Received: 26 September 2021 | Revised: 7 November 2021 | Accepted: 9 November 2021

DOI: 10.1111/anec.12914

ORIGINAL ARTICLE

WILEY

Clinical valuation of ST changes in a group of patients with ventricular arrhythmias: The inSighT Study

Jerzy Krzysztof Wranicz MD, PhD[1] | Michał Kałowski MD[1] | Dirk Bastian MD[2] | Aparna Jaswal MD[3] | Christof Kolb MD, PhD[4] | Edgar Zitron MD, PhD[5] | Iwona Cygankiewicz MD, PhD[1] | Krzysztof Kaczmarek MD, PhD[1]

[1]Department of Electrocardiology, Medical University of Lodz, Lodz, Poland

[2]Department of Cardiology, Section for Clinical Electrophysiology, Fuerth, Germany

[3]Cardiac Pacing and Electrophysiology, Fortis Escorts Heart Institute, New Delhi, India

[4]Deutsches Herzzentrum München, Klinik für Herz- und Kreislauferkrankungen, Abteilung für Elektrophysiologie, Munich, Germany

[5]Department of Cardiology, Universitätsklinikum Heidelberg, Heidelberg, Germany

Correspondence
Krzysztof Kaczmarek, Department of Electrocardiology, Medical University of Lodz, Lodz, Poland.
Email: medkrzych@yahoo.es

Funding information
St.Jude Medical/Abbott, Grant/Award Number: CR-12-014-ID-HV

Abstract

Background: The inSighT study was designed to determine the prevalence of ischemic changes as recorded by implantable cardioverter–defibrillator (ICD) ST deviations in intracardiac electrocardiograms (EGM) over the 24 h preceding malignant ventricular arrhythmias (VT/VF).

Methods: The study enrolled patients with known coronary artery disease (CAD) or high risk of future development of CAD implanted with an ICD equipped with an ST monitoring feature (Ellipse™/Fortify Assura™, St. Jude Medical). Device session records were collected at each in-clinic follow-up. EGM ST levels of the beats over the 15 minutes prior to VT/VF events were compared using a t test with those from a baseline period of 23–24 h prior to the VT/VF event. All events with $p < .05$ were visually inspected to confirm they were evaluable; additional criteria for exclusion from further analysis included inappropriate therapy, aberrant conduction, and occurrence of VT/VF within 24h prior to the current event.

Results: The study enrolled 481 ICD patients (64 ± 11 years, 83% male) in 14 countries and followed them for 15±5 months. A total of 165 confirmed VT/VF episodes were observed, of which 71 events (in 56 patients, 34% of all patients with VT/VF) were preceded by significant ($p < .05$) ST-segment changes unrelated to known non-ischemic causes. None of the analyzed demographic and clinical factors proved to be associated with greater odds of presenting with ST-segment changes prior to VT/VF episode.

Conclusion: In this exploratory study, characteristic ST-segment changes, likely representative of ischemic events, were observed in 34% of all patients with VT/VF episodes.

KEYWORDS
implantable cardioverter–defibrillator, intracardiac electrocardiograms, ST-segment changes, ventricular fibrillation, ventricular tachycardia

1 | INTRODUCTION

The role of implantable cardioverter–defibrillators (ICDs) in the prevention of sudden cardiac death (SCD) is well documented in a number of randomized trials and supported by guideline documents of various cardiac societies (Al-Khatib et al., 2017; Ponikowski et al., 2016; Priori et al., 2015). ICDs detect and terminate malignant, potentially fatal ventricular arrhythmias, but they do not prevent recurrence of further life-threatening arrhythmias. One of the most common reasons for arrhythmic events is the progression of ischemic heart disease (IHD) (Bunch et al., 2007). It has been documented that long-term mortality risk among ICD patients with IHD is correlated with the time elapsed from last coronary revascularization (Barsheshet et al., 2011). Similarly, it is proven that patients implanted with an ICD longer after the myocardial infarction have a higher chance of developing ventricular tachyarrhythmias (Boriani et al., 2012).

