Original research

Frontal T-wave axis deviation and risk of sudden cardiac death in coronary syndromes

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ABSTRACT

Background Frontal T-wave axis (TA) and QRS–T angle (QRSTA) are automated ECG measures that reflect depolarisation–repolarisation abnormalities—and have been linked to mortality. We tested whether increasing deviation in TA/QRSTA predicts adjudicated sudden cardiac death (SCD) events in patients undergoing coronary angiography—elective (chronic coronary syndrome) and acute (acute coronary syndrome)—and whether signals differ when ECGs are recorded post-acute.

Methods Retrospective consecutive cohort at Tampere University Hospital, 2007–2018. Exposures were automatically measured frontal TA and QRSTA (Marquette 12SL). Primary endpoint was SCD event (true SCD, resuscitated SCA or implantable cardioverter defibrillator-terminated VF/very fast ventricular tachycardia) adjudicated to guideline definitions, with Fine–Gray competing-risk models adjusted for clinical covariates. Follow-up through 31 December 2022.

Results We analysed 18 828 patients (elective n=10 303; acute n=8525; median follow-up 9.0 and 7.8 years). In the elective cohort, SCD hazard increased monotonically with TA/QRSTA deviation; severely abnormal TA (<−75° or >165°) HR 4.06 (95% CI 2.94 to 5.61) and severely abnormal QRSTA (≥151°) HR 3.16 (2.35–4.24). Associations persisted after excluding left ventricular ejection fraction (LVEF) ≤35%. In the acute cohort, effects were smaller and non-monotonic (severely abnormal TA HR 2.01 (1.42–2.86); QRSTA HR 1.73 (1.24–2.39)). In a sensitivity cohort with ECGs 30–365 days postangiography (acute 30, n=5529), the TA–SCD association resembled the elective setting (severely abnormal TA HR 2.69 (1.70–4.24)); QRSTA was not significant.

Conclusion Severity of frontal TA deviation is a robust, graded marker of SCD risk in angiography patients, strongest in elective and postacute settings; QRSTA is supportive but weaker. These automated, low-cost metrics could augment SCD risk stratification beyond LVEF, warranting external validation and clinical utility testing.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ In community cohorts, abnormal frontal T-wave axis (TA) and QRS–T angle (TA/QRSTA) relate to higher sudden cardiac death (SCD) risk, but clinical cohorts with angiography and adjudicated SCD are scarce.

WHAT THIS STUDY ADDS

⇒ In 18 828 angiography patients, SCD hazard rose stepwise with worsening TA; the effect was weaker/non-monotonic during acute coronary syndrome but re-emerged when ECGs were recorded 30–365 days later. Severely abnormal TA remained predictive after excluding left ventricular ejection fraction ≤35%.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Frontal TA—an automated ECG output—could refine SCD risk assessment in coronary artery disease, particularly outside the acute phase; prospective validation and incremental value analyses are the logical next step.

INTRODUCTION

Sudden cardiac death (SCD), by definition, occurs within 1 hour of symptom onset. Most often, the underlying cardiac cause is coronary artery disease (CAD).[1] Despite remarkable advancements in the diagnosis and treatment of chronic and acute coronary syndrome (CCS and ACS) in recent years, CAD remains one of the leading causes of death worldwide, with SCD estimated to be responsible for up to half of these deaths.[2 3] The annual incidence of SCD in patients who have undergone previous invasive evaluation for ACS and are on modern medication is roughly 0.5%.[4 5]

The primary prevention of SCD in high-risk patients is accomplished with an implantable cardioverter defibrillator (ICD), yet to date, it is recommended mainly for patients with severely reduced left ventricular ejection fraction (LVEF).[1 6] However, prior studies indicate that only approximately 30% of SCD victims have a severely reduced LVEF,[7 8] highlighting the need for additional markers to identify patients at a high risk of SCD. It is estimated that up to 80% of SCDs are caused by CAD,[9] making patients with CAD an optimal group for risk stratification.

The frontal T-wave axis (TA) and frontal QRS-T angle (the angle between the frontal QRS axis and frontal TA, QRSTA) are both easily assessed from the conventional ECG and are usually included in the automated reports of ECG devices. A normal frontal TA ranges from 15° to 75°, and a normal

frontal QRSTA is defined as an angle of <73° for men and <67° for women, depending on the study.[10] Deviation in their orientations is known to indicate repolarisation (and with QRSTA also depolarisation) abnormalities that are thought to be a result of multiple pathological processes.[10] The association of a deviation in the frontal TA and QRSTA with all-cause and cardiac mortality has been shown previously.[10 11] The frontal TA and QRSTA have been associated with SCD in general population cohorts.[12 13]

The aim of this study was to explore the association between SCD event and the degree to which the frontal TA or QRSTA deviates from normal values in patients undergoing coronary angiography for suspected or known CCS and for ACS. Furthermore, our aim was to test whether TA and QRSTA associate with SCD event similarly in patients with suspected or known CCS and in ACS patients.

METHODS

Study populations

The study population includes retrospective data collected from 18 828 consecutive patients undergoing invasive coronary angiography for known or suspected CCS (elective cohort) or for ACS (acute cohort), between 1 January 2007 and 31 December 2018, at Tays Heart Hospital, which is part of Tampere University Hospital. Tays Heart Hospital is a tertiary care centre for cardiology in the Pirkanmaa region, serving a population of over 500 000 in southern Finland.[14]

Patients included in the study had baseline ECG within 0–30 days of angiography; acute 30 cohort 30–365 days postangiography; rationale: transient ACS changes. If a patient underwent multiple angiographies during the study period, the first angiography was selected as the index event. The classification of CCS and ACS followed the guidelines established by the European Society of Cardiology (ESC) and the American College of Cardiology (ACC).[15–18]

Data collection

Comprehensive medical histories of the patients were obtained from the Mass Data in Detection and Prevention of Serious Adverse Events in Cardiovascular Disease (MADDEC) database. MADDEC collects retrospective data from multiple data sources, including national registries, local hospital health records and biometric data, focusing on cardiological patients. Data on invasive cardiological procedures are prospectively recorded in the registry by the treating cardiologist. A comprehensive description of the MADDEC study is provided elsewhere.[14]

