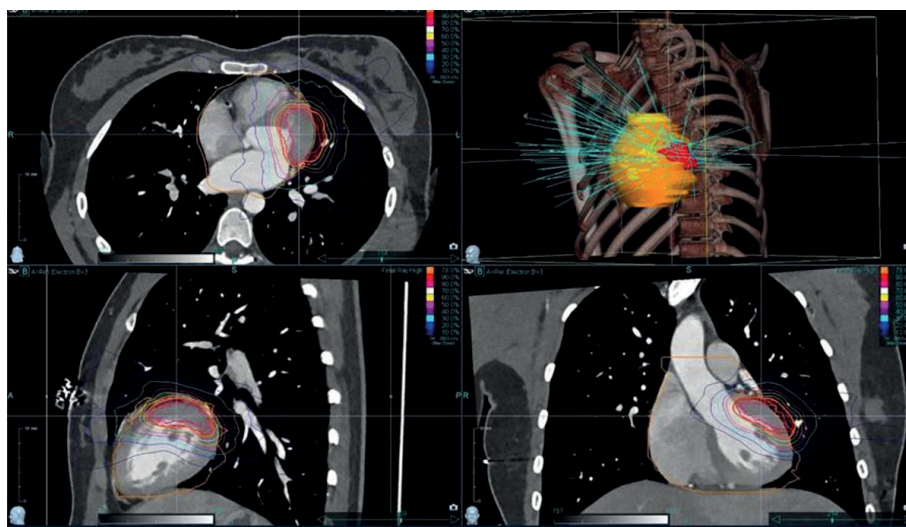


Fig. 2. Dose distribution visualisation, projection of the target area in the left ventricle onto native cardiac CT of a patient in the Precision treatment planning system.

for Ventricular Tachycardia), whose results were published in 2019. This work showed that STAR technology in refractory VT patients reduced VT episodes by 94% and significantly reduced ICD activations [78].

Multicentre initiatives and standardisation

The method's development continued within the largescale panEuropean initiative STOPSTORM (Standardized Treatment and Outcome Platform for Stereotactic Therapy Of Reentrant Tachycardia by a Multidisciplinary consortium) - a consortium aimed at standardising STAR approaches. It includes 31 clinical and scientific institutions from eight European Union countries with significant experience in radiotherapy and interventional arrhythmology. At the time of STOPSTORM launch, 84 STAR procedures had been performed, with 8 of 22 participating centres simultaneously conducting their own national clinical trials. Most centres used 25 Gy in one fraction. Targeting approaches varied: the most common were EAM during VT (96%), pacemapping (75%), and analysis of late potentials and lowvoltage areas in sinus rhythm [48].

Experience from German centres within the consortium reflects not only high clinical interest but also efforts towards harmonisation and standardisation of key aspects of STAR, including targeting, planning, and quality control [1]. Research within STOPSTORM continues: interim reports are regularly published to refine efficacy, safety, and optimise treatment planning.

A significant step was the multicentre STAR benchmark planning study involving 20 clinical centres [79]. Sixtyseven treatment plans were developed for three typical clinical scenarios of sustained VT, covering a wide range of anatomical and technical features, including proximity to critical structures and metal artifacts from implanted devices. Unlike traditional validated protocols, this study intentionally allowed variability to reflect real clinical practice. Based on pooled analysis and expert discussions, STAR planning recommendations were formulated. Expert groups reached consensus on key parameters: prescribed dose (25 Gy single fraction), procedure duration, general requirements for radiation technique, dose calculation methods, and approaches to tradeoffs between PTV coverage and critical structure constraints. However, discussion continues on several technical and clinically relevant aspects. This study also reflected the heterogeneity in STAR treatment planning approaches across European centres and confirmed the need for further harmonisation of both technical and clinical parameters. Given the limited safety and efficacy data in large cohorts, reaching consensus on optimal methodology remains a priority.