Ischemic events are established to cause ST-segment deviations in intracardiac electrocardiograms (EGMs) both in animals and in humans (Fischell et al., 2005; Mendenhall et al., 2012; Williams et al., 2008). Some ICDs manufactured by St. Jude Medical (now Abbott) enable constant beat-to-beat monitoring of the intracardiac ST segment using implanted ICD leads. Similar systems have demonstrated that this approach might be a reliable method of identifying ischemic events (Fischell et al., 2010). However, the predictive value of the ST monitoring feature in identifying pending arrhythmic events remains unknown.

The inSighT study was designed to determine the prevalence of ischemic changes as recorded by ICD ST deviations over the 24 hours preceding malignant ventricular arrhythmias treated by ICD, and to define their temporal relationship to these events. We reasoned that prediction of worsening ischemia could play an important part in allowing physicians to identify ICD patients with greater mortality risk and enabling them to intervene with appropriate therapy.

2 | METHODS

The inSighT study was a prospective, non-randomized, exploratory, international multicenter investigation in patients with recently implanted single- or dual-chamber ICD equipped with an ST monitoring feature and ShockGuard™ algorithm (Ellipse™/Fortify Assura™, St. Jude Medical). Implantation details are summarized in Table 1. Other inclusion criteria included the following: age ≥18 years, the projected burden of ventricular pacing of not more than 20% of the time, diagnosed coronary artery disease (CAD), or high risk of future development of CAD in investigators' opinion. Patients were excluded in case of electrocardiographic abnormalities that could interfere with ST-segment analysis, including long-standing persistent or permanent atrial fibrillation/atrial flutter (AF/AFl), complete heart block, uncontrolled ventricular bigeminy or trigeminy (premature ventricular contractions on regular basis), severe left ventricular hypertrophy resulting in inter-ventricular conduction delay (IVCD), and intermittent bundle branch block (BBB). Other exclusion criteria were pregnancy (ongoing or planned) or declared non-compliance with control visit schedule.

TABLE 1 Implantation details

Implantation details, *n* (%)	
Device model, *n* (%)	
1259 (Fortify Assura VR)	54 (11.25)
1359 (Fortify Assura VR)	60 (12.5)
1277 (Ellipse VR)	102 (21.25)
1377 (Ellipse VR)	54 (11.25)
2259 (Fortify Assura DR)	59 (12.29)
2359 (Fortify Assura DR)	48 (10.00)
2277 (Ellipse DR)	65 (13.54)
2377 (Ellipse DR)	37 (7.71)
Fortify ST	1 (0.21)
Implantation side, *n* (%)	
Left side	470 (97.92)
Right side	10 (2.08)
System position, *n* (%)	
Sub-pectoral	104 (21.67)
Subcutaneous	375 (78.13)
Data missing	1 (0.21)
RV lead position, *n* (%)	
RV apex	381 (79.38)
RV outflow	12 (2.50)
RV septum	82 (17.08)
Other	5 (1.04)

Abbreviation: RV, right ventricle.

Patients were prospectively enrolled between December 2012 and August 2014 by 49 centers in 14 countries (Australia, Canada, Finland, France, Germany, Hong Kong, India, Italy, Japan, Korea, the Netherlands, Poland, Saudi Arabia and the United Kingdom). Each participating site's Institutional Review Board (IRB) or ethics committee accepted the study protocol. The study was registered in ClinicalTrials.gov (NCT01685047). All participating subjects signed written informed consent.

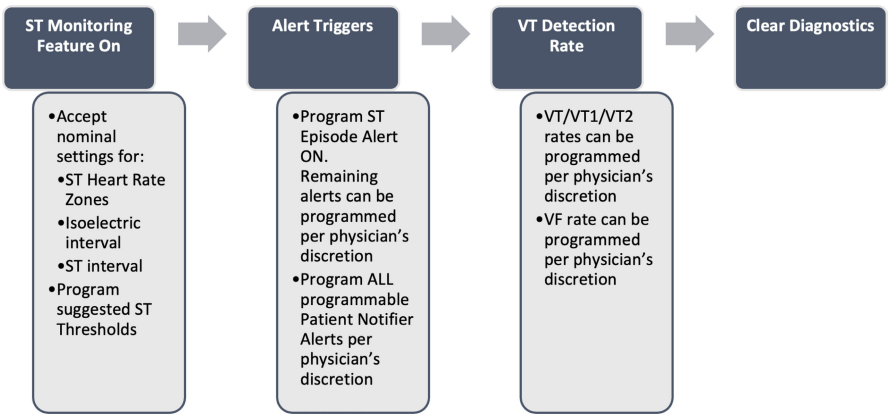
The ST monitoring feature allows reviewing diagnostic data of the ST segment, including ST deviation trend, ST histogram data, and ST episode log. A detailed description of the ST monitoring feature capabilities can be found in Table 2. ShockGuard™ algorithm prevents inappropriate therapy delivery in case of noise generated by ICD lead damage. All devices were programmed according to the algorithm as shown in Figure 1.

