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Spatial QRS-T angle predicts cardiac death in a general population

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Cardiac mortality;
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Aims The aim of this study was to assess the prognostic importance of the spatial QRS-T angle for fatal and non-fatal cardiac events.
Methods and results Electrocardiograms (ECGs) were recorded in 6134 men and women aged 55 years and over from the prospective population-based Rotterdam Study. Spatial QRS-T angles were categorized as normal, borderline or abnormal. Using Cox's proportional hazards model, abnormal angles showed increased hazard ratios of cardiac death (age-and sex-adjusted hazard ratio 5.2 (95% CI 4.0–6.8)), non-fatal cardiac events (2.2 (1.5–3.1)), sudden death (5.6 (3.7–8.5)) and total mortality (2.3 (2.0–2.7)). None of the classical cardiovascular and ECG predictors provided larger hazard ratios. After adjustment for these predictors, the association of abnormal spatial QRS-T angles with all fatal study endpoints remained strong, but the association with non-fatal cardiac events disappeared. Computation of Akaike's information criterion showed that the angle contributed significantly to the prediction of all fatal endpoints by classical cardiovascular and ECG predictors.
Conclusion The spatial QRS-T angle is a strong and independent predictor of cardiac mortality in the elderly. It is stronger than any of the classical cardiovascular risk factors and ECG risk indicators and provides additional value to them in predicting fatal cardiac events.

Introduction

Abnormalities of ventricular repolarization in the electrocardiogram (ECG), such as ST depression, T-wave inversion and QT prolongation, have repeatedly been shown to carry prognostic value for cardiac morbidity and mortality.[1,2] More recently, there has been renewed interest in vectorcardiographic parameters characterizing T-loop morphology to quantify ventricular repolarization.[3–5] A vectorcardiographic parameter that has been found to be a strong and independent risk indicator for cardiac events is the frontal T axis, which reflects the main orientation of electrical heart activity during repolarization.[4]

Another parameter that has recently been proposed as a marker of ventricular repolarization is the spatial QRS-T angle.[6–8] The spatial QRS-T angle is defined as the angle between the directions

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of ventricular depolarization and repolarization. Thus, unlike the T axis, it also takes depolarization into account, akin to the concept of the ventricular gradient.[9] Its usefulness in risk stratification of post infarction patients has been demonstrated,[5] but its prognostic value has not yet been studied in a large population-based cohort. Therefore, we set out to investigate whether an abnormal spatial QRS-T angle is a marker of increased cardiovascular disease and mortality in the Rotterdam Study, a population-based cohort study in men and women aged 55 years and over.

Methods

Study population and baseline data collection

The present study is part of the Rotterdam Study, a population-based cohort study aimed at assessing the occurrence of and risk factors for chronic diseases in the elderly. Objectives and methods of the Rotterdam Study have been described in detail elsewhere.[10] The Rotterdam Study cohort includes 3105 men and 4878 women aged 55 years and over (78% of the eligible population), living in a well-defined suburb of the city of Rotterdam, The Netherlands. The medical ethics committee of Erasmus Medical Centre, Rotterdam, approved the study. Participants gave written informed consent and permission to retrieve information from treating physicians.

Baseline data were collected from 1990 until 1993. A trained interviewer visited all subjects at home and collected information using a computerized questionnaire. The obtained information included current health status, medical history, drug use, smoking behavior, and family history of cardiovascular disease.

Additionally, in 7129 participants, established cardiovascular risk factors were measured at the research centre. Body mass index (BMI) was computed as weight divided by height squared. We defined hypertension as systolic blood pressure $\geqq$160 mmHg or diastolic blood pressure $\geqq$100 mmHg, or the use of antihypertensive medication for the indication of hypertension. Diabetes mellitus was defined as the use of blood glucose lowering medication or a random or post-load serum glucose level $\geqq$11.1 mmol/l.[11] A history of myocardial infarction was considered present in case of a self-report of myocardial infarction confirmed by ECG or additional clinical information, or the presence of an ECG characteristic of prior myocardial infarction. Presence of angina pectoris was established through the Rose questionnaire.[12]

ECG interpretation and measurements

A 12-lead resting ECG was recorded with an ACTA electrocardiograph (ESAOTE, Florence, Italy) at a sampling frequency of 500 Hz, and stored digitally. All ECGs were processed by the Modular ECG Analysis System (MEANS[13]) to obtain ECG measurements and interpretations. The MEANS program has been extensively evaluated,[13,14] including the operation of the waveform recognition algorithm.[15] MEANS determines common onsets and offsets for all 12 leads together on one representative averaged beat, with the use of template matching techniques.[13] Digitally stored ECGs of 6134 (86%) participants were available. Missing ECGs were mainly due to temporary technical problems with ECG recording.