Current and planned randomised controlled trials

The largest ongoing international multicentre randomised trial, approved by US regulatory authorities, is RADIATEVT (Cardiac RADIoablation versus repeat catheter Ablation: a pivotal randomized clinical Trial Evaluating safety and efficacy for patients with highrisk refractory Ventricular Tachycardia, NCT05765175), initiated by Washington University in St. Louis. The trial plans to enrol 380 patients in 30 centres to directly compare noninvasive STAR with repeat invasive ablation in patients with refractory monomorphic VT (ischaemic or nonischaemic

aetiology) with reduced LVEF ($\leq 49\%$) and amiodarone failure or intolerance. Patients will be randomised 1:1 to repeat catheter ablation or noninvasive radioablation using the experimental Varian platform. The procedure requires close interdisciplinary collaboration between interventional arrhythmologists and radiation oncologists. The primary endpoint is to prove noninferiority of STAR with superior safety compared with repeat catheter ablation; secondary endpoints include quality of life and arrhythmia event rate. Although RADIATEVT results are not yet published, its mere conduct underscores the growing interest in STAR as a promising alternative to traditional methods.

Systematic reviews and metaanalyses

A comprehensive analysis of clinical studies on STAR for refractory VT was performed by Gupta et al. [80]. The authors conducted a systematic literature search up to March 2024. The aim was to identify clinical studies describing STAR use in VT patients. Only studies with at least three patients who underwent STAR and provided quantitative data on recurrence rates of arrhythmias and/or ICD interventions were included. Reviews, metaanalyses, duplicate publications, and individual case reports were excluded. Ultimately, 23 studies published between 2017 and 2024, covering 225 patients, were included. The main analytical approach was to calculate rate ratios of VT episodes, antitachycardia pacing activations, and ICD shocks before and after STAR, using a randomeffects model. The time boundary between "before" and "after" was set after a sixweek blanking period to exclude shortterm procedural effects. In addition to efficacy, complication rates and overall survival data were analysed.

Demographic characteristics: mean age 67 years, majority male (~85%), ischaemic cardiomyopathy as aetiology in about half (52%). All patients had severe refractory VT not responding to antiarrhythmic drugs and multiple catheter ablations (mean 1.9 procedures per patient). Several studies emphasised that SBRT was used as a last resort, palliative measure when other options were exhausted.

Almost all cases used a standard radiation dose of 25 Gy in a single fraction; only a few studies reported variations (e.g., 2223 Gy). Mean PTV volume was 84 mL. Followup after procedure ranged from 5.8 to 28 months, average about one year. Thus, this metaanalysis represents the most comprehensive summary of clinical data on STAR for refractory VT to date.

Reports of severe adverse events were relatively rare. The most common were pericardial complications (8%), including radiation pericarditis, effusion, and fibrosis, and pulmonary manifestations such as pneumonitis and interstitial fibrosis (5.8%). Nevertheless, several severe late complications were recorded, notably fistulas between pericardium and oesophagus. Two such cases were reported: oesophagopericardial fistula [81] and gastropericardial fistula, both developing several months after STAR. Despite adverse outcomes, overall STAR demonstrated marked clinical benefit. Before irradiation, the pooled VT episode rate was 25.7 per patientmonth, and ICD interventions were 29 (including ATP and shocks). After STAR, these decreased significantly to 2.3 VT episodes and 3.9 ICD interventions per patientmonth, respectively - roughly a tenfold reduction. The rate ratios (after/before) were 0.10

for VT, 0.09 for ICD shocks, all statistically significant ($p < 0.00001$).

Despite this significant overall reduction, complete remission was not achieved in all patients. Within the first six months after treatment, nearly half of patients experienced recurrent VT, about 30% received ICD shocks, and 20% required repeat ablation - either invasive or another STAR course. Nevertheless, the substantial reduction in arrhythmic burden and painful ICD discharges suggests improved quality of life for most patients.