Main exposure variables

The study populations were divided into subgroups based on the deviation of the TA or QRSTA from the first postangiography ECG. The TA subgroups consisted of a normal TA (15° to 75°), borderline TA deviation (−15° to 14° and 76° to 105°), mildly abnormal TA deviation (−45° to −16° and 106° to 135°), moderately abnormal TA deviation (−75° to −46° and 136° to 165°) and severely abnormal TA deviation (< −75° and >165°).[10] For the QRSTA, the subgroups were as follows: a normal QRSTA (< 73°), borderline QRSTA deviation (73° to 100°), mildly abnormal QRSTA deviation (101° to 125°), moderately abnormal QRSTA deviation (126° to 150°) and severely abnormal QRSTA deviation (≥ 151°) (table 1).[10] The division was made to achieve an equal representation of the severity of the TA and QRSTA deviations in subgroups while maintaining enough patients within each subgroup for statistical analysis.

Table 1 The subgroups of T-wave axes and QRS-T angles

	T-wave axis	QRS-T -angle
Normal	15° to 75°	<73°
Borderline deviation	−15° to 14° and 76° to 105°	73° to 100°
Mildly abnormal deviation	−45° to −16° and 106° to 135°	101° to 125°
Moderately abnormal deviation	−75° to −46° and 136° to 165°	126° to 150°
Severely abnormal deviation	<−75° and >165°	≥151°

The same division was used for both males and females. The TA and QRSTA are both standard computerised ECG measurements that were measured automatically by the Marquette 12SL ECG analysis software (GE HealthCare) to avoid bias from subjective measurements. The frontal QRSTA has not shown significant differences when measured manually or automatically.[19]

Follow-up and endpoints

SCD event was used as the main endpoint in this study. The definition is in accordance with the guidelines set by the American Heart Association, the ACC, the Heart Rhythm Society and the ESC.[6 20]

The definition of true SCD was an unanticipated and sudden natural death from a cardiac cause that occurred within an hour of symptom onset or in a patient who was found dead within 24 hours of being asymptomatic. SCD event, in addition, included patients who suffered a sudden

cardiac arrest (SCA) but survived due to successful resuscitation and otherwise met the criteria for SCD as well as patients who had VF or very fast ventricular tachycardia (≥230 bpm), leading to a haemodynamic collapse, which was successfully aborted by an ICD therapy. Deaths were not classified as SCD if the circumstances surrounding the event or the timeline were unclear of imprecise. Data for endpoint adjudication were obtained from written medical records, death certificates and Statistics Finland. Finnish law mandates that death certificates must include all significant events and circumstances, leading to the death and that a medico-legal autopsy be performed if the fatality was immediate or unexpected[21] (around 50% of the SCD and SCA patients autopsied in the acute cohort), thus providing comprehensive information about the event. Each suspected SCD case was individually evaluated using previously established methods to determine the likelihood of SCD as the underlying cause. In patients with an ICD, device records—including EGM, therapy type and event descriptions—were reviewed to confirm life-threatening arrhythmias with haemodynamic collapse. A more detailed description of the endpoint adjudication has been described previously by Koivunen *et al*.[4]

The follow-up began from the first elective or acute angiography and carried on until the endpoint was reached, until the patient died due to another cause, or until 31 December 2022.

Statistical analysis

Statistical analyses were performed with IBM SPSS Statistics V.27 and Rstudios V.2023.12.1+402, along with the packages cmprsk, meta, survival, rms, ggplot2 and dplyr. A p value <0.05 was considered significant, and all the tests were two-tailed.

In the baseline characteristics, continuous variables were presented as mean and SD if normally distributed, and as median and IQR if non-normally distributed. Normality was assessed visually. Comparisons of the baseline variables were conducted with the χ^2 test for categorical variables and the t-test for continuous variables with a normal distribution. Associations between

TA/QRSTA and baseline variables were assessed using logistic regression models, adjusted for the patient's age and sex.

Regression models were applied to the elective and acute cohort, and additionally to the 'acute 30 cohort', which included ECG data recorded 30–365 days after the angiography in the acute cohort, based on the assumption that the acute phase of ACS could result in transient TA and QRSTA changes. The competing-risk subdistribution hazard model (Fine-Gray) was used to analyse the association between a deviation in the TA or QRSTA and the incidence of SCD event, with death due to another cause considered a competing event.[22] These analyses were adjusted for age, sex and common cardiovascular risk factors. Covariates with missing data were handled using complete-case analysis. The proportion of missing values was low and occurred only in three covariates across the elective, acute and acute 30 cohorts: hypertension (0.13%, 0.81%, 0.89%), diabetes (0.25%, 0.65%, 0.69%) and history of myocardial infarction (0.20%, 0.08%, 0.07%). The adjusting variables were selected from available data based on their previously established significant associations with the risk of serious adverse cardiovascular events. All adjusting variables—except for hypertension and age—were also associated significantly with the hazard of SCD event in multivariable models ($p<0.05$). The proportional hazards assumption was evaluated using the Schoenfeld residual test ($p<0.05$, except for hypertension and age) based on Cox regression models. Multicollinearity in multivariable models was assessed using variance inflation factors (all<2).

The Cox regression model was used for a restricted cubic splines model to illustrate the continuous, unadjusted, association of TA and QRSTA deviation with SCD event. Five splines were used to represent the non-linear association comprehensively throughout the continuum without overfitting the model. An individual patient data meta-analysis of both cohorts was conducted using a fixed-effects model, and additionally a random-effects model was used when heterogeneity was high ($I^2>50\%$).