Mean QRS and T axes were computed from vectorcardiographic X, Y and Z leads, which can, in good approximation, be reconstructed from the standard ECG leads.[16] The mean spatial axes are based on the areas of the wave components of the QRS complex and the T wave. The spatial QRS-T angle is the angle between the mean spatial QRS axis and the mean spatial T axis. The mean frontal T axis is the angle between the X axis and the projection of the mean spatial T axis on the frontal XY plane.

An overall corrected QTc interval was calculated from the common QRS onset and T offset for all 12 leads together. To adjust for heart rate, Bazett's formula was used.[17] ST depression was taken as Minnesota code 4.1 or 4.2 and T-wave inversion as Minnesota code 5.1 or 5.2.[18] Myocardial infarction found on ECG was based on a comprehensive set of criteria that partly derive from the Minnesota code. Left ventricular hypertrophy (LVH) on ECG was defined by using voltage and repolarization criteria, in which the age-adjusted Sokolow criterion[19] and T-wave abnormalities are the main parts.[14,20] Left bundle branch block (LBBB) on ECG was based on established criteria.[21]

Follow-up procedure

Follow-up started at the baseline examination and for the present study lasted until 1 January 2000. The mean follow-up time was 6.7 years (SD 2.3 years). Of all participants, 215 (2.7%) were lost to follow-up. Information on fatal and non-fatal endpoints for the participants enlisted with the general practitioners (GPs) working in the study district (85% of the cohort) was obtained from these GPs.

All of the participating GPs had computerized records, and fatal and non-fatal events of study participants were recorded on their computer file and sent to the Rotterdam Study data centre regularly. Subsequently, research assistants gathered information about these events at the GP offices. All medical records of the participants under the care of GPs outside the study area (15% of the cohort) were checked annually for possible events. Letters and, in case of hospitalization, discharge reports from medical specialists were obtained. With respect to the vital status of participants, information was also obtained regularly from the municipal health authorities in Rotterdam. After notification, cause and circumstances of death were established by questionnaire from the GPs.

Subsequently, two research physicians independently coded all reported events according to the International Classification of Diseases, 10th edition (ICD-10).[22] In case of disagreement, consensus was reached. Finally, a medical expert in cardiovascular disease, whose judgment was considered final, reviewed all events.

We defined cardiac death as death from myocardial infarction or other ischemic heart disease (ICD-10: I20-I25), sudden cardiac death (I46), sudden death undefined (R96), or death from ventricular fibrillation or tachycardia (I49) or congestive heart failure (I50). Non-fatal cardiac events were defined as non-fatal myocardial infarction (I21), coronary artery bypass graft or percutaneous transluminal coronary angioplasty. We defined sudden death as death occurring instantaneously or within 1 hour after onset of symptoms. This included codes I46, R96 and I49. Fatal myocardial infarction was defined as death within 30 days after having had a myocardial infarction (I21).

Data analysis

The spatial QRS-T angle was categorized into three groups: normal (0 to 105°), borderline (105 to 135°) and abnormal (135 to 180°). The threshold for abnormal angles was based on a previous report.[23] Differences in baseline characteristics between participants with normal, borderline and abnormal spatial QRS-T angles were examined with ANCOVA, adjusting for age and sex.

Cox's proportional hazards analysis was used to determine the relative risks of cardiac death, non-fatal cardiac events, sudden death and total mortality associated with borderline and abnormal spatial QRS-T angles, taking participants with normal spatial QRS-T angles as the reference group. In addition, the relative risks of fatal myocardial infarction and death due to congestive heart failure were determined separately. The proportional hazards assumption was tested by drawing log minus log plots of the survival function, which confirmed that the assumption was met. We adjusted for age and sex (model A), and subsequently for classical cardiovascular risk factors (model B) and other ECG risk indicators (model C). All continuous variables were dichotomized, except frontal T axis, which was divided into three categories.[4]

To assess whether the spatial QRS-T angle adds information to models containing known risk factors with regard to prediction of events, Akaike's information criterion (AIC) was used.[24] The spatial QRS-T angle, the classical cardiovascular risk factors and the other ECG risk indicators were added separately to a model containing age and sex, and the AICs obtained this way were compared. Subsequently, the spatial QRS-T angle was added to a model containing age and sex and the classical cardiovascular risk factors and a model containing age and sex and the other ECG risk indicators, including the frontal T axis.