Importantly, no worsening of left ventricular systolic function was observed: mean LVEF before SBRT was 30.9%, after 32.4% ($p = 0.3$, not significant). Given the short followup and severity of patient conditions, these data are encouraging. Pooled survival estimates showed overall survival at 12 months of 72%, and at 24 months 57%, corresponding to approximately 43% deaths over two years. As the authors emphasised, the main cause of death was not arrhythmic events but progression of heart failure [82].

Thus, cardiac STAR demonstrates high potential as a noninvasive treatment for refractory VT in patients with limited therapeutic options. Despite the severity of baseline status and certain risks, a significant reduction in arrhythmia burden without deterioration of cardiac function is observed in most cases. However, the high mortality in

this population underscores the need for further studies to assess longterm efficacy and survival impact.

SERIES OF CLINICAL CASES FROM THE AUTHORS' PRACTICE

The first clinical case of STAR for refractory VT in Russia was performed under the leadership of A.Sh. Revishvili and A.V. Golanov [83]. The transition to human application was based on own largeanimal experimental studies and the international data accumulated by that time confirming the efficacy and safety of the technology [40]. Below are clinical observations obtained within the local clinical pilot programme. Treatment was approved by the local ethics committee; all patients provided informed consent for the procedure and for use of anonymised data in scientific publications.

Radiosurgical treatment was performed in a 57-year-old patient with ischaemic cardiomyopathy and recurrent VT refractory to antiarrhythmic therapy and repeated radiofrequency ablations. Preprocedure electrophysiological mapping allowed precise localisation of arrhythmogenic areas in the interventricular septum and posterolateral apical segments of the left ventricle, forming the basis for target planning. Irradiation was delivered on a TrueBeam linear accelerator (Varian Medical) under respiratory con-

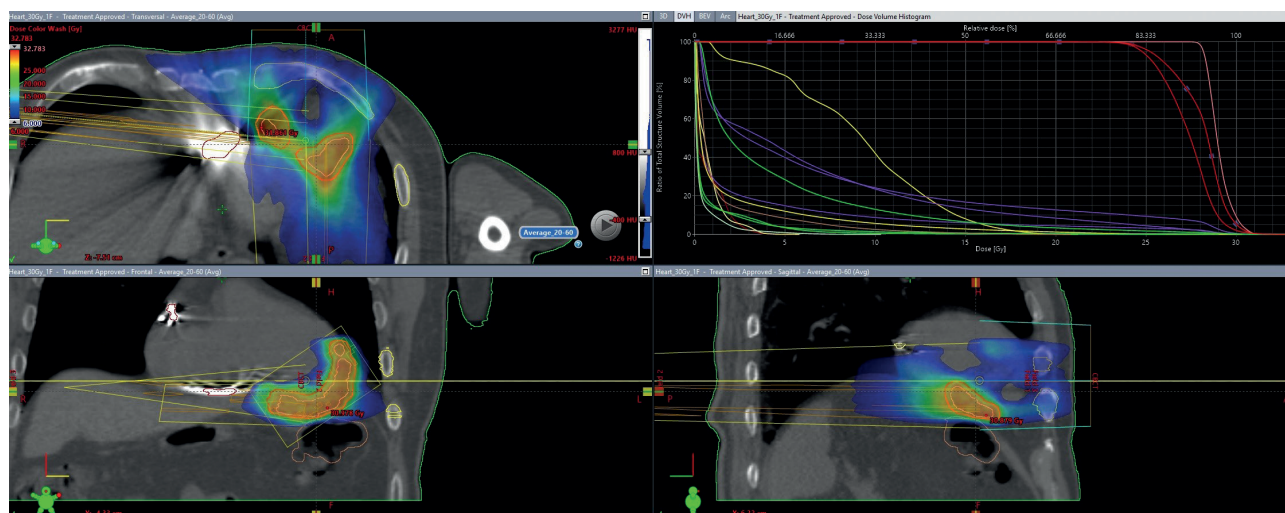


Fig. 3. Dose distribution visualisation, projection of the target area in the left ventricle onto native cardiac CT of a patient in the Eclipse treatment planning system.