RESULTS

Before any exclusions, there were 22 497 patients in the MADDEC registry undergoing their first coronary angiography between the years 2007 and 2018. After excluding patients with incomplete ECG data (n=1076), ventricular pacemaker rhythm, pre-excitation and conduction disorders (left or right bundle branch block or a non-specific intraventricular conduction delay) (n=3139), a total of 18 282 patients were included in the analyses. No patients were lost to follow-up. The general characteristics of both study populations are shown in table 2. Of the 10 303 patients in the elective cohort and of the 8525 patients in the acute cohort, 55.3% (n=5694) and 88.9% (n=7575), respectively, were diagnosed with CAD, respectively. Overall, 46.7% (n=4807) of the elective cohort and 70.7% (n=6025) of the acute cohort had borderline or abnormal TA values, while 26.1% (n=2691) of the elective cohort and 41.6% (n=3544) of the acute cohort had borderline or abnormal QRSTA values. The distributions of both TA and QRSTA values in the cohorts are shown in figure 1A–C. Patients with borderline or abnormal TA or QRSTA values in both study populations tended to be older and had a higher prevalence of multiple cardiovascular risk factors.

Furthermore, angiographic data revealed that patients with borderline or abnormal TA or QRSTA values had more severe CAD, as evidenced by a significantly higher prevalence of three-vessel disease compared with those with normal TA or QRSTA values (online supplemental table 1). In addition, when comparing other ECG characteristics between the normal-value subgroup and the severely abnormal-value subgroups in both cohorts, the severely abnormal TA and QRSTA subgroups had significantly higher mean of ventricular rate, QRS duration and corrected QT interval in both cohorts (online supplemental table 2).

Table 2 General characteristics of the two study populations of patients undergoing angiography for known or suspected chronic coronary syndrome (elective cohort) or for acute coronary syndrome (acute cohort)

	Elective cohort n=10 303	Acute cohort n=8525
Frontal T-wave axis*	38° (12° to 65°)	44° (–20° to 94°)
Frontal QRS-T angle*	34° (14° to 75°)	58° (25° to 110°)
Patient age (years)†	66.5 (10.7)	67.5 (11.8)
Patient sex (men)	59.2% (6103)	66.5% (5667)
Prevalent diabetes	26.9% (2774)	24.0% (2044)
Prevalent hypertension	67.8% (6988)	58.7% (5009)
Prevalent dyslipidaemia	72.1% (7430)	56.3% (4803)
Previous acute or chronic kidney disease	12.5% (1289)	11.2% (954)
Prevalent valvular heart disease	18.7% (1924)	6.2% (530)
Prevalent left ventricular hypertrophy ‡	11.0% (1128)	7.0% (596)
Atrial fibrillation or atrial flutter §	12.2% (1252)	7.9% (676)
Prevalent peripheral arterial disease	4.9% (504)	6.6% (564)
Patient smoking (active)	25.2% (2600)	20.2% (1726)
Previous stroke	0.5% (46)	7.5% (640)
Previous myocardial infarction	10.2% (1055)	14.9% (1267)
Previous PCI	8.6% (882)	9.8% (838)
Previous CABG	8.3% (859)	6.7% (567)
Acute coronary syndrome type		
UAP	–	17.3% (1476)
NSTEMI	–	45.0% (3837)
STEMI	–	37.7% (3214)
Treatment		
Conservative	68.1% (7014)	21.9% (1867)
PCI	20.6% (2121)	66.9% (5705)
CABG	11.3% (1168)	11.2% (955)
LVEF (%)†	56.9 (12.4)	51.8 (11.5)
LVEF≤35%	7.7% (796)	9.7% (828)
Estimated GFR†	82.7 (22.9)	80.1 (21.1)
Number of coronary arteries with stenosis ¶		
0	44.7% (4609)	11.2% (952)
1	22.6% (2325)	40.1% (3419)
2	17.9% (1839)	26.6% (2269)
3	14.9% (1530)	22.1% (1887)

*Median (IQR).
†Mean (SD).
‡According to ECG.
§During hospitalisation.
¶>50% narrowing compared with the diameter of the normal adjacent segment of the artery.
CABG, coronary bypass graft surgery; GFR, glomerular filtration rate; LVEF, left ventricular ejection fraction; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; UAP, unstable angina pectoris.

HR for death associated with abnormal TA and QRSTA values

During the follow-up, which lasted a median of 9.0 (IQR 5.8–12.2) years in the elective cohort and 7.8 (IQR 4.9–11.2)