To assess the value of the spatial QRS-T angle in asymptomatic subjects, all analyses were repeated after excluding subjects with self-reported myocardial infarction confirmed by ECG or additional clinical information at baseline. All analyses were performed using SPSS 9.0 for Windows.

Results

Table 1 shows baseline characteristics of all participants and of those with normal, borderline and abnormal spatial QRS-T angles, with adjustment for age and sex. A strong association with the spatial QRS-T angle was present for age, sex, systolic blood pressure, hypertension, ratio of total to HDL cholesterol, diabetes mellitus, history of angina pectoris and myocardial infarction, and other ECG characteristics. No association was found for current smoking, body mass index and diastolic blood pressure.

During follow-up, 1398 (22.8%) participants died; 312 (5.1%) died from a cardiac-related cause, including 134 (2.2%) sudden deaths. Non-fatal cardiac events were experienced by 339 (5.5%) participants. Table 2 shows the numbers of events and the incidence rates in the three categories of the spatial QRS-T angle.

In participants with borderline and abnormal spatial QRS-T angles, age- and sex adjusted hazard ratios for all study endpoints were significantly increased (Table 3, model A). After adjusting for classical cardiovascular risk factors (Table 3, model

Table 1 Baseline characteristics of all participants and according to three categories of the spatial QRS-T angle

Characteristic	All (*n*=6134)	Normal[a] (*n*=5163)	Borderline[a] (*n*=580)	Abnormal[a] (*n*=391)	*P* value
Age (years)	69.2(8.7)[b]	68.3	73.5	75.9	<0.001
Female sex	59.6%	61.0%	52.1%	52.4%	<0.001
Current smoking	21.0%	20.6%	22.3%	23.3%	0.354
Body mass index (kg/m^2)	26.3(3.7)	26.3	26.5	26.7	0.064
Blood pressure (mmHg)					
Systolic	139.4(22.3)	138.9	142.8	140.7	<0.001
Diastolic	73.5(11.6)	73.4	74.5	72.9	0.074
Hypertension	34.3%	32.5%	42.8%	45.8%	<0.001
Total cholesterol/HDL[c] ratio	5.2(1.6)	5.2	5.3	5.7	<0.001
Diabetes mellitus	10.0%	9.1%	16.0%	20.5%	<0.001
History of angina pectoris	3.8%	3.3%	5.4%	7.7%	<0.001
History of MI[d]	12.6%	9.4%	25.6%	36.9%	<0.001
Abnormal frontal T axis	7.3%	1.5%	24.1%	58.8%	<0.001
Borderline frontal T axis	7.1%	5.9%	14.5%	12.4%	<0.001
MI[d] by ECG	9.2%	6.5%	19.6%	29.0%	<0.001
LVH[e] by ECG	4.8%	2.7%	13.0%	20.7%	<0.001
LBBB[f] by ECG	1.9%	0.1%	3.2%	24.4%	<0.001
ST depression	9.3%	6.3%	22.6%	29.2%	<0.001
T-wave inversion	8.0%	4.0%	25.5%	33.9%	<0.001
QTc interval	431 (27)	429	437	455	<0.001

[a]Normal=0° <QRS-T angle <105°; borderline=105° <QRS-T angle <135°; abnormal=135° <QRS-T angle <180°.
[b]Values are means±SD for continuous variables and percentages for dichotomous variables, adjusted for age and sex where appropriate.
[c]HDL=high density lipoprotein.
[d]MI=myocardial infarction.
[e]LVH=left ventricular hypertrophy.
[f]LBBB=left bundle branch block.