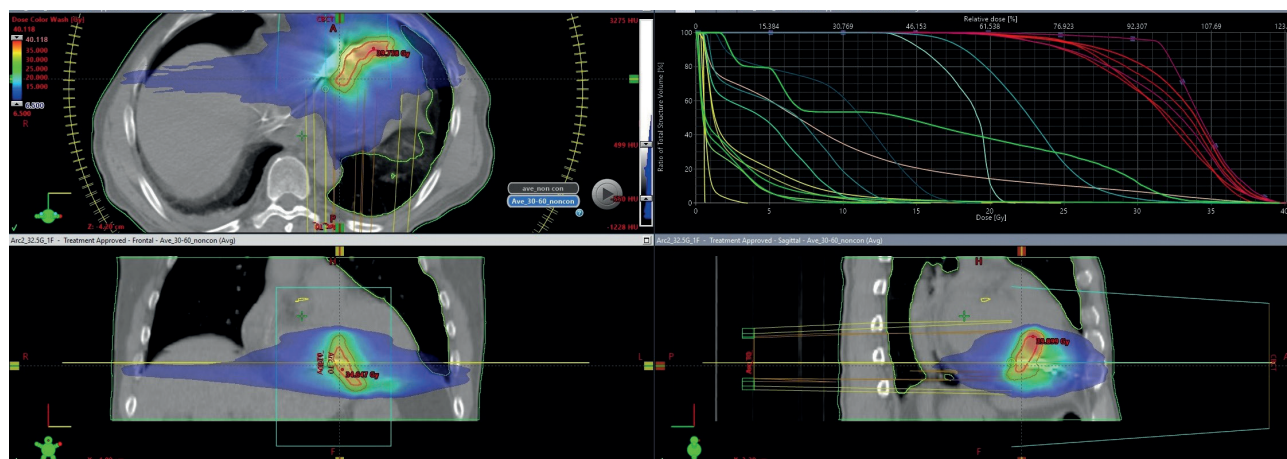


Fig. 4. Dose distribution visualisation, projection of the target area in segments 1416 of the left ventricle onto native cardiac CT of a patient in the Eclipse treatment planning system.

trol, as a single fraction of 25 Gy to 95% of the PTV (46 cm³) via two coplanar arcs. All dose constraints for critical structures were met; dose distribution visualisation is shown in Fig. 3. In the first weeks, rare arrhythmic episodes persisted, but from day 64 and throughout follow-up (450 days), no VT episodes were recorded. No adverse effects or signs of radiation damage to surrounding tissues were observed.

The second patient treated within the local STAR pilot programme was a 60-year-old man with severe ischaemic cardiomyopathy. History included MI, coronary artery bypass grafting, and implantation of a CRTD device (Brava DR). Despite pharmacotherapy and prior RFA, sustained VT episodes persisted, including forms with ventricular rates >110 bpm. Recurrent paroxysms impaired quality of life and increased the risk of adverse outcomes. Given refractory arrhythmia and lack of response to conventional methods, noninvasive STAR was performed. As in the first case, treatment was carried out by a multidisciplinary team led by A.Sh. Revishvili and A.V. Golanov using a TrueBeam linear accelerator [83]. Planning targeted the arrhythmogenic substrate in segments 1416 of the left ventricle. GTV volume was 18.5 cm³, PTV 40.9 cm³. The prescribed dose was 30 Gy in a single fraction, respecting all dose constraints (Fig. 4). The plan consisted of two full coplanar arcs (Fig. 5). From the date of the procedure (07.02.2022), the patient experienced no VT episodes, indicating potential efficacy even in the most complex clinical situations.

These results demonstrate not only technical feasibility but also clinical viability, opening prospects for further application of STAR in Russian arrhythmological practice.

DISCUSSION

STAR is an innovative noninvasive treatment for refractory VT, primarily in patients with postinfarction myocardial scars. The method is at an active stage of investigation, and issues of optimising STAR efficacy and safety continue to be debated.