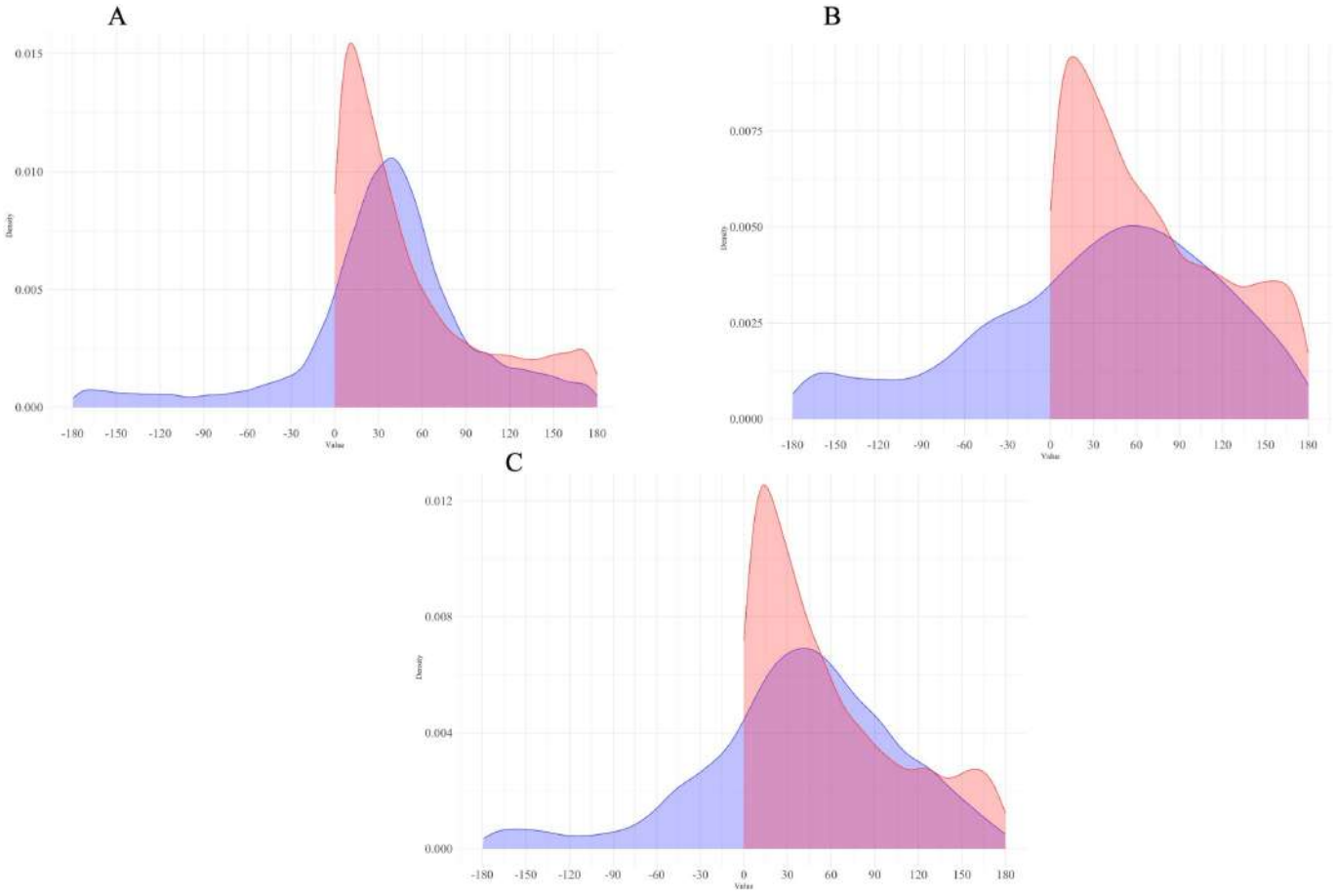


Figure 1 Density plot for the T-wave axis (blue) and QRS-T-angle (red) in the elective cohort (A), the acute cohort (B) and the acute 30 cohort (C).

years in the acute cohort, 3299 (32.0%) patients in the elective cohort and 3107 (36.4%) patients in the acute cohort died. Of these fatalities, 11.0% (n=362) of the deaths occurring in the elective cohort and 13.2% (n=411) of those occurring in the acute cohort were regarded as SCD event (figures 2 and 3). Of these SCD events, 76% and 82%, respectively, were considered true SCDs, while the remainder were SCAs surviving due to either resuscitation (otherwise meeting the criteria for SCD) or lethal ventricular arrhythmia aborted by successful ICD therapy.

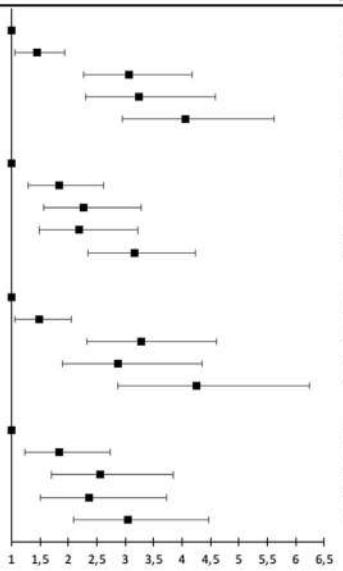
In the elective cohort, both TA and QRSTA values were associated with an increased hazard of SCD event. The HRs for the TA and QRSTA subgroups are presented in figure 2, and the HRs across the continuum of TA and QRSTA values are shown in online supplemental figure 1. The highest HR was observed in patients with a severely abnormal TA (HR 4.06 (95% CI 2.94 to 5.61), p<0.001), with the association remaining significant even when excluding patients with an LVEF of ≤35% (HR 4.25 (95% CI 2.88 to 6.25), p<0.001). The highest corresponding HR associated with the QRSTA was observed in patients with severely abnormal QRSTA values (HR 3.16 (95% CI 2.35 to 4.24), p<0.001).

In the acute cohort, the highest HR was observed among patients with a severely abnormal TA (HR 2.01 (95% CI 1.42 to 2.86), p<0.001), remaining significant even after the exclusion of patients with an LVEF of ≤35% (HR 1.66 (95% CI 1.08 to 2.54), p=0.020) (figure 3 and online supplemental figure 2). Deviation in the frontal QRSTA also showed the highest HR in the severely abnormal subgroup (HR 1.73 (95% CI 1.24 to 2.39) p=0.001), which was equal to the borderline subgroup (HR 1.73 (95% CI 1.31 to 2.29) p<0.001). As regards the TA and especially the QRSTA values, the HRs did not increase proportionally to the severity of the deviation in this context (acute cohort).

A sensitivity analysis was performed using data from ECGs recorded 30–365 days after the angiography in the acute cohort (the 'acute 30 cohort', n=5529), as we speculated that the acute phase of the ACS might cause reversible TA and QRSTA changes (figure 1). In total, the TAs of 677 patients and QRSTAs of 601 patients changed from abnormal to normal. As shown in figure 4 and online supplemental figure 3, the HRs in the acute 30 cohort aligned more closely with that of the elective cohort, with the highest HR observed in patients with a severely abnormal TA (HR 2.69 (95% CI 1.70 to 4.24), p<0.001). This association remained significant even when excluding patients with an LVEF of ≤35% (HR 2.76 (95% CI 1.61 to 4.74), p<0.001). A severely abnormal QRSTA was not associated with SCD event in the acute 30 cohort (HR 1.44 (95% CI 0.91 to 2.28) p=0.118).