Follow-up for all subjects was continued until the last enrolled subject completed their 12-month follow-up visit. Patients were assigned to two groups, depending on physicians' preference to use a remote control system (Merlin.net; SJM) or not. The only

TABLE 2 Description of ST monitoring feature capabilities and ST diagnostic data display

ST monitoring feature	Description
ST deviation trend	Range of ST deviation values (in mV) collected over the past 1 or 6 months, including the min, max, and most frequently occurring ST deviation values for each ST heart zone
ST histogram data	ST histogram displaying all ST deviation values for each heart rate zone.
ST episode log	ST episode log lists the 30 most recent ST episodes stored in the device's memory. The ST monitoring feature is capable of detecting and reporting 2 types of ST Episodes: • Type I—detected in the resting heart rate zone, and the heart rate has not decreased >10 beats per minute over the past 10 min, or an ST episode is detected in an elevated heart rate zone and persists 10 min or longer • Type II—detected in the resting heart rate zone, and the heart rate has decreased >10 beats per minute over the past 10 min, or an ST episode is detected in an elevated heart rate zone and persists less than 10 min
ST diagnostic window	The window shows the following: • ST baseline: frozen rhythm display taken at the time of the episode or the baseline measured 24 h prior to the triggering event. • ST snapshots: The ST snapshots are 4-second recordings of the ST EGM channel taken every 90 s, providing a record of events just prior to the triggering event (last 15 min)

FIGURE 1 Algorithm of ST monitoring feature and ICD programming



difference between the groups was the follow-up schedule. In Group 1, alert events, including ST-segment changes, were collected via a remote control system, and in-clinic follow-up visits were scheduled every 6 months. Group 2 did not use a remote control and had alert events reviewed during device interrogation every 3 months.

Device session records were collected during each in-clinic follow-up. In patients with recorded ventricular tachycardia (VT) or ventricular fibrillation (VF) episodes, the ST-segment deviation over the 15 min prior to the event, each relative to the pre-P-wave level, was compared using Student's *t* test with those from a period of 23–24 h prior to the event. All events with significant ST-segment changes ($p < .05$) were further visually inspected to confirm they were evaluable; additional criteria for exclusion from further analysis included inappropriate therapy or known non-ischemic cause of ST-segment changes, for example, aberrant conduction or occurrence of another VT/VF episode within 24h prior to the analyzed event. Statistical analysis was performed to determine whether there were any demographic or medical factors that could be linked with higher odds of the characteristic ST-segment changes preceding VT/VF episode.

The study protocol also included dobutamine stress echocardiography (DSE) in every patient after confirmed VT/VF episode, unless previous DSE had been performed not later than 6 months before the arrhythmic event and no new symptoms of IHD were present.

Statistical analysis was performed using Statistica software, version 13.3 (StatSoft, Inc.) with statistical significance set at $p < .05$. ST-segment deviation prior to VT/VF event was compared with the baseline using Student's *t* test. A logistic regression model was applied to determine whether there were any demographic and clinical characteristics in the group that displayed the characteristic ST-segment changes.

The study was supported by grants from St. Jude Medical/Abbott.

3 | RESULTS

3.1 | Clinical characteristics

A total of 481 participants were enrolled (mean age was 64 ± 11 years, 83% of subjects were men). Mean time of follow-up was 15 ± 5 months. One of the patients was withdrawn from the study after enrollment due to protocol deviation (the patient was implanted with ICD not containing ST Management Feature). Detailed baseline characteristics of the studied population are described in Table 3.