Table 2 Number of events and crude incidence rates per 1000 person years (with 95% confidence interval), for all participants and according to three categories of the spatial QRS-T angle

Endpoint	All (6134)		Normal (5163)		Borderline (580)		Abnormal (391)	
	Events	Incidence rate	Events	Incidence rate	Events	Incidence rate	Events	Incidence rate
Cardiac death	312	7.3 (6.5–8.1)	173	4.7 (4.0–5.4)	51	14.1(10.2–18.0)	88	41.8(33.1–50.5)
Non-fatal cardiac event	339	8.2 (7.3–9.1)	265	7.4 (6.5–8.3)	38	10.9 (7.4–14.4)	36	18.2(12.3–24.2)
Sudden death	134	3.1 (2.6–3.7)	74	2.0 (1.5–2.5)	23	6.4 (3.8–9.0)	37	17.6(11.9–23.3)
Total mortality	1398	33.5(31.7–35.2)	938	25.9(24.2–27.6)	225	63.7(55.4–72.0)	235	112.9(98.5–127.3)

B), significantly increased hazard ratios were observed for all fatal study endpoints but not for non-fatal cardiac events. After adjusting for the other ECG risk indicators (Table 4, model C), a strong, independent association was found between abnormal spatial QRS-T angles and all fatal study endpoints. Again, this was not the case for non-fatal cardiac events. In the multivariable analyses, abnormal spatial QRS-T angles provided higher hazard ratios for the fatal study endpoints than any of the classical cardiovascular or other ECG risk factors.

In additional analyses of separate components of our cardiac death endpoint, hazard ratios of abnormal spatial QRS-T angles for death from congestive heart failure (*n*=150) were found to be 4.6 (CI 2.5–8.5) adjusted for classical cardiovascular risk factors and 4.5 (2.3–8.6) adjusted for other ECG risk indicators. For fatal myocardial infarction (*n*=93), these hazard ratios were 2.1 (1.0–4.5) and 1.4 (0.6–3.2).

Results remained essentially the same after excluding subjects with self-reported myocardial infarction confirmed by ECG or additional clinical information at baseline (*n*=767).

Table 5 shows Akaike's information criteria (AICs) for the outcome cardiac death. When the predictors were added to model A separately,

Table 3 Hazard ratios for abnormal and borderline spatial QRS-T angles and cardiovascular risk factors for cardiac death, non-fatal cardiac events, sudden death and total mortality

Endpoint (number of events) and risk factors	Hazard ratio (95% confidence interval)	
	Model A[a]	Model B[b]
Cardiac death[c] (*n*=312)		
Spatial QRS-T angle		
Borderline	2.1(1.5–2.8)	1.7(1.2–2.4)
Abnormal	5.2(4.0–6.8)	3.7(2.7–5.0)
Current smoking	1.2(1.0–1.4)	1.0(0.8–1.4)
Body mass index >25kg/m^2	1.1(0.7–1.8)	0.8(0.6–1.0)
Hypertension	1.9(1.6–2.3)	1.7(1.3–2.2)
Total cholesterol/HDL[d] ratio >7.2	2.0(1.7–2.4)	1.3(0.9–1.8)
Diabetes mellitus	1.9(1.6–2.2)	1.8(1.4–2.5)
History of angina pectoris	2.4(1.9–3.0)	1.8(1.2–2.7)
History of myocardial infarction	2.9(2.3–3.6)	1.9(1.5–2.6)
Non-fatal cardiac events (*n*=339)		
Spatial QRS-T angle		
Borderline	1.3(0.9–1.8)	0.9(0.6–1.3)
Abnormal	2.2(1.5–3.1)	1.3(0.9–1.9)
Sudden death[e] (*n*=134)		
Spatial QRS-T angle		
Borderline	2.3(1.4–3.7)	1.9(1.1–1.3)
Abnormal	5.6(3.7–8.5)	4.4(2.8–6.9)
Total mortality (*n*=1398)		
Spatial QRS-T angle		
Borderline	1.6(1.4–1.8)	1.4(1.2–1.7)
Abnormal	2.3(2.0–2.7)	1.8(1.5–2.2)

[a]Model A: all variables entered separately, adjusted for age and sex.
[b]Model B: all variables entered simultaneously, adjusted for age and sex.
[c]Cardiac death: myocardial infarction or other ischemic heart disease, sudden cardiac death, sudden death undefined, ventricular fibrillation or tachycardia, congestive heart failure.
[d]HDL=high-density lipoprotein.
[e]Sudden death: sudden cardiac death, sudden death undefined, ventricular fibrillation or tachycardia.

AIC for the addition of spatial QRS-T angle was significantly higher than the other AICs. Significant AICs were obtained when the spatial QRS-T angle was added to a model containing age, sex, and the classical cardiovascular risk factors (model B), and a model containing age, sex, and the other ECG risk factors, including the frontal T axis (model C). Similar results were found for sudden death and total mortality. However, for non-fatal cardiac events, the spatial QRS-T angle did not have additive predictive value. After repeating the analyses in subjects without myocardial infarction, results remained essentially the same when using models A and B. Using model C, AIC remained significant for total mortality.