At the preclinical level, two main mechanisms of antiarrhythmic action of single-fraction cardiac irradiation have been described. First, at doses above about 30 Gy, radiation-induced myocardial fibrosis and coagulation necrosis develop in the target zone, potentially electrically

isolating the arrhythmogenic substrate. Second, at “moderate” doses (20–25 Gy), electrophysiological modulation occurs without gross scar formation – through activation of signalling pathways (Notch, etc.) that alter ion channel expression. However, clinical data on STAR mechanisms remain heterogeneous, hindering the development of a universal approach to optimal dose and volume selection. This underscores the complexity of radiobiological effects and the need for individualised treatment planning for each patient.

Attempts are being made to standardise STAR internationally. In particular, the STOPSTORM project has developed recommendations for unifying the irradiation methodology and outcome reporting. Preliminary agreement has been reached on the advisability of detailed documentation of STAR procedures according to the ICRU №91 international radiotherapy standard (target volumes, doses, control points, etc.). Undoubtedly, further experience accumulation will refine technical parameters and improve reproducibility across centres.

STAR efficacy in clinical studies is mainly assessed by reduction in VT recurrence rate in patients with previously refractory arrhythmia. In most studies, a reduction in episodes is observed after 6 weeks postirradiation [84–86]. One study reported an 88% reduction in VT burden within 6 months [77].

The largest metaanalysis to date, by A. Gupta et al., showed a statistically significant reduction in VT episodes, ATP activations, and ICD shocks after STAR (approximately 10fold, $p < 0.00001$), confirming the method’s pronounced antiarrhythmic effect [80].

Importantly, the reduction in arrhythmic activity was not accompanied by worsening of systolic function – LVEF remained stable after irradiation, suggesting not only efficacy but also functional safety of the approach. Nevertheless, recurrences persisted in some patients, sometimes requiring repeat ablation, including another STAR course.

The high mortality in the analysed cohort, mainly due to heart failure progression, underscores the need for better patient selection and further study of long-term outcomes. Also, standardisation of the technique remains an issue: different studies used varying targeting and irradiation protocols, which may affect reproducibility.

Against this background, technical aspects of plan-

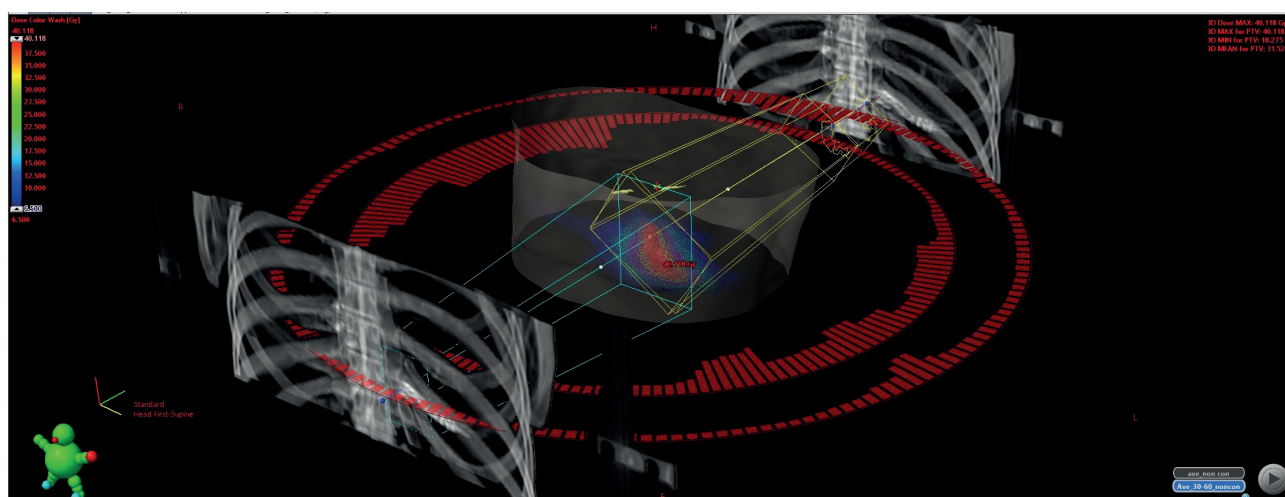


Fig. 5. Irradiation scheme with two full coplanar arcs.