TABLE 3 Participants' characteristics

Group's characteristic	Study group ($n = 480$)
Age, mean ± SD	63.95 ± 11.29
Men, *n* (%)	400 (83.33)
Height (cm), mean ± SD	170.46 ± 8.48
Weight (kg), mean ± SD	79.22 ± 18.46
Baseline medical data	
Baseline EF (%), mean ± SD	33.97 ± 11.98
NYHA class, *n* (%)	
Class I	140 (29.17)
Class II	241 (50.21)
Class III	94 (19.58)
Class IV	3 (0.63)
Indication for implant, *n* (%)	
Primary prevention	296 (61.67)
Secondary prevention	184 (38.33)
VF	112 (60.87)
VT	72 (39.13)
Medical history, *n* (%)	
Ischemic cardiomyopathy	366 (76.3)
Non-ischemic cardiomyopathy	105 (21.8)
Hypertrophic cardiomyopathy	9 (1.9)
Atrial arrhythmia, *n* (%)	
Atrial fibrillation	76 (15.83)
Atrial flutter	14(2.92)
Ventricular arrhythmia, *n* (%)	
Premature ventricular contractions	27 (5.63)
Non-sustained ventricular tachycardia	24 (88.89)
Ventricular bigeminy	2 (7.41)
Ventricular trigeminy	1 (3.70)
Monomorphic ventricular tachycardia	107 (22.29)
Ventricular fibrillation	79 (16.46)
Polymorphic ventricular tachycardia	10 (2.08)
Other	23 (4.79)
Relevant comorbidities, *n* (%)	
Chronic obstructive pulmonary disease	42 (8.75)
Neurovascular (TIA or CVA)	24 (5.00)
Other	204 (42.50)
Baseline pharmacological treatment, *n* (%)	
ACE inhibitors	312 (65.00)
ARBs	74 (15.42)
Adrenergic block	1 (0.21)
Aldosterone antagonists	180 (37.50)
Antiplatelet medication	343 (71.46)
Anti-arrhythmic	98 (20.42)
Anticoagulants	125 (26.04)
Beta-blockers	422 (87.92)
Calcium channel blockers	56 (11.67)
Cardiac glycosides	16 (3.33)
Diuretics	301 (62.71)
Nitrates	45 (9.38)
Statins	358 (74.58)
Other	97 (20.21)

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; ARVD, arrhythmogenic right ventricle dysplasia; CVA, cerebrovascular accident; EF, ejection fraction; SD, standard deviation; TIA, transient ischemic attack; VF, ventricular fibrillation; VT, ventricular tachycardia.

3.2 | *Follow-up events*

Out of the 480 followed subjects, 26 (5.4%) died during the study. Nine deaths (34.6%) were related to cardiac causes, the remaining deaths were non-cardiac ($n = 6$; 23%), or the cause of death remained unknown ($n = 11$; 42,3%). There were 235 all-cause hospitalizations that occurred in 140 subjects during the follow-up. The mean duration of all-cause hospitalization was 9.29 ± 15.90 days. Only 11 hospitalizations in 9 subjects occurred due to ICD shocks. The mean duration of hospitalization due to this cause was 4.18 ± 2.99 days.

3.3 | *ICD-documented ventricular tachyarrhythmias*

In total, 245 ICD therapies (appropriate and inappropriate) in 117 subjects (24%) were observed, as illustrated in the Graphical Abstract. Further analysis led to the exclusion of 80 events due to missing EGM data ($n = 70$), occurrence of event prior to enrollment ($n = 2$), or inappropriate therapy ($n = 8$). Out of 165 confirmed VT/VF episodes with complete EGM data, 93 (58%) episodes in 69 subjects were preceded by significant ST-segment changes. Further 22 events were excluded due to known, non-ischemic causes of ST changes (aberrant conduction ($n = 16$), or another ventricular arrhythmia episode within 24 h prior to analyzed event ($n = 6$)). Ultimately, 71 ventricular events (43% of confirmed VT/VF episodes) in 56 subjects (12% of the study population and 34% of all patients with ventricular arrhythmias) were confirmed as preceded by characteristic ST-segment changes. Out of the 71 episodes, 54 (76%) were monomorphic VTs, while the remaining 17 episodes (24%) were polymorphic VTs or VF.

3.4 | *Model predicting ST-segment changes*

None of the analyzed demographic and clinical factors proved to be associated with greater odds of presenting with ST-segment changes prior to VT/VF episode (Table 4).