Discussion

An abnormal spatial QRS-T angle was the strongest marker of increased risk of cardiac mortality, including sudden death, in men and women aged 55 years and over, compared to classical cardiovascular risk factors and other ECG risk indicators. The association between the spatial QRS-T angle and cardiac mortality was independent of the classical cardiovascular and other ECG predictors, including the frontal T axis. No association was found between the abnormal spatial QRS-T angle and non-fatal cardiac events when classical cardiovascular or other ECG predictors were taken into account. Furthermore, the spatial QRS-T angle provided an additional contribution to the prediction of cardiac mortality by classical cardiovascular and other ECG predictors.

The concept of the spatial QRS-T angle has been known for a long time,[23] but has recently gained new interest. Zabel et al. used a parameter, total cosine R to T, that resembles the spatial QRS-T angle and found that this parameter permits accurate assessment of post-myocardial infarction risk.[5] Dilaveris et al. showed that the spatial QRS-T angle is a measure of ventricular repolarization, that differs between myocardial infarction patients and control subjects.[6] Furthermore, they found that

Table 4 Hazard ratios for abnormal and borderline spatial QRS-T angles and ECG risk indicators for cardiac death, non-fatal cardiac events, sudden death and total mortality

Endpoint (number of events) and risk factors	Hazard ratio (95% confidence interval)	
	Model A[a]	Model C[b]
Cardiac death[c] (*n*=312)		
Spatial QRS-T angle		
Borderline	2.1(1.5–2.8)	1.3(0.9–1.8)
Abnormal	5.2(4.0–6.8)	2.7(1.9–4.0)
Frontal T axis		
Borderline	3.0(2.2–4.1)	2.0(1.4–2.8)
Abnormal	4.1(3.1–5.4)	1.2(0.8–1.9)
Myocardial infarction by ECG	2.4(1.8–3.1)	1.6(1.2–2.1)
LVH[d] by ECG	2.8(2.1–3.9)	1.4(0.9–2.0)
LBBB[e] by ECG	2.7(1.7–4.2)	1.2(0.7–2.1)
ST depression	2.6(2.0–3.4)	1.4(1.0–2.0)
T-wave inversion	3.2(2.4–4.1)	1.4(1.0–2.2)
QTc interval >440 ms	1.7(1.4–2.2)	1.5(1.2–1.9)
Non-fatal cardiac events (*n*=339)		
Spatial QRS-T angle		
Borderline	1.3(0.9–1.8)	0.8(0.6–1.2)
Abnormal	2.2(1.5–3.1)	1.0(0.6–1.6)
Sudden death[f] (*n*=134)		
Spatial QRS-T angle		
Borderline	2.3(1.4–3.7)	1.6(0.9–2.7)
Abnormal	5.6(3.7–8.5)	3.4(1.9–6.0)
Total mortality (*n*=1398)		
Spatial QRS-T angle		
Borderline	1.6(1.4–1.8)	1.3(1.1–1.5)
Abnormal	2.3(2.0–2.7)	1.8(1.5–2.2)

[a]Model A: all variables entered separately, adjusted for age and sex.
[b]Model C: all variables entered simultaneously, adjusted for age and sex.
[c]Cardiac death: myocardial infarction or other ischemic heart disease, sudden cardiac death, sudden death undefined, ventricular fibrillation or tachycardia, congestive heart failure.
[d]LVH=left ventricular hypertrophy.
[e]LBBB=left bundle branch block.
[f]Sudden death: sudden cardiac death, sudden death undefined, ventricular fibrillation or tachycardia.

the spatial QRS-T angle is the only repolarization marker to be significantly increased in treated hypertensive patients who show repeat office measurements of high blood pressure, indicating that it is a sensitive and early marker of the repolarization alterations in systemic hypertension.[7] These studies suggest that abnormal spatial QRS-T angles are associated with worse cardiac outcomes. Furthermore, spatial characteristics of ventricular repolarization have been shown to assess ECG qualities that are different from conventional ECG parameters,[3] and may thus have additional value, which was reflected in our Akaike analysis.

To our knowledge, this study is the first to report on the predictive value of the spatial QRS-T angle within a large, prospective, population-based cohort. The strength of our study lies in the fact that ECG data are available for more than 6000 participants and large numbers of incident events are present, which provides good statistical power. Also, the follow-up period is relatively long, there is little loss to follow-up, and information about many cardiovascular risk factors is collected at baseline, giving us the possibility to take possible confounders into account.