ning and dose delivery gain particular relevance. However, there remains a high risk of recurrence, especially with heart failure progression or new arrhythmogenic substrates [47, 57]. Moreover, the 25Gy dose to the ITV may be insufficient at the periphery of the target, especially with cardiac and respiratory motion. This is confirmed by the detection of reentry mechanisms at the edge of the irradiated scar in patients who underwent repeat catheter ablation.

Historically, cardiac irradiation was limited due to cardiotoxicity risks, particularly observed in nonsmall cell lung cancer treatment [87, 88]. According to AAPM TG101, the heart volume receiving 16 Gy (V16) should not exceed 15 cm³, and the maximum dose 22 Gy [17, 73, 74]. These constraints are particularly relevant when considering STAR in patients with significant systolic dysfunction.

Irradiation of large heart volumes in such patients may lead to further deterioration of contractile function. Ionising radiation affecting extensive myocardial areas carries the risk of cardiomyocyte loss and loss of contractility of remaining viable tissue. Accumulated data indicate a correlation between increased irradiated volume (ITV) and worsening pump function, up to heart failure decompensation. Therefore, caution is required in target selection, especially in patients with already reduced EF.

Complications and safety of STAR

In published studies, acute toxicity of STAR is described as relatively low, while late complication rates may be underestimated due to short followup (usually up to 1 year). The most commonly described complications are transient nausea, pericarditis, oesophagitis, and pneumonitis (mild to moderate severity) [74]. In the vast majority of cases, they regress with conservative therapy and do not lead to permanent disability.

Pericardium

Pericarditis is a manifestation of acute toxicity and has been described in clinical series, occasionally requiring medical therapy [74]. Pericardial fibrosis has also been reported [89]. In animal studies, sterile pericardial effusions were observed, typically decreasing over time [32].

Oesophagus and gastrointestinal tract

Oesophagitis is a critical complication due to the anatomical proximity of the oesophagus to the posterior heart wall. Severe complications have been reported, including fatal oesophagopericardial fistula and gastropericardial fistula requiring surgery [90]. Currently, no evidence supports pharmacological prevention of these rare but serious complications. Emphasis is placed on careful planning: strict dose constraints for the oesophagus and stomach, use of diaphragmatic breathing, and other beamsparing measures.

Lungs and bronchi

Radiation pneumonitis is among the most frequently reported acute complications after STAR and is detected on followup CT, usually at 3 months. According to K. Banfill et al., lung changes occur in about 2030% of patients, mostly benign, with only a few requiring corticosteroids [74].

Risk is higher in patients with preexisting interstitial lung disease. Data from radiotherapy for cancer patients show that the probability of radiation-induced lung injury, including pneumonitis and subsequent fibrosis, depends on total dose and irradiated lung volume (Nuytens et al., 2016), which should be considered in STAR planning [91].

In experimental settings at high doses (37.5 Gy), severe bronchial complications, including bronchomediastinal fistula, have been described [39].

Coronary arteries and valves

Development of coronary atherosclerosis 23 years after STAR has been reported, especially when vessels are close to the PTV. In such cases, longterm followup with planned ischaemia assessment is advisable.

In the Czech study (Hašková et al., 2024), 25% of patients showed worsening of mitral regurgitation several months after STAR, three requiring surgery [20]. Another observation (M.H. Van der Ree et al., 2023) showed deterioration of valve function by ≥ 2 grades in some patients at one year [92].

Rapid progression of aortic stenosis has also been reported [89]. Therefore, regular echocardiography is recommended, especially if valves are near the irradiation zone [73].