TABLE 4 Odds ratio of ST-segment change detection prior to VT/VF episode in relation to analyzed medical factors

Analyzed factor	*p*-value	Odds ratio	Lower 95% confidence limit for odds ratio	Upper 95% confidence limit for odds ratio
No anti-arrhythmic medication	.8372	1.098	0.449	2.689
No beta-blockers	.2735	1.856	0.614	5.617
No statins	.4740	0.682	0.240	1.943
Non-ischemic cardiomyopathy	.4320	0.655	0.229	1.880
Ischemic cardiomyopathy	.1891	0.490	0.169	1.420
Monomorphic ventricular tachycardia	.8737	0.935	0.407	2.145

Demographic and medical characteristics of patients with arrhythmias preceded by ST-segment changes and those with no ST-segment changes prior to arrhythmic events were not significantly different, as summarized in Table 5.

3.5 | *Relation between ST-segment changes and confirmed ischemia*

Only 6 episodes of the acute coronary syndrome were observed in the study population during the follow-up: 2 myocardial infarctions with ST-segment elevation myocardial infarction (STEMI) and 4 episodes of unstable angina. Five patients underwent coronary revascularization: 4 subjects by percutaneous coronary intervention and 1 by coronary artery bypass grafting.

A total of 230 VT/VF episodes in 112 subjects were classified as requiring a DSE test according to study protocol, including 71 events with confirmed significant ST-segment changes. Unfortunately, only 15 DSE tests in 14 subjects were performed. The results of DSE tests are summarized in Table 6.

4 | DISCUSSION

To our knowledge, this is the first study designed not only to investigate the possibility of detection of EGM ST-segment changes related to ischemia but also to assess the prevalence of ischemic changes as recorded by ICD ST deviations over the 24 h preceding malignant ventricular arrhythmias treated by ICD, and to define their temporal relationship to these events. The principal finding of the study is that a relatively high number of ventricular events (43%) were preceded by significant ST-segment changes. Most of these episodes were monomorphic VTs ($n = 54$; 76%).

The concept of analyzing ST-segment changes in EGMs in order to diagnose ischemic events was previously investigated both in animals and in humans. Early studies included transcutaneous insertion of an electrode to the right ventricle in pigs or in human patients undergoing transcutaneous coronary angioplasty to record ST-segment changes during balloon inflation in the coronary arteries (Fischell et al., 2005; Mendenhall et al., 2012; Williams et al., 2008). Intracardiac electrograms proved to be more sensitive in detecting ST-segment changes than surface ECG (Fischell et al., 2005; Siegel et al., 1982; Theres et al., 2002). The next stage of studies included implantation of dedicated devices in order to continuously monitor ST segment, first in animals, then in human patients (Fischell et al., 2006, 2010). The first human study by Fischell et al. reported 4 cases (among 37 observed patients) in which ST-segment changes related to coronary artery occlusion and/or ruptured arteriosclerotic plaque were detected by an implantable intracardiac ischemia detecting device. The system used in this study showed promising sensitivity and specificity; however, it did not have a functionality of cardioversion and/or defibrillation. The reported median time from ST-segment alarm to hospital medical contact was as short as 19.5 minutes (Fischell et al., 2010). All those facts led to the development of ICDs featuring the ability of constant beat-to-beat monitoring of the intracardiac ST segment.

In this study, a total of 245 ICD interventions (appropriate and inappropriate) in 117 subjects (24%) were observed during a mean follow-up time of 15 ± 5 months. This incidence is comparable with the rate described in real-life ICD patients (Lelakowski et al., 2012; Proclemer et al., 2013; Wasiak et al., 2020; Wilkoff et al., 2004).