The individual patient data meta-analyses of the elective and acute cohorts as well as in the elective and acute 30 cohorts are shown in figure 5. Despite the significant heterogeneity observed between the elective cohort and the acute cohort, the HRs remained significant in all subgroups, with the highest HR observed in patients with a severely abnormal TA (HR 2.95 (95% CI 2.32 to 3.74), p<0.001 with the fixed-effects model). In the individual patient data meta-analysis of the elective cohort and the acute 30 cohort, the HRs remained significant in all subgroups, increasing proportionally to the severity of the deviation (severely abnormal TA: HR 3.54 (95% CI 2.72 to 4.61), p<0.001, with the fixed-effects model). The HR associated

		N of patients	Follow-up time*	Total deaths (%)	SCDs (%)	HR for SCD (95% CI)	P-value
TA	Normal	5 496	9.6 (6.4-12.7)	1371 (24.9%)	106 (1.9%)	1.00	
	Borderline	2 438	9.0 (5.9-12.4)	743 (30.5%)	72 (3.0%)	1.44 (1.06-1.94)	0.018
	Mildly abnormal	962	8.0 (4.9-11.6)	433 (45.0%)	69 (7.2%)	3.07 (2.27-4.17)	<0.001
	Moderately abnormal	625	6.9 (3.8-10.5)	339 (54.2%)	49 (7.8%)	3.25 (2.30-4.59)	<0.001
	Severely abnormal	782	7.0 (4.2-10.4)	413 (52.8%)	66 (8.4%)	4.06 (2.94-5.61)	<0.001
QRSTA	Normal	7 612	9.5 (6.3-12.6)	1935 (25.4%)	178 (2.3%)	1.00	
	Borderline	829	8.0 (5.2-11.4)	374 (45.1%)	43 (5.2%)	1.84 (1.29-2.61)	0.001
	Mildly abnormal	582	7.7 (4.8-10.9)	293 (50.3%)	38 (6.5%)	2.27 (1.57-3.28)	<0.001
	Moderately abnormal	524	6.7 (3.9-10.3)	274 (52.3%)	34 (6.5%)	2.19 (1.49-3.22)	<0.001
	Severely abnormal	756	6.6 (3.7-10.2)	423 (56.0%)	69 (9.1%)	3.16 (2.35-4.24)	<0.001
TA, LVEF >35%	Normal	5 150	9.5 (6.4-12.6)	1250 (24.3%)	86 (1.7%)	1.00	
	Borderline	2 219	9.0 (5.9-12.3)	650 (29.3%)	59 (2.7%)	1.48 (1.06-2.06)	0.022
	Mildly abnormal	772	8.3 (5.1-11.7)	343 (44.4%)	54 (7.0%)	3.27 (2.32-4.60)	<0.001
	Moderately abnormal	484	7.1 (4.1-10.8)	268 (55.4%)	31 (6.4%)	2.87 (1.89-4.36)	<0.001
	Severely abnormal	545	7.1 (4.3-10.6)	284 (52.1%)	44 (8.1%)	4.25 (2.88-6.25)	<0.001
QRSTA, LVEF >35%	Normal	7 097	9.4 (6.3-12.6)	1760 (24.8%)	143 (2.0%)	1.00	
	Borderline	715	8.3 (5.3-11.7)	312 (43.6%)	34 (4.8%)	1.84 (1.24-2.61)	0.002
	Mildly abnormal	466	7.5 (4.9-10.9)	246 (52.8%)	32 (6.9%)	2.56 (1.70-3.84)	<0.001
	Moderately abnormal	382	7.1 (3.3-10.4)	197 (51.6%)	25 (6.5%)	2.37 (1.51-3.72)	<0.001
	Severely abnormal	510	7.0 (3.9-10.6)	280 (54.9%)	40 (7.8%)	3.05 (2.09-4.46)	<0.001



Each subgroup compared to the group with a normal TA or QRSTA

* = Median (IQR)

LVEF = Left ventricular ejection fraction

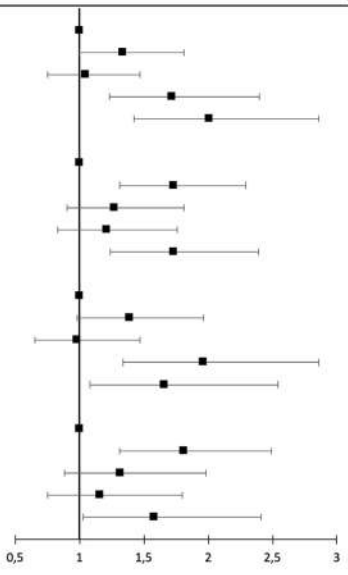
All models are adjusted for age, sex, prevalent hypertension, prevalent diabetes, prevalent statement of LVH on ECG, previous acute or chronic kidney disease, history of stroke or MI, and number of diseased coronary arteries during the angiography.

Figure 2 HRs for SCD estimated with a competing-risk subdistribution hazard model (Fine-Gray) in the elective cohort. LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; QRSTA, QRS–T angle; SCD, sudden cardiac death; TA, frontal T-wave axis.

with a severely abnormal TA remained significant even when including only patients with CAD according to the angiography in both individual patient data meta-analyses.

As in the analyses of individual cohorts, the association between QRSTA and SCD event was less pronounced than it was between the TA and SCD event. The random-effects