Interaction with implantable devices

According to a multicentre study, STAR at the employed doses is not associated with clinically significant ICD dysfunction: sensing, pacing thresholds, and impedance remained stable, and a single device reset had no clinical consequences [93].

Nevertheless, distal electrode location should be considered during planning. Irradiation of myocardium at the contact site could theoretically raise pacing thresholds and lead to loss of capture, potentially limiting device therapy.

According to published data, maximum dose to ICD electronic components varied from 0.04 to 0.603 Gy, with mean values around 0.02 Gy. Some authors (e.g., Blanck et al., 2020) recommend limiting the maximum dose to electronics to ≤ 0.5 Gy [75]. Monitoring dose to device components should be a mandatory part of planning [94].

Mortality

Patients with VT refractory to catheter ablation are at high risk of death. In several studies, oneyear mortality reached 28%, comparable to 32% observed with early VT recurrence after ablation [95]. VT itself may be both a cause of death and a marker of progressive heart failure. In the VANISH trial comparing ablation with pharmacotherapy, oneyear mortality was 1015%, with no significant difference between groups [96]. Similar data are reported by a metaanalysis, whereas an individualised metaanalysis showed a significant mortality reduction after ablation [97, 98]. Most patients included in recent SBRT studies had previously undergone multiple ablations, reflecting the severity of their condition. Many were not considered candidates for further procedures and thus could not be included in traditional randomised trials. According to a metaanalysis, more than half of deaths within one year after STAR were due to heart failure progression, and only 6.5% to arrhythmia recurrence [82]. Therefore, the impact of STAR itself on survival remains to be determined.

FUTURE DIRECTIONS

One of the key technical limitations of STAR is cardiac and respiratory motion. Displacement of the arrhythmogenic substrate reduces dose delivery accuracy and may lead to underdosing of PTV periphery. This is especially critical when using high doses on small volumes.

In an experiment by C.Q.M. Reis et al., a gating system (beam delivery synchronised with a specific respiratory or cardiac phase) synchronised with ECG was tested using a dynamic phantom simulating heart and lung motion [75]. Results showed high concordance of delivered dose with the plan (up to 9899% gamma pass rate) with ECG gating, whereas without motion management accuracy dropped to 5567% (especially with combined heart and respiration).

These findings underscore the importance of implementing gating and/or tracking in clinical STAR, particularly when the target is near radiosensitive structures. The authors noted stable operation of the TrueBeam linear accelerator in highfrequency intermittent mode, critical for singlefraction delivery. Although the study used a physical model (no living patient) and limited scenarios, it convincingly demonstrates the advantages of motion management technologies. Clinical implementation of such systems - ECG synchronisation, optical myocardial tracking, robotic tracking systems - may improve precision and safety, especially for targets near critical structures.

STAR is an innovative and rapidly evolving method whose clinical potential requires thorough investigation in wellcontrolled studies. Its efficacy should be evaluated in the context of current progress in invasive electrophysiology, including the introduction of pulsed field ablation and ultralow temperature cryoablation.

A key nearterm priority remains the conduct of high-quality prospective and comparative studies. These should include detailed characterisation of patient clinical profiles, description of antiarrhythmic therapy used, ablation data, and selection criteria. Standardised monitoring of arrhythmia recurrence, survival, and other clinically relevant cardiac outcomes is essential. Special attention should be paid to longterm monitoring of cardiac and valve function, as well as systematic recording of radiotherapy complications using unified terminology. In this context, international initiatives such as the STOPSTORM consortium can play a leading role in harmonising methodological approaches and standardising reporting.

Despite accumulated evidence, several fundamentally important unresolved questions remain. First, a deeper understanding of the molecular and electrophysiological mechanisms of STAR action is required - including effects on the conduction system, ion channel expression and function, microcirculation, inflammatory response, and neurocardiac interactions. Second, the optimal dosing and fractionation regimen, including the possibility of reirradiation for recurrences, needs to be defined. Determining the radiosensitivity of the arrhythmogenic substrate and the timing of clinical effect is of paramount importance.