The majority of episodes with significant ST-segment changes were monomorphic VTs, that is, 54 episodes (76%). However, in groups of patients with arrhythmic episodes preceded and not preceded by ST-segment changes, the proportion of VTs was almost the same (51/71 vs 52/72, respectively). Interestingly, ST-segment changes prior to VT/VF events were present with comparable frequency in patients with ischemic cardiomyopathy and in those with non-ischemic cause of heart failure (but at the risk of developing CAD in investigators' opinion) (Table 5). The mechanism in which VT occurs in survivors of myocardial infarction is well established to be related to re-entry circuits involving scar tissue (Bolli, 1986). In some patients after myocardial infarction, the development of new ongoing ischemia can create a zone of slow conduction or block that, together with preexisting scar, can be a trigger for VT. In this setting, the ST-segment changes characteristic for ischemia can precede VT episodes. Occurrence of monomorphic VT during early phase (72 h) of the first myocardial infarction is also possible, although it is reported to be rare, not exceeding 3% of cases, according to data from a study by Hatzinikolaou-Kotsakou et al. (2007). There are many suggested mechanisms of VT induction during an early phase of the first acute ischemic event (with no scar-related substrate). Most studies highlight a possible role of intramural re-entry or triggered

TABLE 5 Comparison between demographic and medical characteristics of patients with and without ST-segment changes prior to arrhythmic events

Parameter	ST changes ($N = 56$)	No ST changes ($N = 50$)
Age, mean ± SD	63.5 ± 13.3	65.8 ± 11
Gender—male, *n* (%)	50 (89.3)	41 (82)
Height (cm), mean ± SD	169.6 ± 8.4	170.6 ± 7.7
Weight (kg), mean ± SD	74.9 ± 16.3	82.8 ± 22.4
Baseline medical data		
LV ejection fraction (%), mean ± SD	34.3 ± 13.1	33.2 ± 12.4
NYHA class, *n* (%)		
Class I	18 (32.1)	13 (26.0)
Class II	26 (46.4)	25 (50.0)
Class III	11 (19.6)	11 (22.0)
Class IV	0 (0)	1 (2.0)
Indication for implant, *n* (%)		
Primary prevention	23 (41.1)	26 (52)
Secondary prevention	33 (58.9)	24 (48)
Etiology, *n* (%)		
Ischemic cardiomyopathy	33 (58.9)	33 (66)
Non-ischemic cardiomyopathy	18 (32.1)	18(28)
Hypertrophic cardiomyopathy	2 (3.6)	1 (2)
Hypertrophic cardiomyopathy	2 (3.6)	1 (2)
History of atrial arrhythmia, *n* (%)		
Paroxysmal atrial fibrillation	9 (16.1)	9 (18)
Paroxysmal atrial flutter	2 (3.6)	1 (2)
History of ventricular arrhythmia, *n* (%)		
Premature ventricular contractions	5 (8.9)	6 (12)
Monomorphic ventricular tachycardia	24 (43.2)	21 (42)
Ventricular fibrillation	11 (19.6)	7 (14)
Polymorphic ventricular tachycardia	0 (0)	2 (4)
Other ventricular arrhythmia	1 (1.8)	3 (6)
Relevant comorbidities, *n* (%)		
Chronic obstructive pulmonary disease	5 (8.9)	7 (14)
Neurovascular	2 (3.6)	0 (0)
Previous TIA	1 (50)	0 (0)
Previous CVA	1 (50)	0 (0)
Other comorbidities	26 (46.8)	20 (40)
Baseline pharmacological treatment, *n* (%)		
ACE inhibitors	30 (53.6)	25 (50)
Aldosterone antagonists	19 (33.9)	15 (30)
Cardiac glycosides	1 (1.8)	4 (8)
Anticoagulants	14 (25)	14 (28)
Beta-blockers	44 (78.6)	43 (86)
ARBs	8 (14.3)	11 (22)
Nitrates	3 (5.4)	5 (10)
Adrenergic block	0 (0)	1 (2)
Anti-arrhythmic	22 (39.3)	13 (26)
Statins	34 (60.7)	31 (62)
Calcium channel blockers	4 (7.1)	4 (8)
Diuretics	34 (60.7)	30 (60)
Antiplatelets	32 (57.1)	36 (72)
Other medications	14 (25)	10 (20)

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockers; ARVD, arrhythmogenic right ventricle dysplasia; CVA, cerebrovascular accident; EF, ejection fraction; SD, standard deviation; TIA, transient ischemic attack; VF, ventricular fibrillation; VT, ventricular tachycardia.

activity related to the high sympathetic drive or mechanical stretching (Gantenberg & Hageman, 1992; Pogwizd & Corr, 1987).

All the analyzed demographic and clinical factors fail to prove to be associated with greater odds of presenting with ST-segment changes prior to VT/VF episode. Also, the use of beta-blockers, well known for their anti-ischemic activity, did not have an impact on reducing odds of ST-segment changes preceding arrhythmic event (Dézsi & Szentes, 2017).