		N of patients	Follow-up time*	Total deaths (%)	SCDs (%)	HR for SCD (95% CI)	P-value
TA	Normal	2 500	8.3 (5.4-11.6)	732 (29.3%)	84 (3.4%)	1.00	
	Borderline	2 056	8.0 (5.3-11.3)	703 (34.2%)	100 (4.9%)	1.34 (1.00-1.81)	0.051
	Mildly abnormal	1 603	7.9 (4.8-11.3)	566 (35.3%)	66 (4.1%)	1.05 (0.75-1.47)	0.783
	Moderately abnormal	1 145	7.7 (4.7-11.0)	470 (41.0%)	75 (6.6%)	1.72 (1.23-2.40)	0.001
	Severely abnormal	1 221	6.2 (3.3-10.0)	636 (52.1%)	86 (7.0%)	2.01 (1.42-2.86)	<0.001
QRSTA	Normal	4 981	8.4 (5.5-11.7)	1450 (29.1%)	183 (3.7%)	1.00	
	Borderline	1 103	7.7 (4.7-10.8)	439 (39.8%)	75 (6.8%)	1.73 (1.31-2.29)	<0.001
	Mildly abnormal	819	7.2 (4.3-10.7)	382 (46.6%)	45 (5.5%)	1.27 (0.90-1.81)	0.177
	Moderately abnormal	739	6.5 (3.8-10.1)	355 (48.0%)	43 (5.8%)	1.21 (0.83-1.76)	0.327
	Severely abnormal	883	6.1 (3.0-9.7)	481 (54.5%)	65 (7.4%)	1.73 (1.24-2.39)	0.001
TA, LVEF >35%	Normal	2 148	8.7 (5.8-11.7)	597 (27.8%)	61 (2.8%)	1.00	
	Borderline	1 729	8.3 (5.7-11.5)	556 (32.2%)	74 (4.3%)	1.39 (0.98-1.96)	0.064
	Mildly abnormal	1 334	8.3 (5.3-11.6)	445 (33.4%)	45 (3.4%)	0.98 (0.65-1.47)	0.928
	Moderately abnormal	910	8.1 (5.0-11.1)	363 (39.9%)	58 (6.4%)	1.96 (1.34-2.86)	<0.001
	Severely abnormal	922	7.1 (4.2-10.5)	459 (49.8%)	49 (5.3%)	1.66 (1.08-2.54)	0.020
QRSTA, LVEF >35%	Normal	4 325	8.7 (5.9-11.8)	1213 (28.0%)	137 (3.2%)	1.00	
	Borderline	893	8.1 (5.0-11.0)	336 (37.6%)	55 (6.2%)	1.81 (1.31-2.49)	<0.001
	Mildly abnormal	638	7.6 (4.4-11.1)	294 (46.1%)	32 (5.0%)	1.32 (0.88-1.98)	0.180
	Moderately abnormal	560	7.2 (4.4-10.5)	256 (45.7%)	28 (5.0%)	1.16 (0.75-1.80)	0.509
	Severely abnormal	627	6.7 (3.8-10.0)	321 (51.2%)	35 (5.6%)	1.58 (1.03-2.41)	0.035



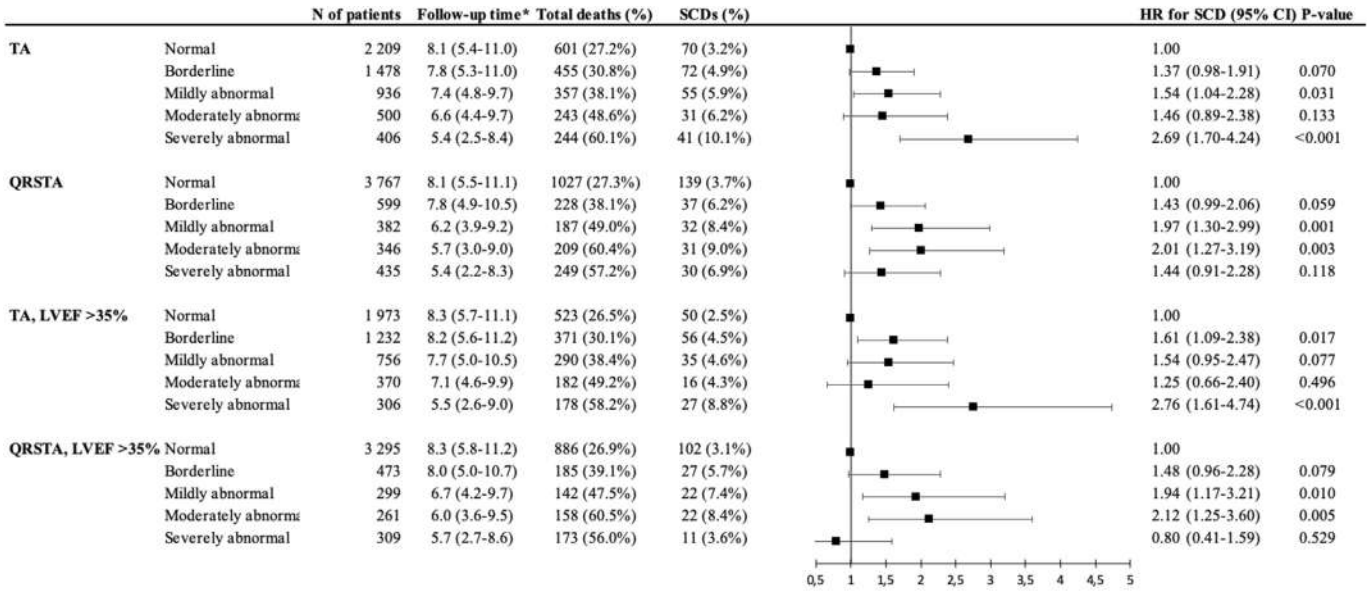
Each subgroup compared to the group with a normal TA or QRSTA

* = Median (IQR)

LVEF = Left ventricular ejection fraction

All models are adjusted for age, sex, prevalent hypertension, prevalent diabetes, prevalent statement of LVH on ECG, previous acute or chronic kidney disease, history of stroke or MI, number of diseased coronary arteries during the angiography, and the type of ACS (only in the ACS data).

Figure 3 HRs for SCD estimated with a competing-risk subdistribution hazard model (Fine-Gray) in the acute cohort. ACS, acute coronary syndrome; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; QRSTA, QRS–T angle; SCD, sudden cardiac death; TA, frontal T-wave axis.