Furthermore, the choice of optimal target and targeting strategy remains open. Is it more appropriate to irradiate the entire scar zone or only the critical reentry circuit components? Validation of methods for integrating imaging data with electrophysiological and functional studies, including assessment of noninvasive EAM (ECGI) accuracy compared with invasive guidance, is needed.

Detailed analysis of the effect of STAR on myocardium, coronary arteries, and valves requires longterm

observation and meticulous evaluation of delayed effects. Only on the basis of such data can the true safety profile be determined.

A decisive step in proving STAR's effectiveness will be the conduct of randomised trials comparing it with conventional treatment modalities in various clinical scenarios. This includes comparison with repeat catheter ablation in patients with failed primary intervention, as well as with catheter ablation in highrisk patients. If efficacy is confirmed in these groups, STAR could be considered as a firstline therapy in certain patient categories.

Thus, while possessing significant potential as a noninvasive alternative in malignant arrhythmia treatment, STAR requires further validation based on robust, reproducible data. Expansion of its role in clinical practice should rely exclusively on results from highlevel randomised and multicentre studies.

LIMITATIONS AND PROSPECTS FOR STAR IMPLEMENTATION IN THE RUSSIAN FEDERATION

Despite encouraging clinical results from leading foreign centres, widespread implementation of STAR in routine clinical practice in the Russian Federation is currently limited by several systemic and technical factors. The procedure requires specialised equipment, primarily linear accelerators with STAR capabilities, as well as a wellfunctioning multidisciplinary team including an arrhythmologist, radiotherapist, radiologist, medical physicist, and cardiac imaging specialist [99]. Such infrastructure is currently available only in a limited number of centres, substantially reducing reproducibility.

Moreover, STAR is not yet included in the clinical guidelines of the Ministry of Health of the Russian Federation and does not have official status as an approved medical technology. This restricts its use in routine practice, precludes funding within the compulsory health insurance system, and complicates the unification of patient selection and procedural approaches. Significant challenges also relate to imaging: precise target definition considering cardiorespiratory motion requires hightech methods such as 4DCT, ECGsynchronised studies, and integration of anatomical and electrophysiological data. The absence of such capabilities in some centres negatively affects planning quality and, consequently, potential treatment efficacy.

In addition, the lack of national clinical protocols and guidelines forces clinicians to adapt foreign approaches, which is not always feasible given domestic infrastructure specifics. The absence of longterm national followup data, especially regarding delayed complications (effects on conduction system, coronary arteries, valves), necessitates particular caution in patient selection, limiting the use of the method in patients with predictably long life expectancy. Nevertheless, prerequisites for STAR development already exist in Russia. The first clinical procedure in the country was successfully performed by a multidisciplinary team led by A.Sh. Revishvili and A.V. Golanov using a TrueBeam linear accelerator.

In the future, the development of the method will be facilitated by establishing a regulatory framework, expand-

ing the number of centres with the necessary competence, and integrating Russian institutions into international research consortia. With institutional support and continued clinical piloting, STAR may occupy an important place in the therapy of refractory ventricular arrhythmias, especially when conventional approaches have been exhausted.

CONCLUSION

STAR is one of the most discussed innovative approaches for the treatment of refractory VTs. To date, published data demonstrate a marked reduction in arrhythmia burden after a single 25Gy fraction. Initial clinical results are encouraging, particularly in patient groups where other methods have failed or are technically impossible.

However, the method remains novel and insufficiently studied. In some cases, VT recurrences occur within the first/second year of followup, and the impact on longterm survival and cardiac function remains an open question. It must also be considered that the procedure requires a high degree of technical equipment and coordination among specialists, as well as individual patient selection with mandatory multidisciplinary discussion.

Nevertheless, the potential of the method is evident. With continued scientific support and accumulation of clinical experience, STAR may establish a solid place among treatment strategies for patients with severe, resistant ventricular arrhythmias - primarily as a noninvasive alternative in highrisk cases.

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