Despite the study protocol, only 15 DSE tests (in 14 subjects) were performed. Moreover, only 6 episodes of acute coronary syndrome were observed in the study population during follow-up: 2 STEMI and 4 episodes of unstable angina. This resulted in a lack of data to confirm a likely relation of ST-segment changes with ongoing ischemia, which is the most important limitation of our study. Predictive value of ST-segment changes detected by ICD was investigated by Watanabe et al. (2018). In this study, 173 patients after implantation of ICD with the ST-segment monitoring function were followed for a mean of 23.3 ± 7.7 months. Significant ST changes were observed in 15 patients (8.7%), most of which were asymptomatic. Patients were tested for coronary artery stenosis using coronary angiography and/or for ischemia with myocardial perfusion imaging. The sensitivity, specificity, and negative predictive values of the ST monitoring feature to detect ischemia were 75.0%, 72.5%, and 93.5%, respectively, and to predict stenosis were 76.9%, 83.5%, and 97.5%, respectively.

The prevalence of the significant ST-segment changes prior to VT/VF episodes in our study was relatively high (43% of all

TABLE 6 Outcomes of the dobutamine stress echocardiographies

Reason for stopping the test, *n* (%)	
Target heart rate reached	6 (40)
Achieving dobutamine maximum dose	3 (20)
Ventricular arrhythmias (PVCs, non-sustained VT)	3 (20)
Left ventricle thrombus on baseline images	1 (6.67)
Data missing	1 (6.67)
Stress test endpoints, *n* (%)	
Target heart rate was achieved	10 (66.67)
New wall-motion abnormalities detected	1 (6.67)
Worsening wall-motion abnormalities detected	3 (20.00)
Significant arrhythmias occurred	3 (20.00)
Hypotension occurred	0 (0.00)
Severe hypertension occurred	0 (0.00)
Intolerable symptoms suffered by patient	1 (6.67)

Abbreviations: PVC, premature ventricular contractions; VT, ventricular tachycardia.

confirmed VT/VF episodes). Nevertheless, the predictive value of the ST monitoring feature in identifying pending arrhythmic events has to be established yet.

The study has an important limitation, which is a lack of information whether conversion of arrhythmia by device was similarly effective in both groups. Such analysis could shed light on a possible mechanism of analyzed ventricular arrhythmias.

5 | CONCLUSIONS

In this exploratory study, ST-segment changes preceding VT/VF episodes, likely representative of ischemic events, were observed in more than 1 of 3 of all patients with VT/VF episodes and in 12% of all the patients enrolled in the study. ST-segment changes were observed with similar frequency in patients with known coronary artery disease and those with a non-ischemic cause of heart failure.

ACKNOWLEDGMENT

We gratefully acknowledge an effort of all inSighT trial investigators from all participating centers.

CONFLICT OF INTEREST

Jerzy Krzysztof Wranicz has received lecturer fees from St. Jude Medical/Abbott. Dirk Bastian has received lecturer fees from St. Jude Medical/Abbott and Medtronic. Christof Kolb has received travel support and lecture honoraria from Abbott, Biotronik, Boston Scientific, Bristol Myer Squibb, Medtronic, MicroPort, and Novartis; study compensation to the employer from Abbott, Biotronik, Boston Scientific, MicroPort, and ResMed Foundation; and advisory board fees from MicroPort. Edgar Zitron has received lecturer fees from St. Jude Medical/Abbott and Medtronic. Krzysztof Kaczmarek has received travel grants and lecturer fees from St. Jude Medical/Abbott. Michał Kałowski, Aparna Jaswal, and Iwona Cygankiewicz declared no conflict of interest.

ORCID

Krzysztof Kaczmarek https://orcid.org/0000-0003-1356-5249

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How to cite this article: Wranicz, J. K., Kałowski, M., Bastian, D., Jaswal, A., Kolb, C., Zitron, E., Cygankiewicz, I., & Kaczmarek, K. (2022). Clinical valuation of ST changes in a group of patients with ventricular arrhythmias: The inSighT Study. *Annals of Noninvasive Electrocardiology*, 27, e12914. https://doi.org/10.1111/anec.12914