Measurement of the spatial QRS-T angle is likely to be less susceptible to noise and problems of definition than many of the more conventional ECG parameters. Accurate determination of waveform recognition points, in particular the end of the T wave, is less critical for calculation of the QRS-T angle. Thus, the spatial QRS-T angle is likely to be a much more robust and reproducible measurement than QT dispersion, which has also been used to quantify ventricular repolarization but was shown to have several methodological limitations.[25,26] Determination of the spatial QRS-T angle requires a computer program, but the algorithm can easily be implemented on modern electrocardiographs, which are nowadays equipped with sufficient computing power.

Table 5 Akaike's information criterions for addition of spatial QRS-T angle to models predicting cardiac death

Model	+Added variable	AIC	*P* value
Model A[a]	+Spatial QRS-T angle	120.5	<0.001
Model A	+Current smoking	−0.7	0.248
Model A	+Body mass index >25 kg/m^2	−1.7	0.579
Model A	+Hypertension	38.7	<0.001
Model A	+Total cholesterol/HDL[b] ratio >7.2	9.8	0.001
Model A	+Diabetes mellitus	35.3	<0.001
Model A	+History of angina pectoris	8.8	0.001
Model A	+History of myocardial infarction	72.5	<0.001
Model A	+Frontal T axis	105.7	<0.001
Model A	+Myocardial infarction by ECG	31.7	<0.001
Model A	+LVH[c] by ECG	32.0	<0.001
Model A	+LBBB[d] by ECG	13.0	<0.001
Model A	+ST depression	41.4	<0.001
Model A	+T-wave inversion	58.9	<0.001
Model A	+QTc interval >440 ms	20.8	<0.001
Model B[e]	+Spatial QRS-T angle	57.3	<0.001
Model C[f]	+Spatial QRS-T angle	14.9	<0.001

[a]Model A: age, sex.
[b]HDL=high-density lipoprotein.
[c]LVH=left ventricular hypertrophy.
[d]LBBB=left bundle branch block.
[e]Model B: age, sex, current smoking, body mass index >25 kg/m^2, hypertension, total cholesterol/HDL ratio >7.2, diabetes mellitus, history of angina pectoris and history of myocardical infarction.
[f]Model C: age, sex, frontal T axis, myocardial infarction by ECG, left ventriculcar hypertrophy by ECG, left bundle branch block by ECG, ST depression, T-wave inversion, QTc interval >440 ms.

The spatial QRS-T angle was found to be associated with fatal cardiac events, but not with non-fatal cardiac events. This could be ascribed to the occurrence of lethal rhythm disturbances, which play an important role in fatal cardiac events. An abnormal spatial QRS-T angle may reflect, possibly subclinical, damaged areas of the myocardium that could distort the normal spread of electrical forces through the myocardial wall. As a result, subjects with an abnormal spatial QRS-T angle may be prone to ventricular rhythm disturbances that result in fatal events, and to a lesser extent in non-fatal events. This explains the high predictive value of abnormal spatial QRS-T angles for sudden death in our study and also the stronger association with death from congestive heart failure than with fatal myocardial infarction. The former results in arrhythmic death more often than the latter.[27]

Several aspects of the study warrant further consideration. Firstly, the pathophysiological mechanisms underlying repolarization abnormalities and their relationship with changes in the QRS-T angle are largely unresolved.[6] Secondly, the thresholds to distinguish between different spatial QRS-T angle categories may further be optimized. In our analyses, the threshold for abnormal angles was based on a previous study.[23] When we repeated our analyses after decreasing the thresholds by 15°, results did not change materially. We also considered a dichotomization of spatial QRS-T angles based on percentiles, taking the upper limits of intervals that contained 5, 10 or 15% of the QRS-T angles as boundaries, but again results remained essentially the same. However, for risk stratification purposes optimum boundaries, possibly age and gender specific, may need to be determined. Finally, our study population consisted of people aged 55 years and over. Whether the spatial QRS-T angle is an important predictor of cardiac events in younger age groups as well requires further study.

In conclusion, the spatial QRS-T angle is the strongest predictor of cardiac mortality in the elderly compared to classical cardiovascular risk factors and other ECG risk indicators, regardless of pre-existing myocardial infarction. If this finding is confirmed and expanded by future population-based studies, measurement of the spatial QRS-T angle could be considered in clinical practice for the identification of individuals at high risk of cardiac death.

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