		N of patients	Follow-up time*	Total deaths (%)	SCDs (%)	HR for SCD (95% CI)	P-value
TA	Normal	2 209	8.1 (5.4-11.0)	601 (27.2%)	70 (3.2%)	1.00	
	Borderline	1 478	7.8 (5.3-11.0)	455 (30.8%)	72 (4.9%)	1.37 (0.98-1.91)	0.070
	Mildly abnormal	936	7.4 (4.8-9.7)	357 (38.1%)	55 (5.9%)	1.54 (1.04-2.28)	0.031
	Moderately abnorma	500	6.6 (4.4-9.7)	243 (48.6%)	31 (6.2%)	1.46 (0.89-2.38)	0.133
	Severely abnormal	406	5.4 (2.5-8.4)	244 (60.1%)	41 (10.1%)	2.69 (1.70-4.24)	<0.001
QRSTA	Normal	3 767	8.1 (5.5-11.1)	1027 (27.3%)	139 (3.7%)	1.00	
	Borderline	599	7.8 (4.9-10.5)	228 (38.1%)	37 (6.2%)	1.43 (0.99-2.06)	0.059
	Mildly abnormal	382	6.2 (3.9-9.2)	187 (49.0%)	32 (8.4%)	1.97 (1.30-2.99)	0.001
	Moderately abnorma	346	5.7 (3.0-9.0)	209 (60.4%)	31 (9.0%)	2.01 (1.27-3.19)	0.003
	Severely abnormal	435	5.4 (2.2-8.3)	249 (57.2%)	30 (6.9%)	1.44 (0.91-2.28)	0.118
TA, LVEF >35%	Normal	1 973	8.3 (5.7-11.1)	523 (26.5%)	50 (2.5%)	1.00	
	Borderline	1 232	8.2 (5.6-11.2)	371 (30.1%)	56 (4.5%)	1.61 (1.09-2.38)	0.017
	Mildly abnormal	756	7.7 (5.0-10.5)	290 (38.4%)	35 (4.6%)	1.54 (0.95-2.47)	0.077
	Moderately abnorma	370	7.1 (4.6-9.9)	182 (49.2%)	16 (4.3%)	1.25 (0.66-2.40)	0.496
	Severely abnormal	306	5.5 (2.6-9.0)	178 (58.2%)	27 (8.8%)	2.76 (1.61-4.74)	<0.001
QRSTA, LVEF >35%	Normal	3 295	8.3 (5.8-11.2)	886 (26.9%)	102 (3.1%)	1.00	
	Borderline	473	8.0 (5.0-10.7)	185 (39.1%)	27 (5.7%)	1.48 (0.96-2.28)	0.079
	Mildly abnormal	299	6.7 (4.2-9.7)	142 (47.5%)	22 (7.4%)	1.94 (1.17-3.21)	0.010
	Moderately abnorma	261	6.0 (3.6-9.5)	158 (60.5%)	22 (8.4%)	2.12 (1.25-3.60)	0.005
	Severely abnormal	309	5.7 (2.7-8.6)	173 (56.0%)	11 (3.6%)	0.80 (0.41-1.59)	0.529

Each subgroup compared to the group with a normal TA or QRSTA

* = Median (IQR)

LVEF = Left ventricular ejection fraction

All models are adjusted for age, sex, prevalent hypertension, prevalent diabetes, prevalent statement of LVH on ECG, previous acute or chronic kidney disease, history of stroke or MI, number of diseased coronary arteries during the angiography, and the type of ACS (only in the ACS data).

Figure 4 HRs for SCD estimated with a Fine-Gray subdistribution hazard model in the acute cohort with ECG data recorded 30–365 days after the angiography ('acute 30 cohort'). ACS, acute coronary syndrome; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; QRSTA, QRS–T angle; SCD, sudden cardiac death; TA, frontal T-wave axis.

estimates for models with high heterogeneity are presented in online supplemental figure 4.

DISCUSSION

In cohorts of patients who underwent elective angiography for known or suspected CCS (elective cohort) and of ACS patients diagnosed invasively by means of angiography (acute cohort), both with an up to 16-year follow-up period, a severely abnormal frontal TA (< −75° and >165°) was a strong indicator of SCD event, even after excluding patients with an LVEF of ≤35%. A severely abnormal frontal QRS-T angle (≥151°) was also a strong indicator of SCD event, but to a lesser extent. In the elective cohort, when dividing patients into subgroups based on the severity of the TA deviation, and when inspecting the TA as a continuous variable, a positive correlation was observed between the severity of the deviation and the hazard of SCD event. SCD event included both true fatal SCDs and cases where SCD would have occurred in the absence of successful resuscitation or appropriate ICD therapy.

Deviations in the frontal TA and QRSTA have long been recognised as a marker of repolarisation abnormality (with QRSTA also indicating a depolarisation abnormality) of multifactorial aetiology that results from pathophysiological changes in myocardial ionic channel mechanisms, increased myocardial fibrosis, haemodynamic factors and ischaemia.[23–26] The deviations have been shown to be associated with a higher risk of mortality in multiple general population studies, as well as in STEMI patients.[10 11 27]

Deviations in the frontal TA or QRSTA have been demonstrated to increase the risk of SCD in previous studies.[12 13 28] Interestingly, Aro *et al* noted that the association between a frontal QRSTA deviation and SCD was primarily driven by the frontal TA, as the QRS axis was not independently associated with the risk of SCD.[12] In a study by Chua *et al*, a frontal TA deviation did not significantly increase the risk of SCD, whereas a QRSTA deviation did.[13] Similar findings regarding frontal QRSTA were reported from Garcia *et al* in a cohort of patients with type 2 diabetes.[28] These studies were population-based and had a lower incidence of detected SCDs, they included smaller numbers of patients in the TA or QRSTA deviation subgroups and had only one or two subgroups representing these deviations.[12 13 28] None of the previously mentioned studies had angiographic data or data concerning the LVEF, except for Chua *et al*, who provided LVEF data for approximately half of the study patients.

The strengths of our study lie in the vast cohort of consecutive broad-spectrum patients who were invasively diagnosed via coronary angiography for coronary syndromes and followed for a relatively long, up to 16-year, period, as well as the comprehensive knowledge of the patients' medical information, the precise SCD event descriptions obtained from death certificates and the high rates of medico-legal autopsies (around 50% of the SCD and SCA cases in this study[4]). This is due to Finland's legislation, which mandates a medico-legal autopsy in cases where the death is not caused by a pre-existing known disease, if a physician has not treated the patient during the last illness or if the death was otherwise unexpected.[21]

The limitations of our study include the usage of the automatically measured frontal TA and QRSTA instead of spatial measurements, although a meta-analysis by Zhang *et al* found that the risk ratio of all-cause death was only slightly lower for the frontal QRSTA compared with the spatial QRSTA.[11] Regarding the reference values for the frontal QRSTA and TA, a limitation of our study is that we did not use sex-specific values, which might have affected the results. Furthermore, there is some variability in the reference values of frontal QRSTAs between studies, as previously discussed by Tanriverdi *et al*.[29] The acute 30 cohort

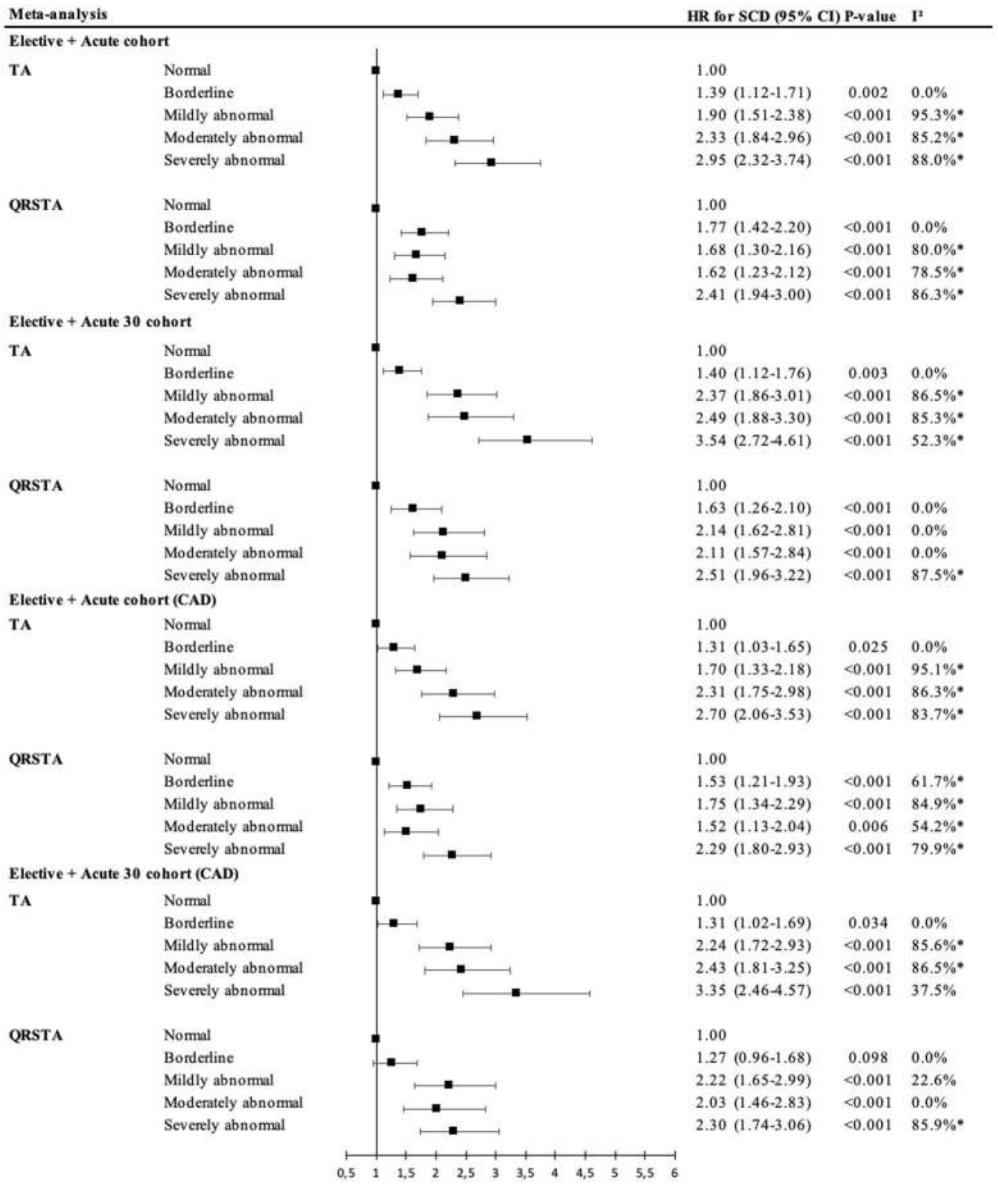


Figure 5 Meta-analyses of the cohorts done with a fixed-effects model. *Additional meta-analysis done with random-effects model in online supplemental figure 4. Acute 30 cohort = acute cohort with the ECG data recorded 30–365 days after the angiography, CAD = only includes patients with CAD according to the angiography. Each subgroup compared to the group with a normal TA or QRSTA. CAD, coronary artery disease; QRSTA, QRS–T angle; TA, frontal T-wave axis.

is subjected to immortal time bias, as it includes only patients with ECG data recorded 30–365 days postangiography, thereby excluding those who suffered an SCD before day 30. However, it reflects a real-world scenario in which the postacute SCD risk is assessed among patients who have survived the prehospital phase, in whom deviations in the frontal TA and QRSTA are not transient, and who would be more likely to benefit from an ICD. Further limitations include potential residual confounding inherent to retrospective studies and the single-centre nature of our data.

In summary, a deviation in the frontal TA in patients undergoing coronary angiography for known or suspected CCS, as well as in invasively diagnosed ACS patients in a stable setting, was associated with a significantly higher HR for SCD event, with the degree of the association depending on the severity of the deviation. This association was also observed among patients with an LVEF of over 35%. For deviation in the frontal QRSTA, effects were smaller and non-monotonic in the acute phase. In situations where these ECG markers are affected by transient causes, such as in ACS, the TA and QRSTA are less robust predictors of SCD. In the future, the frontal TA could be incorporated into risk scores assessing high-risk SCD patients who may benefit from an ICD.

Contributors All authors meet the ICJME authorship criteria. KK is the corresponding author and guarantor and takes responsibility for communication with the journal during the submission and review process. KK: conceptualisation, writing—original draft, review and editing, analysis, visualisation. JR: conceptualisation, writing—review and editing. L-PL: analysis, data curation, writing—review and editing. HP: writing—review and editing. MK: data curation, writing—review and editing. KN: writing—review and editing. JTT: conceptualisation, writing—review and editing, methodology, analysis, supervision. JH: conceptualisation, writing—review and editing, methodology, analysis, supervision.

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Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval Not applicable.

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